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Temporal and diagnostic influences on the expression of comorbid psychopathology symptoms in infants and toddlers with Autism Spectrum Disorder

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ABSTRACT

In an attempt to increase the specificity and sensitivity the diagnostic criteria for ASD has been recently modified. Aside from the impact the diagnostic rates, these changes also have implications for the study of comorbid symptoms in the ASD population. As we refine the criteria for the ASD category we must also seek to improve our understanding of the manifestation of comorbid psychopathology within ASD populations. The current study sought to examine diagnostic and temporal influences on the expression of comorbid psychopathology symptoms in 205 infants and toddlers ranging in age from 17 to 37 months. Participants were separated into two groups based on their diagnoses (i.e., Autism Spectrum Disorder [ASD] and atypical development without an ASD). The *BISCUIT-Part 2* was administered on two separate occasions, with the initial and follow up assessment occurring within one of two time intervals (4–8 months, or 9–13 months). Results from the current study indicate that the time between initial and follow up assessments is a significant factor influencing symptom expression. In addition to the temporal influence, it was observed that children diagnosed with ASD exhibit significantly less stable symptoms of comorbid psychopathology. Implications of these findings are discussed.

The neurodevelopmental origin of Autism Spectrum Disorder (ASD; Matson & Kozlowski, 2011; Rivet & Matson, 2011; Weinkauf, Zeug, Anderson, & Ala'i-Rosales, 2011) is supported by research which suggests irregular brain activity in regions related to social interactions (i.e., mirror neurons; McPartland, Coffman, & Pelphrey, 2011). While this abnormality has been proposed as a possible genetic marker for ASD, the presence of an ASD is still diagnosed based upon deficits in three core areas: social interaction, communication skills, and restricted and repetitive behaviors (Fodstad, Matson, Hess, & Neal, 2009; Matson & Wilkins, 2009; Rivet & Matson, 2011; Weinkauf et al., 2011; Xianchen, Hubbard, Fabes, & Adam, 2006). However, recent diagnostic revisions in the DSM-5 included changes to the ASD subtypes, content, and symptom structure which maintained only two core areas of impairment (for in-depth reviews of diagnostic changes please see, Grzadzinski, Huerta, & Lord, 2013; Mahjouri & Lord, 2012). Researchers have repeatedly demonstrated that comorbid psychopathology and challenging behaviors are also often present in ASD populations (Bakken et al., 2010; Davis et al., 2010; Funabiki, Kawagishi, Uwatoko, Yoshimura, & Murai, 2011; Horovitz et al., 2011; Matson & Neal, 2009; Simonoff et al., 2008).

Although comorbid diagnoses were not prevalent in ASD populations historically, this was not due to the absence of symptomology (Lainhart, 1999; Matson & LoVullo, 2009). Rutter (1968) suggested that the ambiguity of diagnostic criteria for ASD was a contributing factor, while others have argued that the unique presentation of psychopathological symptoms in individuals with ASD makes differential diagnosis increasingly more difficult (Gillberg, 2010). Regardless of previous

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causation, researchers have compiled evidence of comorbid psychopathology symptoms and their impact in ASD populations (Bakken et al., 2010; Gillberg, 2010; Hayashida, Anderson, Paparella, Freeman, & Forness, 2010; LoVullo & Matson, 2009; Matson & Minshawi, 2006; Matson & Nebel-Schwalm, 2007; Nebel-Schwalm & Matson, 2008; Smith & Matson, 2010a; Smith & Matson, 2010b; Smith & Matson, 2010c). Not only has the presence of comorbid symptoms been confirmed in ASD, but multiple researchers have demonstrated that they occur at significantly higher rates in ASD groups and are often targeted for intervention (Barthélémy et al., 1992; Dawson, Matson, & Cherry, 1998; Fodstad, Rojahn, & Matson, 2010; Gillberg & Billstedt, 2000; Hess, Matson, & Dixon, 2010; Holden & Gitlesen, 2007; Leyfer et al., 2006; Matson & Nebel-Schwalm, 2007; Matson, Fodstad, Mahan, & Sevin, 2009b; Paclawskyj, Matson, Bamburg, & Baglio, 1997; Tsai, 1996).

Not only does the presence of comorbid psychopathology symptoms complicate diagnosis, it also impacts the development and provision of treatment (Matson & Minshawi, 2007; Matson & Nebel-Schwalm, 2007). These complications impact the individual receiving treatment as well as those involved in treatment implementation (Gray, Ansell, Baird, & Parr, 2011; Matson, Dempsey, & Fodstad, 2009). Highly individualized treatment plans are often necessary to address comorbid symptoms in an individual with an ASD (LoVullo & Matson, 2009). The implementation of additional interventions for some comorbid disorders such as psychotropic medication, may be used in conjunction with the behavioral interventions frequently used to address symptoms of ASD (LoVullo & Matson, 2009; Self, Hale, & Crumrine, 2010).

Until recently the focus on early identification and the use of empirically supported interventions for young children with ASD has been hindered by a relative lack of appropriate assessments. Further, few assessments with a normative ASD group have been developed to asses for symptoms of comorbid psychopathology. While the Child *Behavior Checklist for ages 1.5–5* (*CBCL*; Achenbach & Rescorla, 2000) and *Behavioral Assessment System for Children-Second Edition* (*BASC-2*; Reynolds & Kamphaus, 1992) both assess for comorbid symptomology, they do not include a normative ASD group (Matson & Sipes, 2010). The *BASC-2* and *CBCL* are also of limited utility in the early identification of infantile ASD populations due to their normative age range. Matson and colleagues (2010) identified these limitations as contributing factors to our limited knowledge of comorbid symptom manifestation in children with ASD.

With the development of the *Baby and Infant Screen for Children with aUtIsm Traits-Part 2* (*BISCUIT-Part* 2) Matson, Boisjoli, Hess, and Wilkins (2011) provided researchers and clinicians with an empirically validated measure of comorbid symptoms in young children with ASD and atypical development. In the current study, assessment of comorbidity in infants and toddlers with and without ASD was carried out utilizing two separate administrations of the *BISCUIT-Part 2*.

The term "comorbid" within the context of this article is in reference to the presence of ASD and one or more co-occurring psychiatric symptoms within the same individual. The current study sought to analyze the diagnostic and temporal influences on comorbid symptom expression. Comorbid symptom stability was compared across participants falling into two diagnostic categories (i.e., Autism Spectrum Disorder [ASD] and atypically developing without an ASD [atypically developing]) in order to determine if diagnosis significantly predicts concomitant psychopathological expression. In addition to diagnostic influence, temporal influences were also examined for each diagnostic category by inspecting the time interval between initial and follow up assessment (i.e., 4–8 months, and 9–13 months). Symptoms were considered "stable" if they did not differ significantly between the initial and follow up assessment periods.

1. Method

1.1. Participants

The current study included 205 infants and toddlers of which 78% were male. The average age was 23 months, (see Table 1 for demographic information). Participants were part of a state-wide screening program which provides services to children and their families when an infant or toddlers (up to 36 months of age) identified with a developmental delay or a medical condition, placing them at risk for a developmental delay.

Table 1
Participant demographics.

	ASD	Atypical	Total sample
	n = 67	*n* = 138	*N* = 205
Age in months (SD)	23.09 (3.72)	23.33 (3.56)	23.26 (3.86)
Gender			
Male	77.6%	78.3%	78%
Female	22.4%	21.7%	22%
Ethnicity			
Caucasian	44.8%	50.0%	48.3%
African–American	44.8%	41.3%	42.4%
Hispanic	3.0%	5.8%	4.9%
Other/unspecified	7.5%	2.9%	4.4%

Note: SD, standard deviation.