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Impaired implicit learning of syntactic structure in children with developmental language disorder: Evidence from syntactic priming

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Abstract

Background and aims: Implicit learning mechanisms associated with detecting structural regularities have been proposed to underlie both the long-term acquisition of linguistic structure and a short-term tendency to repeat linguistic structure across sentences (structural priming) in typically developing children. Recent research has suggested that a deficit in such mechanisms may explain the inconsistent trajectory of language learning displayed by children with Developmental Learning Disorder. We used a structural priming paradigm to investigate whether a group of children with Developmental Learning Disorder showed impaired implicit learning of syntax (syntactic priming) following individual syntactic experiences, and the time course of any such effects.

Methods: Five- to six-year-old Italian-speaking children with Developmental Learning Disorder and typically developing age-matched and language-matched controls played a picture-description-matching game with an experimenter. The experimenter's descriptions were systematically manipulated so that children were exposed to both active and passive structures, in a randomized order. We investigated whether children's descriptions used the same abstract syntax (active or passive) as the experimenter had used on an immediately preceding turn (no-delay) or three turns earlier (delay). We further examined whether children's syntactic production changed with increasing experience of passives within the experiment.

Results: Children with Developmental Learning Disorder's syntactic production was influenced by the syntax of the experimenter's descriptions in the same way as typically developing language-matched children, but showed a different pattern from typically developing age-matched children. Children with Developmental Learning Disorder were more likely to produce passive syntax immediately after hearing a passive sentence than an active sentence, but this tendency was smaller than in typically developing age-matched children. After two intervening sentences, children with Developmental Learning Disorder no longer showed a significant syntactic priming effect, whereas typically developing age-matched children did. None of the groups showed a significant effect of cumulative syntactic experience.

Conclusions: Children with Developmental Learning Disorder show a pattern of syntactic priming effects that is consistent with an impairment in implicit learning mechanisms that are associated with the detection and extraction of abstract structural regularities in linguistic input. Results suggest that this impairment involves reduced initial learning from each syntactic experience, rather than atypically rapid decay following intact initial learning.

Implications: Children with Developmental Learning Disorder may learn less from each linguistic experience than typically developing children, and so require more input to achieve the same learning outcome with respect to syntax. Structural priming is an effective technique for manipulating both input quality and quantity to determine precisely how Developmental Learning Disorder is related to language input, and to investigate how input tailored to take into account the cognitive profile of this population can be optimised in designing interventions.

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Structural priming, syntactic priming, implicit learning, syntax, developmental language disorder, specific language disorder

Introduction

For children with developmental language disorder (DLD), language learning proceeds slowly or along an inconsistent trajectory that is not associated with a known biomedical aetiology (see Bishop, Snowling, Thompson, Greenhalgh, CATALISE Consortium, 2016, for a consensus in adopting the term DLD). Recent research has suggested that this impaired language development, particularly evident for grammar, may arise from a deficit in implicit learning mechanisms that underlie the detection and extraction of structural patterns in input (Evans, Saffran, & Robe-Torres, 2009; Hsu & Bishop, 2011; Ullman & Pierpont, 2005; see Obeid, Brooks, Powers, Gillespie-Lynch, & Lum, 2016, for a meta-analysis). In this paper, we report a study that used a structural priming paradigm to investigate whether children with DLD show the same pattern of immediate, persistent, and cumulative implicit learning of syntactic structure as typically developing (TD) children.

Ullman (2001, 2004) proposed a declarative-procedural model of language in which the acquisition and use of lexical and general semantic knowledge implicates the declarative memory system, whereas acquisition and use of rule-governed aspects of language (such as syntax and morphology) implicates the procedural memory system. Whereas learning in declarative memory is at least partly explicit (Chun, 2000; Daselaar, Fleck, Prince, & Cabeza, 2006), learning in procedural memory is assumed to primarily implicate the implicit (non-conscious) acquisition and application of knowledge (though see e.g. Andringa & Curcic, 2015; Lichtman, 2016, for evidence that procedural learning may also involve an explicit component). Evidence from a range of linguistic and non-linguistic tasks, including artificial grammar learning (AGL) and serial reaction time (SRT) tasks, supports the claim that people can incidentally learn sequential regularities in ways that affect their subsequent behaviour, without necessarily having explicit awareness of this knowledge (e.g. Abrahamse, Jiménez, Verwey, & Clegg, 2010; Reber, 1969). Under Ullman's account, children's syntactic development occurs gradually and implicitly through repeated exposure to input, drawing on generalized procedural learning mechanisms. Ullman and Pierpont (2005) adopted this framework to propose that the characteristic pattern of grammatical impairment alongside relatively spared vocabulary found in children with DLD arises from a generalised deficit in procedural learning mechanisms whose effects are not limited to language.

Implicit learning for language development

Recent research has tested the procedural learning deficit hypothesis by examining children with DLD's performance in tasks involving AGL, probabilistic categorical learning, and statistical learning, and has found results consistent with a procedural learning deficit. For example, studies manipulating the quantity of exposure to a particular pattern of events (e.g. tracking transitional probabilities in speech) have shown that although children with DLD can learn such patterns, they nevertheless have difficulties generalizing these patterns when they are exposed to new material, compared to TD controls (see Evans et al., 2009). Other research has found a deficit in implicit learning of repeated sequences of nouns, with 7–11-year-old children with DLD showing the same level of performance as younger (grammar-matched) TD children (Hsu & Bishop, 2014). Children with DLD also demonstrate deficits in implicit learning of non-linguistic patterns (e.g. sequences of visual patterns in a SRT task), consistent with a domain-general impairment (Hsu & Bishop, 2014; Lum, Gelgec, & Conti-Ramsden, 2010; Tomblin, Mainela-Arnold, & Zhang, 2007). Hsu and Bishop found further that children with DLD were not impaired in pursuit rotor motor learning, suggesting that the deficit relates to sequence-specific information.

But although such evidence suggests an implicit learning deficit in DLD, it does not show directly that children with DLD have an implicit learning deficit that affects their language. To address this issue, we turn to *structural priming*, or the tendency for people to repeat linguistic structure across otherwise unrelated sentences (see Branigan & Pickering, 2017 and Pickering & Ferreira, 2008, for reviews).

Syntactic priming in language development

Bock (1986) reported priming of syntactic structure (i.e. *syntactic priming*) in adult language production, whereby adults were more likely to use a passive structure to describe a picture (e.g. *The church was struck by lightning*) after repeating a passive sentence describing a different event (e.g. *The boy was woken by the alarm clock*) than after an equivalent active sentence (e.g. *The alarm clock woke the boy*).

Recent studies have similarly demonstrated robust syntactic priming effects in TD children's production, with pre-school- and school-aged children showing an increased tendency to produce particular structures after hearing and/or repeating sentences involving the same structure (e.g. Bencini & Valian, 2008; Huttenlocher, Vasilyeva, & Shimpi, 2004; Messenger, Branigan, McLean, & Sorace, 2012; Rowland, Chang, Ambridge, Pine, & Lieven, 2012). This tendency occurred even for structures that they would not normally spontaneously produce (e.g. passives). Repetition of structure occurred independent of repetition of open- or closed-class lexical content or meaning, and between comprehension and production, implicating abstract amodal syntactic representations (e.g. Bock, 1989; Bock & Loebell, 1990; Branigan, Pickering, & Cleland, 2000; Messenger, Branigan, McLean, et al., 2012; Pickering & Branigan, 1998).

Syntactic priming effects have been explained as a manifestation of implicit learning (e.g. Bock & Griffin, 2000; Chang, Bock, & Dell, 2006): Processing the structure of a *prime* sentence causes adjustments to the underlying system that supports syntactic processing, and these adjustments persist to affect processing of a subsequent *target* sentence. For example, exposure to a passive sentence strengthens the weights associated with the passive structure, increasing the likelihood of using a passive structure in subsequent processing. Furthermore, the effects of individual exemplars persist and accumulate, so that ultimately the syntactic system accurately reflects the input to which it has been exposed. Thus, syntactic priming effects index speakers' ability to detect and dynamically respond to the frequencies of abstract syntactic structures in the input, with individuals varying in their learning rate (i.e. the extent to which weights are changed in response to each experience, and the rate at which these weight changes decay). Some accounts suggest that other mechanisms (e.g. residual activation of lexical and syntactic representations within the language processing system following use, as assumed to underlie lexical priming effects) may also be implicated in immediate priming effects, alongside such implicit learning (e.g. Branigan & McLean, 2016; Coco et al. 2012; Hartsuiker, Bernolet, Schoonbaert, Speybroeck, & Vanderelst, 2008; Reitter, Keller, & Moore, 2011).

Chang et al. (2006) argued that implicit learning explains not only immediate syntactic priming effects, but also how children initially acquire syntax. This claim is consistent with evidence that both children and adults show cumulative syntactic priming effects when given repeated and extensive exposure to a structure during an experiment (e.g. Branigan & McLean, 2016; Garraffa, Coco, & Branigan, 2015; Kaschak, Loney, & Borregine, 2006), and from one experimental session to another (Branigan & Messenger, 2016; Kaschak, Kutta, & Schatschneider, 2011; see also Vasilyeva, Huttenlocher, & Waterfall, 2006), as well as evidence that children's long-term syntactic development reflects the statistics of the input to which they are exposed (e.g. Abbot-Smith, Lieven, & Tomasello, 2001; Akhtar, 1999; Huttenlocher, Waterfall, Vasilyeva, Vevea, & Hedges, 2010; Theakston, Lieven, Pine, & Rowland, 2000).

The proposal that implicit learning mechanisms are implicated in syntactic development and processing, and – critically – that they can be tapped by syntactic priming is supported by Kidd (2012), who showed that individual differences in four- to six-year-old TD children's implicit statistical learning predicted their sensitivity to cumulative effects of syntactic input. Children who showed better learning of visual sequences in an SRT task were more likely to produce passive sentences after exposure to a set of passive prime sentences than children who showed worse visual sequence learning (although sequence learning did not predict whether children were immediately primed, i.e. produced a passive immediately after exposure to a single passive prime).

Syntactic priming paradigms therefore offer a direct means of testing implicit syntactic learning in children. If children with DLD have impaired implicit learning mechanisms that affect their ability to learn grammar, then this should be reflected in aberrant patterns of syntactic priming effects, compared to TD children. Their specific patterns of priming effects are also potentially informative about the nature of their impairment. Although some studies have examined the extent to which children with DLD are influenced by prior exposure to syntactic structures (e.g. Leonard, Miller, Deevy, et al., 2002; Leonard, Miller, Grela, et al., 2000; Riches, 2012), few studies have investigated whether children with DLD show different patterns of syntactic priming from TD children. Miller and Deevy (2006) found no difference in immediate priming between English-speaking TD children and children with DLD for simple intransitive and transitive sentences. Garraffa et al. (2015) and Foltz, Thiele, Kahsnitz, and Stenneken (2015) examined syntactic priming of subject relative clauses in Italian- and German-speaking children respectively, and found comparable immediate priming in both DLD and TD groups. However, Garraffa et al. found that children with DLD showed a smaller learning effect with cumulative exposure: Whereas each experience of a relative clause increased TD children's overall likelihood of producing a relative clause by 2%, the corresponding increase was only 1% in children with DLD.

Research question

Garraffa et al.'s (2015) study provides some preliminary evidence for impaired implicit learning of syntax in DLD, but leaves open the precise nature of the impairment. In the current study, we sought to address this question by focusing on the time-course of syntactic priming effects in TD and DLD children. Specifically, do children with DLD show reduced initial learning when exposed to a structure, or intact initial learning that has an atypically rapid rate of decay, or both reduced initial learning and a more rapid rate of decay? We investigated passive structures, which have been well studied in both TD children's (e.g. Gordon & Chafetz, 1990; Maratsos, Fox, Becker, & Chalkley, 1985; Volpato, Verin, & Cardinaletti, 2016) and DLD children's (e.g. Bishop, Adams, & Rosen, 2006; Marinis & Saddy, 2013) development. Importantly, they have been shown to be susceptible to immediate and persistent syntactic priming in TD children (e.g. Bencini & Valian, 2008; Branigan & McLean, 2016; Branigan & Messenger, 2016; Messenger, Branigan, & McLean, 2011; Messenger, Branigan, McLean, et al., 2012).

We therefore conducted an experiment in which five- to six-year-old Italian-speaking children with DLD and groups of typically developing age-matched (*TDA*) and language-matched (*TDL*) controls alternated describing picture cards with an experimenter as part of a picture-matching game (Branigan, McLean, & Jones, 2005). We manipulated the structure of the experimenter's descriptions (*active* vs. *passive prime* conditions) in a within-participants design, and examined how this manipulation affected children's likelihood of producing a passive description on a subsequent turn. Thus, we examined whether children produced more passive target descriptions after hearing a passive prime description than an active prime description. We further compared children's tendency to repeat structure immediately following the experimenter's description and after two intervening utterances (i.e. three turns later; *no-delay* vs. *delay* conditions), to determine the persistence of effects of syntactic experience. We also examined whether children's tendency to repeat structure changed with increasing experience of passives within the experiment, to investigate syntactic learning from cumulative experience.

Thus, our experiment investigated differences between children with DLD and TD children in response to experimentally controlled syntactic experience when language exposure (over the lifetime) was controlled (TDA group) and when language ability was controlled (TDL group). If children with DLD have impaired implicit language learning mechanisms, then they should show a weaker tendency than age-matched TD children to be influenced by the structure of the experimenter's descriptions. The time-course of such effects would be informative about the nature of the implicit learning impairment, with weaker immediate priming suggesting that children with DLD experience reduced initial learning from an individual exemplar compared to TD children with similar lifetime language experience, whereas reduced persistence of priming over intervening utterances would imply a faster rate of decay in learning. Weaker effects of increasing exposure would imply a reduced cumulative learning effect, such that learning from each new experience would build on learning from previous experiences to a lesser extent in children with DLD as in TD children of the same age.

Comparisons with language-matched TD children allowed us to examine whether children with DLD showed a delayed learning trajectory (consistent with evidence from other studies of non-linguistic implicit learning; Hsu & Bishop, 2014) or alternatively a qualitatively different learning trajectory from TD children. In the former case, we would expect them to show a similar pattern of performance to the language-matched TD group alongside a different pattern from the age-matched TD group; in the latter case, we would expect them to show a different pattern of performance from the language-matched TD group, as well as the age-matched TD group.

Method

Participants

Forty-two Italian children participated: 14 children with DLD (MCA = 5;8) were matched group-wise with 14 TD children based on chronological age (age in months; $t(25) = 0.54$, $p = 0.6$), and with 14 TD children based on receptive grammatical abilities measured using the Italian version of the Test for Reception of Grammar (TROG-2; Suraniti, Ferri, & Neri, 2009) (number of blocks passed; $t(24) = -0.16$, $p = 0.9$).

Children with DLD were recruited from a clinic treating language impairment, and were diagnosed as DLD by expert neuropsychologists. The inclusion criteria adopted by the screening protocol included: (a) a normal non-verbal IQ (>85 in the standardised Italian version of the WPPSI-3 Wechsler Preschool and Primary Scale of Intelligence; Cianchetti, C. & Sannio-Fancello, G. (2007), and (b) a performance of at least 1SD below the mean on two or more measures of expressive and receptive language based on the Test Neuropsicologico Prescolare (TNP), a normed battery of tests standardly used in Italy to assess expressive and receptive vocabulary and grammar skills in Italian preschool children (sentence-level structures tested include sentential negations, dative sentences, and subject

relatives; Cossu & Paris, 2007). All children included as DLD matched all inclusion criteria.

None of the children had any hearing impairment, autistic spectrum disorder, or other known syndromes. Parents of children included in the study provided informed consent. The study was approved by the clinic's scientific committee and complied with APA ethical standards.

We also assessed all children using the Italian versions of: (a) the Test for Reception of Grammar, version 2 (TROG-2, Suraniti et al., 2009); (b) the Peabody Picture Vocabulary Test IV (PPVT-4, Stella, Pizzoli, & Tressoldi, 2000); (c) non-word repetition (*Prove di Prerequisito per la Diagnosi delle Difficoltà di Lettura e Scrittura 2*, PRCR2, Cornoldi, Miato, Molin, & Poli, 2009); and a sentence repetition task (see Garraffa et al., 2015, for details), to corroborate children with DLD's diagnosis and verify that TD children had normal language development. All TD children performed within the normal range (based on standard scores) in all language tests (see Table 1). We used a paired t-test to examine whether the DLD differed from TDA on Age and TDL on the TROG-2, and found that the DLD were significantly older than the TDA; $t(13) = 2.68$, p-value $= 0.01$, but their performance on TROG-2 was not significantly different than the TDL group; $t(13) = -0.32$, $p = 0.7$.

Table 1. Overview of groups (DLD; TDA; TDL): mean ± standard deviation (and range) for age (in years), WPPSI-III (non-verbal IQ), and performance on linguistic tasks (TNP, TROG-2, PPVT-4, PRCR-2, and sentence repetition). TNP subtests scores, PPVT-4 and Sentence repetition are based on number of correct responses. TROG-2 scores are measured in terms of block passed in the test. PRCR-2 scores record the number of syllables correctly produced.

Group	DLD	TDA	TDL
Age	5.8 ± 0.3 (5–6.3)	5.7 ± 0.4 (5.1–6.4)	4.8 ± 0.3 (4.3–5.2)
WPPSI-III	101.5 ± 8.1 (88–112)	101.2 ± 4.9 (94–114)	99.8 ± 5.6 (88–110)
TNP Receptive grammar	4.1 ± 1.3 (2–6)	–	–
TNP Expressive grammar	3.6 ± 1.2 (2–6)	–	–
TNP Receptive vocabulary	9.5 ± 0.6 (8–10)	–	–
TNP Expressive vocabulary	9.2 ± 0.4 (9–10)	–	–
TROG-2 blocks	6.3 ± 1 (4–8)	15.6 ± 2 (12–18)	6.4 ± 1.2 (5–8)
PPVT-4	103 ± 11.3 (80–116)	116 ± 13.1 (98–134)	105 ± 10 (90–121)
PRCR-2 (non word repetition)	11 ± 3.8 (7–18)	23 ± 1.3 (15–21)	15.5 ± 2.44 (12–21)
Sentence repetition	4.4 ± 1.2 (2–6)	8.6 ± 1.3 (6–10)	7 ± 0.8 (6–8)

DLD: developmental language disorder; TDA: typically developing age-matched group; TDL: typically developing language-matched group.

Materials

We used 24 experimental items based on Messenger, Branigan, McLean, et al.'s (2012) materials, adapted to Italian (Manetti, 2013). Each item consisted of a prime picture and an associated prime sentence, a target picture, and two filler pictures. There were two versions of each prime description, active and passive. The pictures depicted a transitive event involving an animal agent and a human patient; there was no overlap in action or characters between each prime picture and its associated target picture. Filler pictures depicted individual objects. In the no-delay conditions, the prime and target pictures appeared consecutively, followed by the filler pictures; in the delay conditions, the two fillers appeared between the prime and target (see Figure 1 for example trials). Functional to the game, there were also six 'Snap' items (in which the experimenter and child had identical pictures), four depicting transitive actions (two paired with an active prime description and two paired with a passive prime description), and two depicting individual objects. We produced four Latin squared lists, such that across the four lists each target occurred once in each of the four priming conditions and within a list six targets occurred in each of the four priming conditions. Each participant received one list in an individually randomized order.

Design. We used a $3 \times 2 \times 2$ mixed design, with Group (DLD vs. TDA vs. TDL) as a between-participants and within-items factor, and prime (active vs. passive) and delay (no-delay vs. delay) as within-participants and -items factors.

Response coding

We coded children's responses following Messenger, Branigan, McLean, et al. (2012), adapted to Italian. Responses were coded as *Active* if they contained an overt or null subject bearing the agent role, active transitive verb, and direct object (full NP) bearing the patient role; *Passive,* if they contained an overt or null subject bearing the patient role, passive auxiliary *venire* or *essere* ('to come' or 'to be'), past participle, *da* (by), and oblique object bearing the agent role; or *Other* (all other responses). Complete active/passive utterances were further coded for structural and morphological errors (e.g. auxiliary omissions, incorrect

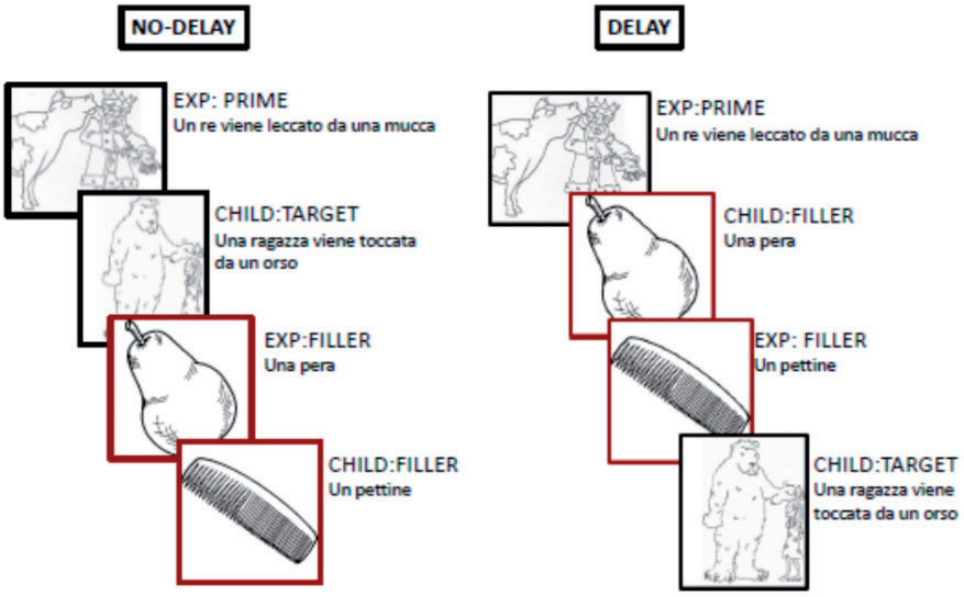

Figure 1. Example trials in passive prime/no-delay and passive prime/delay conditions.
Exp: experimenter's picture description; *Child*: child's description.

inflections); see Appendix for details of the coding scheme.

Results

Analysis

Our analyses focused on the production of passive responses (out of a total of 867 observations divided in: 266 DLD, 302 TDA, 299 TDL responses) in the three different groups of children (DLD, TDA and TDL). We used a strict coding criterion (complete correct [well-formed] actives and passives, i.e. responses coded as ACC and PAC[1]) to derive our dependent measure, a binomial where 1 indicated the production of a passive response, and 0 indicated the production of an active response; all other responses were excluded (i.e. considered as null-values (NA); 13.5% responses).

We analysed the data using used linear mixed-effects (LME) models as implemented in the lme4 R package (Bates, Maechler, Bolker, & Walker, 2015). We adopted an LME modelling approach because it allows simultaneous modelling of by-participant and by-item random effects. This approach is advantageous for analysing small samples as in the current study (Muth et al., 2016), as it avoids data aggregation. Such aggregation results in less precise estimates when correlations are unequal across repeated measures residuals and may yield biased estimates, especially with small samples.

The dependent measure was modelled as a function of prime (active, passive), delay (no-delay, delay), and group (DLD and TDA in the comparison of children with DLD and TDA children; DLD and TDL in the comparison of children with DLD and TDL children). In further analyses, we also included cumulative (an incremental variable indexing how many passive primes a child had experienced prior to the current trial[2]). The impact of cumulative priming was assessed for each group of children independently to obtain an estimate that reflected each specific group's performance. All fixed effects were centred to reduce collinearity. The random effects were child (28 levels), entered as a between-participants variable, and item (28 levels).

For each analysis, we first fitted mixed-effects models with a maximal random effects structure (i.e. random variables introduced as intercepts and uncorrelated slopes; an approach known to result in the lowest Type 1 error rate; Barr et al., 2013) and a full fixed-effects structure (i.e. all main effects and their interaction). Then, in order to have models that reflected the experimental design but were also parsimonious in the number of parameters and did not overfit the data (Matuschek, Kliegl, Vasishth, Baayen, & Bates, 2017), we reduced our models utilizing the 'step' function of the R package lmerTest (Kuznetsova, Bruun Brockhoff, & Haubo Bojesen Christensen, 2014). This method performs a backward selection of the model, for both the random- and fixed-effect structure, by iteratively removing terms that do not significantly improve the model fit ($p < 0.1$ for random effects and $p < 0.05$ for fixed effects); see Kuznetsova et al. (2014). The results tables report the coefficients, standard errors, and t-values of the final model. We derived

p-values for the fixed effects in the LME models from F-tests based on Satterthwaite approximation of the effective degrees of freedom (Satterthwaite, 1946).

Comparison of children with DLD and TDA children

Table 2 shows response frequencies by group and condition; Figure 2 shows the percentage of passives (out of all complete correct active and passive responses) produced by each group in each condition. Table 3 shows the results of the mixed-effects analyses. There was an effect of *Group*: Children with DLD were less likely to produce passives than TDA children; an effect of *Prime*: Children were more likely to produce a passive structure after hearing a passive prime than after hearing an active prime; and an effect of *Delay:* Children were more likely to produce a passive structure immediately after hearing a prime than when two fillers intervened between prime and target.

Critically, the final model also included interactions between *Group, Prime* and *Delay*. In particular, we found significant two-way interactions between: (a) *Group: Prime*: Children with DLD were less likely than TDA children to produce a passive response after hearing a passive prime compared to after hearing an active prime (DLD 12% priming effect vs. TDA 25% priming effect, calculated as the difference in passive production following passive vs. active primes); and (b) *Prime: Delay*: Children were more likely to produce a passive response following a passive prime when the prime immediately preceded the target than when the prime and target were separated by two fillers (no-delay 28% priming effect vs. delay 10% priming effect). However, the three-way interaction of *Group: Prime: Delay* was not significant: The reduction in priming when the prime and target were separated by two fillers did not differ reliably between children with DLD and TDA children.

Follow-up analyses investigating each group separately showed a significant priming effect in the DLD group in the no-delay condition (19%) but not in the delay condition (5%); in contrast, there was a significant priming effect in the TDA group in both the no-delay (39%) and the delay (14%) conditions (Table 5).

Comparison of DLD and TDL groups

Table 4 shows the results of the mixed-effects analyses. There was an effect of *Prime*: Children were more likely to produce a passive structure after hearing a passive prime than after hearing an active prime; an effect of *Delay:* Children were more likely to produce a passive structure immediately after hearing a prime than when two fillers intervened between prime and target; and an interaction of *Prime: Delay*: Children were more likely to produce a passive response following a passive prime when the prime immediately preceded the target than

Table 2. Total frequency of responses in each scoring category by group and condition.

	ACC	ACM	ACU	ARC	PAC	PAM	PAS	PAU	PRC	OOO
Active no-delay										
DLD	72	5	4	1	0	0	0	0	0	2
TDA	77	1	1	0	2	0	0	0	0	3
TDL	76	5	0	0	0	0	0	0	0	3
Passive no-delay										
DLD	54	3	3	0	13	5	0	2	2	2
TDA	42	0	0	0	27	11	1	2	0	1
TDL	49	1	2	0	22	5	0	2	0	3
Active delay										
DLD	68	3	6	0	0	0	0	0	0	4
TDA	78	2	1	0	0	0	0	0	0	3
TDL	76	4	2	0	0	0	0	0	0	2
Passive delay										
DLD	57	10	8	1	2	0	0	0	0	4
TDA	66	1	0	0	11	3	1	0	0	2
TDL	69	4	1	0	6	3	0	0	0	1

ACC: complete correct active; ACM: complete active with morphological error; ACU: complete active without aspect auxiliary; ARC: complete reversed active; PAC: complete correct passive; PCM: complete passive with morphological error; PCU: complete passive without aspect auxiliary; PRC: complete reversed passive; OOO: other response (see Appendix for details of coding scheme); DLD: developmental language delay group; TDA: age-matched typically developing group; TDL: language-age-matched typically developing group.

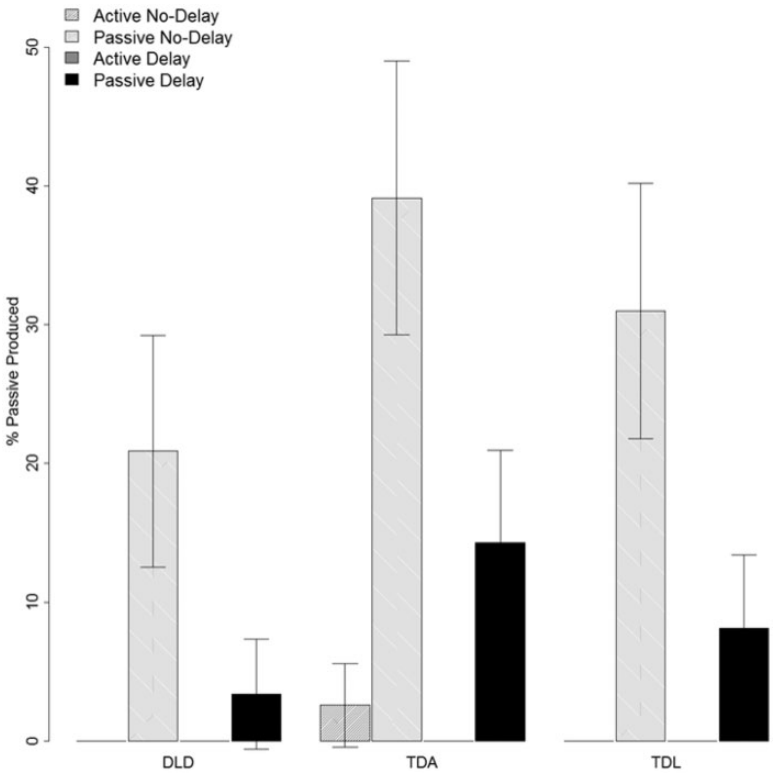

Figure 2. Bar plots, mean and confidence interval of passive target responses as a % of all active/passive target responses by group and condition.

Table 3. Group comparison of passive responses (developmental language delay group (DLD) vs. age-matched typically developing group (TDA)). Coefficients of mixed-effects models for the dependent variable (1/0: produced or not, passive), modeled as a function of the centered and contrast-coded predictors: Prime (non-primed = −.5, Primed = .5), Delay (Delayed = −.5, Non-Delayed = .5), and Group (SLI = −.5, TDA = .5). Our random variables were Child (28) and Item (29). The model was initially fitted with a maximal random-effect structure (i.e. intercepts and random slopes), and a full fixed-effect structure (i.e. main effects and interactions). The table shows the terms that were retained following backward-reduction of this model on both fixed and random effects.

Predictor	β	SE	t
Intercept	0.1	0.01	8.76***
Prime	0.19	0.02	8.5***
Delay	0.11	0.02	4.83***
Group	0.08	0.02	3.37***
Prime: Delay	0.2	0.04	4.41***
Prime: Group	0.13	0.04	2.92**
Delay: Group	0.05	0.04	1.06
Prime: Delay: Group	0.05	0.09	0.52

°$p < 0.1$; *$p < 0.05$; **$p < 0.01$; ***$p < 0.001$.

Table 4. Group comparison of passive responses (developmental language delay group (DLD) vs. language-matched typically developing group (TDL)). Coefficients of mixed-effects models for the dependent variable (1/0: produced or not, passive), modeled as a function of the centered and contrast-coded predictors: Prime (non-primed = −.5, primed = .5), delay (delayed = −.5, non-delayed = .5), and group (SLI = −.5, TDA = .5). Our random variables were Child (28) and Item (29). The model was initially fitted with a maximal random-effect structure (i.e. intercepts and random slopes), and a full fixed-effect structure (i.e. main effects and interactions). The table shows the terms that were retained following backward-reduction of this model on both fixed and random effects.

Predictor	β	SE	t
Intercept	0.08	0.01	5.74***
Prime	0.16	0.03	5.74***
Delay	0.10	0.02	4.20***
Prime: Delay	0.21	0.04	5.28***

°$p < 0.1$; *$p < 0.05$; **$p < 0.01$; ***$p < 0.001$.

when the prime and target were separated by two fillers (no-delay 24% priming effect vs. delay 6% priming effect). However, the final model did not include the *Group* variable: There was no difference between the DLD and TDL groups in their overall production of

Table 5. Coefficients of mixed-effects models with maximal random structure for the number of passives produced as a function of the contrast-coded predictors prime (non-primed = −.5, primed = .5) fitted separately to each group of children (developmental language delay (DLD), language-matched typically developing group (TDL), age-matched typically developing group (TDA)), and delay condition (no-delay, delay). We report the beta coefficient, the t-value, and mark its associated significance using*.

	DLD				TDL				TDA			
	No-delay		delay		No-delay		delay		No-delay		delay	
	β	t	β	t	β	t	β	t	β	t	β	t
Intercept	0.1	3.6**	0.01	1.42	0.14	4.81***	0.03	2.4*	0.2	6.34***	0.07	3.44**
Prime	0.2	3.6**	0.03	1.43	0.3	4.81***	0.08	2.4*	0.37	5.3***	0.14	3.44**

°$p < 0.1$; *$p < 0.05$; **$p < 0.01$; ***$p < 0.001$.

passive responses, likelihood of producing a passive response after hearing a passive prime, or susceptibility to priming when the prime and target were immediately consecutive or separated by two fillers. Follow-up analysis of the TDL group separately showed a significant priming effect in both the no-delay (31%) and delay (8%) conditions (see above for analysis of the DLD group; Table 5).

Assessing the effect of cumulative priming in each group

Table 6 shows the results of further mixed-effects analyses including *Cumulative* as an additional variable, to assess the effect of cumulative experience of passive primes. These analyses confirmed significant effects of *Prime*, *Delay,* and a *Prime*: *Delay* interaction, as in our previous analyses. They did not show a significant effect of *Cumulative* in the final model for any of the groups. However, the final model for both the TDA and TDL groups, but not the DLD group, included *Cumulative*, indicating that this variable significantly improved model fit for both TD groups.

Discussion

In a syntactic priming experiment, we investigated how exposure to syntax affected syntactic production in atypical and typical language development. Previous research has suggested that syntactic priming effects reflect implicit learning of syntactic structure. Children with DLD and groups of age-matched and language-matched TD children described pictures of transitive events after hearing the experimenter describe a different transitive event using an active or passive description on the immediately previous turn, or three turns earlier.

All groups of children showed an overall effect of syntactic experience, displaying a higher likelihood of producing a passive sentence after hearing a passive prime than after hearing an active prime. In all three

Table 6. Cumulative effect: coefficients of mixed-effects models for the dependent variable (1/0: Produced or Not, Passive). Each model is fit independently for each group of children. The dependent variable is modeled as a function of the centered and contrast-coded predictors: prime (non-primed = −.5, primed = .5), delay (delayed = −.5, non-delayed = .5), group (SLI = −.5, TDA = .5), and cumulative (number of passives previously processed). The random variables were Child (28) and Item (29). The model was initially fitted with a maximal random-effect structure (i.e. intercepts and random slopes), and a full fixed-effect structure (i.e. main effects and interactions). The table shows the terms that were retained following backward-reduction of this model on both fixed and random effects.

Group	Predictor	β	SE	t
DLD	Intercept	0.06	0.01	4.57***
	Prime	0.12	0.02	4.54***
	Delay	0.07	0.02	2.82**
	Prime: delay	0.17	0.05	3.14**
TDA	Intercept	0.13	0.01	7.33***
	Prime	0.24	0.03	6.81***
	Delay	0.12	0.03	3.6***
	Cumulative	0.005	0.004	1.2
	Prime: delay	0.21	0.07	2.9**
	Prime: cumulative	0.008	0.008	1.06
	Delay cumulative	0.003	0.008	0.44
	Prime: delay: cumulative	0.004	0.01	0.24
TDL	Intercept	0.09	0.01	5.95***
	Prime	0.19	0.03	6.13***
	Delay	0.11	0.03	3.5***
	Cumulative	0	0.03	0.05
	Prime: delay	0.22	0.06	3.6**
	Prime: cumulative	0.0	0.007	0.06
	Delay cumulative	0.001	0.007	0.17
	Prime: delay: cumulative	0.002	0.01	0.17

°$p < 0.1$; *$p < 0.05$; **$p < 0.01$; ***$p < 0.001$.

groups, this syntactic priming effect was stronger when the prime immediately preceded the target than when it appeared three turns earlier. Critically, children with DLD were not affected by syntactic experience in the same way as age-matched TD children. Specifically, they showed a reduced priming effect – in other words, they were less likely than age-matched controls to produce a passive sentence after hearing a passive prime. There was no difference between the DLD and TDA groups in the rate at which priming reduced over time and intervening material. However, whereas the TDA group still showed facilitation for passives three turns after hearing a passive prime (i.e. after two intervening utterances), the DLD group did not. In contrast, the DLD and TDL groups did not differ significantly in their susceptibility to priming, or in the rate at which priming decayed. Further analyses did not find a significant cumulative effect of syntactic experience in any of the groups: Neither TD nor DLD children showed a significant increase in their likelihood of producing passive responses with increasing exposure to passive primes as the experiment progressed.

Our results cast light on the nature of children with DLD's language impairment. Previous research has suggested that children with DLD have a domain-general deficit in implicit learning mechanisms that are associated with the detection and extraction of abstract structural regularities, but has not directly demonstrated a learning deficit that specifically affects syntax. Our study provides a demonstration of a syntactic learning deficit, by showing that children with DLD are less sensitive than age-matched controls to the syntactic characteristics of the input that they hear. Although our children with DLD were influenced by each syntactic experience, as evidenced by the fact that they showed facilitation for producing passive syntax immediately after comprehending passive syntax, they were influenced to a significantly lesser extent than TD children of the same age.

More interestingly for the implicit learning hypothesis, our pattern of results suggests further that their impairment was primarily associated with reduced initial learning from each syntactic experience, rather than intact initial learning that was undermined by abnormally rapid decay. The effects of prior syntactic experience did not reduce over time/intervening material at a significantly higher rate in the DLD group than the TDA group. However, because the groups differed in their initial learning rates, they showed differences in the persistence of observable priming effects: Whereas TDA children still showed significant priming after two intervening utterances, children with DLD did not. In other words, because children with DLD's initial learning was small, the facilitatory effect of hearing the experimenter's prime utterance decayed entirely within two utterances; in contrast, because TDA children's initial learning was larger, the facilitatory effect of the experimenter's utterance was still influential – albeit to a reduced degree – after two utterances.

We note that this pattern implies that children with DLD may suffer 'double jeopardy' for syntactic learning. Assuming that children learn through the act of producing syntactic structure as well as through the act of comprehending it (whether by strengthening representations or by refining relevant processing mechanisms), an initial syntactic experience can initiate a virtuous circle, whereby a partner's original use of a structure can induce a child to re-use that structure when the opportunity presents itself (i.e. when she wants to express a meaning that is compatible with that structure), and this act in itself yields learning that encourages the subsequent re-use of that structure (and so leads to its further consolidation). Thus for TDA children, each new syntactic experience presents both immediate and on-going opportunities to learn through their own production, as their strong initial learning yields facilitation that endures over at least three utterances. In contrast, each new syntactic experience presents children with DLD with more limited opportunities, both because they are less likely to re-use syntax immediately, and because facilitation does not persist. Thus the implicit learning deficit influences the extent to which children may learn syntax indirectly (via their own productions) as well as directly (via input from others).

In sum, our study suggests that children with DLD show some implicit learning for syntactic structure, but this learning is impaired relative to TD children of the same age. To examine whether our results were confounded by morphological complexity, we carried out additional analyses that included responses involving morphological errors. Interestingly, we found an identical pattern of effects when we used this less restrictive inclusion criterion as when we did not (i.e. analysing only morphologically well-formed responses). Because children with DLD still showed a reduced tendency to produce passives after exposure to a passive even when we ignored morphological accuracy, we can be confident that children with DLD's reduced tendency to produce passive syntax after hearing passive syntax in our study reflects a syntactic impairment rather than simply difficulties in producing passive morphology (Leonard, Miller, Grela, et al., 2000).

In this regard, we note that the children with DLD in the present study and reported in other studies (e.g. Marinis & Saddy, 2013) made relatively few errors in passive morphology. Indeed, they made relatively more errors in active morphology, as did typically developing language-matched controls (see Table 3 for a detailed summary of errors). One possible explanation for this

discrepancy between active and passive structures, with more errors reported for active structures, could lie in the specific characteristics of our prime and target sentences. After hearing a passive prime, children could reuse the inflected auxiliary verb word form (*viene*) that they had heard (and so had been lexically primed) with an appropriate past participle. In contrast, active sentences offered many morphological options for inflection (e.g. progressive or perfective aspect, both of which required the use of an auxiliary verb that was not present in the prime) and correspondingly more opportunities for errors. In support of this, the majority of errors in children with DLD's active responses were auxiliary omissions.

In contrast to the reliable differences that we found between children with DLD and TD children who were matched for age, we found no differences in immediate and persistent priming between children with DLD and TD (younger) children who were matched for language. That is, children with DLD showed an immature profile of performance, compatible with a delay account of their language learning. This pattern is consistent with the claim that children with DLD experience reduced implicit syntactic learning, such that each new experience gives rise to a smaller learning effect than in typical children and hence they behave like younger children with less lifetime language experience. It is also consistent with evidence regarding procedural learning of non-linguistic sequences in children with DLD, which similarly showed the same performance in children with DLD as younger TD children (Hsu & Bishop, 2014).

Although we found evidence for a persistent learning effect (over two intervening utterances) in TD children, none of our groups demonstrated a significant cumulative learning effect over the course of the experiment. This stands in contrast to Garraffa et al. (2015), who found cumulative learning for subject relative clauses in both TD children and children with DLD, with the latter showing a significantly smaller effect. We suggest that the disparity in results may be related to the relative complexity and frequency of the two target structures, with passives being acquired later and used less frequently in Italian than subject relatives. A cumulative learning effect may therefore take longer to manifest for passives, a structure with more competitor alternatives, and our experiment may have been too short to detect such an effect. This factor may have been additionally exacerbated by the small sample size in our study. We note that previous evidence for children's cumulative learning of English passives involved a substantially longer experiment with twice as many experimental items and a substantially larger sample than the current study (Branigan & McLean, 2016).

Our failure to detect a significant cumulative learning effect may therefore reflect both specific characteristics of the Italian passive structure, and a lack of power in our study. Some tentative support for this conclusion comes from our finding that including an index of cumulative exposure to passive primes significantly improved model fit for TD but not DLD children, with TDA children in particular showing a numerical trend towards an increased likelihood of producing a passive response with increasing exposure to passive primes. This evidence is of course only suggestive and must be treated with considerable caution pending further research, but is consistent with the possibility of a cumulative learning effect in TD children that might be detected with greater power. The absence of such an effect in the DLD group is similarly consistent with impaired cumulative learning in children with DLD.

An outstanding question concerns the extent to which children with DLD's performance might have been influenced by impairments in language processing and other abilities, beyond an impairment in implicit learning. Some accounts of syntactic priming assume that immediate priming has both implicit learning and residual activation components (Branigan & McLean, 2016; Hartsuiker et al., 2008; Reitter et al., 2011). In these accounts, immediate priming has a partial source in the transient activation of lexical and syntactic representations that are implicated during processing of the prime sentence. It is therefore possible that children with DLD might have shown reduced immediate priming in part because of difficulties in processing the prime sentence, and an accordingly reduced activation-based contribution to the overall priming effect.

We cannot rule out this possibility, but we note that such activation-based priming is assumed to be very short-lived (in contrast to priming arising from implicit learning; Ferreira & Bock, 2006), and so any activation-based deficit should have resulted in a relatively greater reduction in immediate priming than in persistent priming (i.e. a Prime: Delay: Group interaction); we do not find evidence for this in our study. However, the question of how activation mechanisms might contribute to syntactic priming effects alongside implicit learning remains contentious, and we cannot draw any strong conclusions from our data.

It is also possible that differences in priming between the DLD and TDA groups might reflect differences in the strength with which the relevant syntactic structures were represented. We would expect that children with DLD would have weaker or less stable syntactic representations than TD children, as a consequence of their implicit learning deficit. However, these representations do not seem likely to be in themselves a primary cause of reduced priming (although the absence of the relevant representations altogether would necessarily preclude any priming effect – that is, there is a minimal

level of representation that is required for priming to occur). An increasing body of evidence from both children and adults has shown that weaker or less entrenched representations are in fact more susceptible to priming than stronger or more entrenched representations, as an error-based learning account would predict (Bernolet & Hartsuiker, 2010; Bock, 1986; Branigan & Messenger, 2016; Jaeger & Snider, 2013; Peter et al., 2015; see also Garraffa et al., for relevant evidence from children with DLD).

Finally, children with DLD's performance might have been influenced by difficulties in temporarily maintaining verbal material in memory. Impairments in explicit repetition are characteristic of DLD (as evidenced by our sample's poor performance in an explicit repetition task; see Table 1), so it is possible that our children with DLD showed reduced priming because they were less able to maintain the structure of the prime in memory. Such a possibility is compatible with impaired implicit learning (i.e. implicit learning may be contingent on being able to maintain a representation of the input sufficiently for the relevant structure to be identified and extracted). Our study was not designed to discriminate effects of verbal working memory deficits, but we note that in a previous syntactic priming study, children with DLD showed a considerable disparity between their ability to explicitly repeat sentences and their propensity to implicitly repeat a partner's sentences (Garraffa et al., 2015), suggesting that processing a prime sentence and subsequently reusing its structure in a target response does not involve the same memory demands as, for example, explicit sentence repetition. Moreover, in additional analyses that included non-word repetition performance (a measure of the ability to retain verbal material in short term memory) as a predictor, we found that non-word repetition performance did not predict children with DLD's structural priming effects (though interestingly, it did predict TDA children's priming). These analyses suggest that there was not a direct relationship between children with DLD's ability to maintain verbal material in short term memory and their tendency to re-use a previously heard syntactic structure.[3]

Our results have implications for the development of effective interventions. They show that children with DLD – like TD children – can benefit from exposure to input that contains complex syntactic structure, in ways that support their immediate syntactic production. Concretely, the syntactic structure of a conversational partner's utterance can serve to scaffold children's language use in the next utterance. The morphology of a partner's utterance may also play a scaffolding role. Thus, children with DLD benefit from interactive language use in a dialogue context.

However, the learning effect of each experience is smaller than in TD children, implying that children with DLD require more input to derive the same learning outcome. From a therapeutic perspective, they may therefore benefit most when given intensively repeated exposure to complex structures. Our results further suggest that the rate at which children are repeatedly exposed to structures and given the opportunity to re-use these structures in their own production may be critical. Facilitation for the primed structure is effective only over short timescales. Children may therefore derive the greatest benefit when they are rapidly given the opportunity to re-use the primed structure.

Methodologically, our study shows how syntactic priming paradigms can be used to successfully elicit structures that children would not spontaneously produce, and highlights the potential of these paradigms for discriminating the time-course of syntactic learning and for identifying the optimal format and delivery of language input for successful learning. This potential was identified by Leonard (2001) in his review on the primacy of priming in grammatical learning, where syntactic priming was identified as a facilitator in children with a need for developing and consolidating grammatical skills. A further step forward in applying structural priming for intervention plans would be to calibrate generalization of the learning effect, required for any therapeutic intervention, within a time-based manipulation such as that used in the design of this study.

In conclusion, we have shown that children with DLD are sensitive to syntactic structure in the input in a manner that is consistent with implicit syntactic learning, but that such learning appears to be impaired compared to TD children. In particular, children with DLD appear to learn less from each new syntactic experience, in ways that impact their long-term syntactic development.

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Notes

1. Further analyses that included active and passive responses involving structural or morphological errors (i.e. responses coded as ACM, ACU, PAM or PAU) yielded identical results.
2. Further analyses that included in the cumulativity variable the number of passives previously produced yielded identical results.
3. Analyses for the DLD and TDA groups separately showed that for the DLD children, non-word repetition performance was not a significant predictor of passive production alone, or in interaction with Prime, Delay, or Prime and Delay (all $p > .05$); note further that the relevant coefficients were negative (i.e. that higher likelihood of passive production was numerically (non-significantly) associated with lower non-word repetition performance). For TDA children, non-word repetition performance was not a significant predictor of passive production alone, or in interaction with Prime or Delay (all $p > .05$), but was a significant predictor in interaction with Prime and Delay ($p < .05$).

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Appendix. Coding scheme with examples.

Coding categories	Example	Code
Complete active	*Un cavallo colpisce un pompiere* 'a horse is hitting a fireman' *una fata lecca il maiale* 'a fairy licks a pig' *un cavallo insegue un soldato* 'a horse chased a soldier'	ACC
Active with morphological error: omitted or over-regularized morphology	*Un cavallo colpire un pompiere* 'a horse to-hit a fireman' *un cavallo colpito un pompiere* 'a horse hitted a fireman'	ACM
Active without aspect auxiliary	*E un maiale mangiando la fata* 'and a pig ø eating a fairy'	ACU
Reversed complete active	*Un uomo sta abbracciando una pecora* 'a man is hugging a sheep'	ARC
Complete passive	*Una regina viene baciata da una pecora* 'a queen is being kissed by a sheep'	PAC
Passive with morphological error: omitted or over-regularized morphology	*Un soldato sollevare da un orso* 'a soldier's being holded by a bear'	PAM
Passive without aspect auxiliary	*Una strega graffiata da un elefante* 'a witch ø being grabbed by an elephant'	PAU
Reversed complete passive	*Una mucca viene leccata da un re* 'a cow's being licked by a king'	PRC
Other response	*Il maiale è sopra* 'the pig is on'	OOO