Autism spectrum disorder (ASD) is an increasingly diagnosed developmental disorder in children (Fombonne, 2005; World Health Organisation, 1992). One of the core features of ASD is significantly impaired communication (American Psychiatric Association, 2013) and these pragmatic language deficits, in turn, may impede general learning significantly (Loveland & Tunali-Kotoski, 2005). This literature review will focus on a striking, frequently described, linguistic characteristic of children with ASD (Brock, 2011; Dale & Crane-Thoreson, 1993; Seung, 2007): personal pronoun reversal (PPR).

PPR is characterised by the inverse use of pronouns - usually first (“I”) and second person (“you”). When asking for a biscuit, for example, a child may say “You want a biscuit” (Lombardo & Baron-Cohen, 2010, p. 393). It is assumed that PPR is not caused by a general linguistic impairment, as other aspects of the language of children with ASD are not differently developed to those of neurotypical children (Seung, 2007). This raises the question: what is the exact mechanism causing PPR? Interestingly, while incidence rates of PPR are higher in child populations with ASD (when compared to child populations without a diagnosis of ASD; Lee, Hobson, & Chiat, 1994; Tager-Flusberg, Paul, & Lord, 2005), PPR is not unique to children with ASD. More specifically, there is evidence that young, neurotypical children (e.g. Charney, 1980; Chiat, 1982; Chumak-Horbatsch, 2003; Loveland, 1984), blind children and other non-typically developing children also produce PPR (e.g. Brown, Hobson, Lee, & Stevenson, 1997).

This literature review sought to critically evaluate predominant theories of PPR in children with ASD in light of the available data on PPR. In particular, Kanner’s (1943) behaviourist view of PPR as echolalia, cognitive conceptualisations of PPR as a result of impaired understanding of discourse roles (e.g. Tager-Flusberg, 1994) or due to impaired action-memory (Dunphy-Lelii & Wellmann, 2012) and the psychosocial approach to PPR as resulting from an impaired theory of mind (Boucher, 2003) were examined. These particular theories were chosen, as they each have influenced how scholars have come to think about PPR in ASD, yet, this review argues, all fail to adequately explain the available evidence on PPR.

Kanner’s Original View
The Austro-American psychiatrist Leo Kanner (1943) was the first to provide a systematic description of what is now termed autism spectrum disorder (ASD). In his seminal article, he reported a small number of children (all approximately 5 years of age) with ASD who used personal pronouns in a non-normative, inverse manner. Kanner (1943) originally explained PPR as echolalia: “Personal pronouns are repeated just as heard, with no change to suit the altered situation” (p. 244; my emphasis). Here, echolalia can be understood from a behaviourist perspective as simple imitation (Dale & Crane-Thoreson, 1993; Evans & Demuth, 2011). However, several strands of evidence contradict this. First, Dale and Crane-Thoreson (1993) showed in a sample of 30 children (aged 1 year and 8 months) that imitativeness and PPR rates were negatively correlated. Second, case study evidence suggests that a large proportion of PPR is non-imitative (e.g. Evans & Demuth, 2011). Third, even if echolalia could account for some PPR, one cannot simply assume that the same underli-
ing mechanism drives non-echolalic PPR (Lee et al., 1994). Additionally, declaring PPR a result of imitation does not explain why children with ASD might imitate personal pronouns as heard (Brock, 2011). While there are some suggestions that echolalia may have discursive functions (Evans & Demuth, 2011), these too fail to explain why it occurs in children with ASD to begin with. In sum, the behaviouristic notion of PPR as simple imitation can only explain a small proportion of the PPR evidence reported in the literature.

Cognitive Theories of PPR

With the declining predominance of behaviourism in psychology since the 1970s, explanations of PPR in terms of simple imitation have been increasingly rejected (Lee et al., 1994) in favour of conceptualisations of PPR as a specific cognitive impairment. What unites different cognitive theories of PPR is an understanding of PPR as a lack of competence to fully understand the relation between self and others (Evans & Demuth, 2011). This, in turn, is assumed to be interconnected with the wider social communicative problems commonly associated with ASD (Volkmar & Klin, 2005). According to discourse-role theory, the predominant cognitive perspective on PPR, what makes pronouns so complex and demanding is their deictic nature (Lee et al., 1994). That is, the correct use of pronouns, unlike names, depends on who produces an utterance and to whom it is addressed. Thus, children with ASD are assumed to produce PPR because they are either impaired in, or lack an understanding of, the different discourse roles that a conversational context affords (Tager-Flusberg, 1994). Discourse-role theory leads to two main hypotheses: children may reverse pronouns because they (a) fail to comprehend discourse roles and/or (b) have a deficit in pronoun production/fail to perform deictic shifting.

Understanding of Discourse Roles

When conceptualising PPR as a failure to comprehend discourse roles, one would expect that children with ASD should misunderstand statements whose correct understanding relies on pronouns. However, Lee et al. (1994) found little evidence of errors in pronoun comprehension in children and adolescents with ASD, despite anecdotal evidence of everyday errors in pronoun use in the same participants. In a lab-based experiment comparing a total of 50 children (mean age 15 years and 7 months) with and without ASD (matched for both chronological and mental verbal age), they found that children with ASD were “near-perfect” (ibid.) in answering questions such as “What are you wearing/what am I wearing?” Similarly, they could correctly point to a photograph of either themselves or the experimenter respectively, when instructed to “Point to the picture of...” – demonstrating a good understanding of discourse roles. In a similar piece of research, Jordan (1989) compared pronoun comprehension of 3 groups of 11 children (aged 3 to 10 years) matched for mental verbal age: one group with ASD, one with intellectual disabilities and one of neurotypical children. He found no impairment in pronoun comprehension in children with ASD compared to the other two control groups when asked to follow requests such as “Make the doll kiss you/me”, strongly illustrating that PPR in children with ASD is unlikely to be caused by an impaired understanding of discourse roles.

Findings from research into the relationship between spatial versus linguistic perspective-taking, however, appear to contradict this conclusion. In both cross-sectional and longitudinal studies, Loveland (1984) demonstrated that within a group of 27 children (aged 2 years to 3 years and 3 months), the relationship between the ability of spatial perspective-taking was strongly related to the correct comprehension of pronouns and vice versa. That is, only children who demonstrated a good understanding of the spatial implications of different discourse roles (i.e. “what somebody else sees may be different to what I can see”) understood personal pronouns completely. Similar results were found by Ricard, Girouard, and Décarie (1999) in a study of 12 English-speaking and 12 French-speaking children, aged 1 year and 6 months.

At first, these findings seem to contradict the above-cited evidence by Jordan (1989) and Lee et al. (1994). Importantly however, the children participating in the latter studies were diagnosed with ASD – which was not the case for the children in Loveland’s (1984) or Ricard et al.’s (1999) samples. It may thus be the case that PPR in children with ASD is caused by a different underlying mechanism/deficit than in neurotypical children (Dale & Crain-Thoreson, 1993; Tager-Flusberg et al., 2005). In sum, while there is some evidence for PPR as impaired comprehension of discourse roles in the general child population, there is no evidence suggesting children with ASD actually fail to understand discourse roles as indicated by pronouns.

Shifting Between Discourse Roles

Alternatively, PPR may be due to a problem in performing deictic shifting (Evans & Demuth, 2011). Impairment in deictic shifting can be framed as the failure to apply the above-discussed understanding of discourse roles and thus shift the perspective from listener to speaker when producing speech accordingly. Supporting this notion, a single-subject study found PPR production to be common in a 4-year old girl with ASD with 76% of pronouns reversed (Olive, Lang, & Davis, 2008). However, of the only two published systematic studies of PPR production, both have found PPR to be significantly less common. Tager-Flusberg (1994) recorded six children with ASD aged 3–9 years interacting with their mothers at home over the course of 12–26 months. While this study found that all children produced reversed pronouns, they did so only in 13% of all pronouns produced. Similarly, Lee and colleagues (1994) showed experimentally that only a minority of pronouns (13%) were reversed by children with ASD when answering questions, with correctly used pronouns frequently occurring alongside these. Thus, while there is some evidence of common PPR in speech production in children with ASD, it appears to only occur in a minor-
ity of all pronouns produced. The apparent inconsistent nature of the PPR evidence necessitates further research into the specific conditions under which PPR occurs. In sum, the discourse-role theory of PPR lacks support from empirical research regarding both pronoun comprehension and pronoun production of children with ASD.

**Ultimate Explanations for PPR**

In addition to the lack of empirical support, discourse-role theory of PPR can be criticised for failing to address the core of the problem. Discourse-role theory can be described as (theoretically) accounting for the *proxy cause* of PPR, that is, the mechanism of *how* PPR occurs. However, it fails to account for the *ultimate reasons* behind PPR, that is, *why* it occurs (Scott-Philip, Dickins, & West, 2011). Over the decades, different accounts for PPR have been proposed; however, their common denominator is the suggestion of a fundamentally impaired self in children with ASD. In his seminal article on ASD, Kanner (1943) himself suggested that the children studied by him lacked a coherent sense of self. Psychoanalytic theorists suggested that children with ASD may have a fundamentally impaired or even absent ego, with Bettelheim (1967) referring to the autistic child’s self as an “empty fortress”. Children with ASD who do not refer to themselves as “I” (as a result of PPR), classical psychoanalytic theory argues, are seen as employing an ego defence mechanism (Lee et al., 1994). More modern psychoanalytic theorists have proposed that PPR in children with ASD is a result of their impairment in identifying with others (Hobson, 2010), also dubbed “interpersonal relatedness” (Hobson, García-Pérez, & Lee, 2010). However, in psychology the currently predominant perspective regarding the ultimate cause for PPR is impairment of the so-called theory of mind (ToM) in children with ASD (Boucher, 2003).

A ToM perspective on PPR essentially suggests that the social and communicative impairments of ASD are mutually connected (Tager-Flusberg, 1999) through a lack of the cognitive domain that is ToM (Belkadi, 2006). The appeal of ToM as an ultimate explanation of PPR lies in its potential explanatory power, as it is also the dominant theory explaining the wider behavioural, cognitive and affective features associated with ASD (Boucher, 2003). ToM is a *meta-representational theory* (Smith, Cowie, & Blades, 2003) proposing that children have to learn that other people have minds and that these are, in turn, separate and different to theirs (Baron-Cohen, Leslie, & Frith, 1985; Tager-Flusberg, 2000). Thus, a failure to develop a (full) ToM means a lack in awareness and understanding of the difference between (one’s) self and (that of) others (Hobson, 2010). This is in line with the predictions of the discourse-role theory of PPR.

Many psychologists have explained their PPR findings in terms of an “impairment” of their participants’ ToM (e.g. Hobson, 2010; Tager-Flusberg, 1999). However, ToM itself is heavily contested as a concept (Belkadi, 2006, but see also Loveland, 2001; Tager-Flusberg, 2011; Verhaeghe, 2004). The primary issue for the ToM perspective on PPR is its claim of universality. While some evidence indicates that PPR occurs in all children with ASD (Tager-Flusberg et al., 2005), if the underlying deficits were as pervasive as the assumed impairment (or even lack) of ToM in children with ASD, arguably a 100% PPR rate would be expected. Whichever ultimate theory is proposed to explain PPR, it has to account for its occurrence in only a minority of pronouns produced in the face of apparent unimpaired pronoun comprehension.

Recent research suggests an alternative to the ToM perspective on PPR. In a study comparing typically-developing 3- and 5-year-olds with 7-year-old children with ASD, Dunphy-Lelii and Wellman (2012) suggested that PPR in children with ASD may be due to an impairment in memory for actions in a way that makes it difficult for children with ASD to “track the self versus another” (p. 221). This action-memory deficit could account for the unequal impairment regarding pronoun production versus pronoun comprehension: memory problems would not be expected to impact on a child’s ability to understand discourse roles in an utterance, but could still disrupt correct pronoun production. However, this is both (a) a very recent suggestion and thus, at the time of writing, under-explored; and (b) cannot account, like ToM, for the fact that pronoun production is only impaired in the minority of pronouns produced. In sum, both the ToM and the action-memory theory of PPR fail to convincingly explain the pattern of PPR evidence available.

**Criticisms of the Cognitive Theories of PPR**

While this literature review so far has offered some criticism of specific PPR research studies and theories, there are wider issues in this body of research concerning metatheory, theory, and methodology. These shortcomings in the PPR literature not only call for caution when interpreting existing evidence, but also highlight the need for a more complex, contextual approach to the study of PPR.

**Theoretical Critique**

On a theoretical level, the exact nature of ASD itself is still a highly contentious topic, which has obvious implications for the study of PPR. Most (clinical) psychologists understand ASD to be a “valid and well-established diagnostic category” (Volkmar & Klin, 2005, p. 5). However, the behavioural heterogeneity witnessed in people with ASD makes it difficult to assume a reified disorder, even if conceptualised as a spectrum (Hyman, 2010; Waterhouse, 2013). That is, the behaviour displayed by people diagnosed with ASD may be so vastly different, even in terms of the diagnostic *core features* of ASD, that it appears difficult to justify the assumption of a common, underlying disorder. Similarly, even when two persons with ASD display the same characteristics at a certain age, these may develop very differently over time (Gschwind & Levitt, 2007). Moreover, while today, all persons with autistic traits are classified under the diagnostic label “ASD”, a number of sub-types of ASD (e.g. Asperger’s Syndrome) exist. However, there is no consensus within the literature as to whether these subtypes differ in their use of pronouns. Thus, the generalisability of the above-discussed
Methodological Critique

The low methodological quality of some of the existing evidence in this field presents further problems. Although there are a number of clinical reports of PPR available (e.g. Kanner, 1943; Ricks & Wing, 1975), there are few experimental studies (Hobson et al., 2010). Of these, many did not control for the presence of ASD (e.g. Dale & Crain-Thoreson, 1993) when investigating PPR, while others did not have appropriate control groups (e.g. Olive et al., 2008), limiting the validity of the available evidence. Furthermore, longitudinal studies, which could elucidate the development of PPR in children with ASD over the course of time, are almost completely absent from this field of research (Evans & Demuth, 2011). In the context of the heterogeneous nature of ASD, a crucial observation emerges: the inconclusive nature of the existing PPR research and theory may be a direct result of the heterogeneous nature of the population studied. Phrased differently, there may be a number of different mechanisms underlying PPR, and thus individuals assumed to share a common deficit may actually have different deficiencies (Tager-Flusberg et al., 2005). Thus, drawing conclusions based on the available evidence appears unwise as PPR effects reported in a study may not apply to all its participants, let alone generalise beyond the sample studied.

Meta-theoretical Critique

The literature on PPR is also plagued with problems on a meta-theoretical level: all studies cited so far assume an organismic model of development, which assumes human development to denote an actively developing individual in a passive environment (Lerner, 2002). Yet, for more than 20 years, evidence in favour of the competing contextual perspective of development has been mounting (Lerner, Theokas, & Bobek, 2005). In this perspective, development is understood to be a reciprocal, multilevel, non-linear relationship between the person and their environment (Bronfenbrenner & Morris, 2006; Lerner et al., 2010).

Consequently, human development must be understood through an analysis of the cultural, social, political and economic systems a person is immersed in rather than focussing on the behaviour of a single “individual” disconnected from their environment. That is, cognition and context must be regarded as inseparable (Liverta-Sernpio & Marchetti, 1997). Taking a contextual perspective means understanding phenomena as constantly changing processes embedded in a constantly changing world (Lerner, 2002). Such an understanding of human development then necessitates scientific enquiry to focus on the temporal and relational nature of a phenomenon such as PPR (Lerner et al., 2010). A contextual approach could thus account for change and (temporal) inconsistency in a phenomenon, as evidenced in the above-cited literature on PPR.

Particularly problematic in the extant PPR literature is the focus on psychological factors at the expense of contextual factors. However, the social and linguistic environment of children with ASD is likely to be dissimilar from that of typically developing children (Tager-Flusberg et al., 2005). Studies have shown that parents of children with ASD adapt the complexity and style of the language they use to communicate with their children to their perceived linguistic capabilities (Konstantareas, Zajdeman, Homatidis, & McCabe, 1988; Venuti, de Falco, Esposito, Zaninelli, & Bornstein, 2012). There also appears to be a relationship between the complexity of language parents employ when talking to their children with ASD and the latter’s linguistic development (e.g. Goodwin, Fein, & Naigles, in press). This strand of evidence suggests a potentially strong influence of contextual variables on the linguistic development of children with ASD, lending further support to calls for an ecological perspective on ASD in general and on PPR in particular, with a focus not on the individual but on the person-environment system (Loveland, 2001). Taking such an ecological perspective would mean considering that the environment may not typically provide a good model for correct pronoun use, precisely because of their deictic nature (Evans & Demuth, 2011). Combined with Oshima-Takane and Benaroye’s (1989) finding that children with ASD may fail to attend to personal pronouns when observing speech between third parties, this could then provide a truly social explanation of PPR.

Taking a contextual perspective would also mean studying the “organism in relation” (Looff, 1973 as cited in Lerner, 2002, p. 72), i.e. the organism’s relationships, rather than the organism itself. Thus, the utility of the predominant, organismic approach to PPR (i.e. the conceptualisation of PPR as a “deficit” in the competence to use pronouns “correctly”) seems especially questionable given that (a) even neurotypical children may occasionally display PPR (Tager-Flusberg et al., 2005) and (b) children with ASD do not produce PPR consistently or frequently (Tager-Flusberg, 1994). A cognitive or linguistic deficit alone cannot account for the occurrence of PPR (Charney, 1980), thus necessitating contextual, longitudinal research into the trajectory of PPR as a relational phenomenon. In sum, a critique of the existing PPR research and theory from a contextual perspective highlights an unwarranted understanding of PPR as an individualised psychological deficit.

Conclusion

To date, PPR has been described as one of the key features of ASD (Seung, 2007). While good-quality, systematic, longitudinal and naturalistic research is lacking in this field,
the existing evidence suggests that PPR may be common in children with ASD, yet infrequent and thus far from being a key feature (Evans & Demuth, 2011). The (meta-)theoretical shortcomings of the prevailing organismic research and theory in this field have failed to adequately explain the pattern of PPR evidence. Additionally, it has contributed to a culture of individualised deficit-thinking associated with PPR while largely ignoring contextual factors and explanations. Thus, the field requires new, more complex contextual theories of PPR that can elucidate the conditions under which PPR occurs.

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