

Profiles of Internalizing Symptomatology and Social Motivation in Youth with ASD
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ABSTRACT

Although clinically significant symptoms of anxiety or depression are present in nearly one in two people with autism spectrum disorder (ASD), little is known about how these symptoms may relate to social motivation, a key construct in the etiology of ASD. The aim of the current study was to examine patterns of anxiety, depression, ASD symptomatology, and social motivation in youth with ASD. Using a large public dataset of well-characterized youth with ASD ($N = 195$), we examined varying patterns of these symptoms via latent profile analysis (LPA). Three distinct classes emerged: one with moderate ASD severity and low levels of comorbid psychopathology, a second with more severe ASD symptoms and similarly low anxiety and depression, and a third with significantly elevated anxiety and depression. Neither sex nor age differed significantly among these classes, and indices of social interest early in development did not predict class membership. Implications of these symptom patterns for assessment and treatment of comorbidity in ASD are discussed.

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GENERAL AUDIENCE ABSTRACT

A large proportion of individuals with autism spectrum disorder (ASD) experience symptoms of anxiety and depression. Anxiety and depression might be related to social motivation, which is a key concept associated with the development of ASD. The current study examined variation in anxiety, depression, social motivation, and ASD symptom severity in a large sample of youth with ASD. Individuals in the sample were classified in groups by a statistical model. Models produced three separate groups: one group with moderate symptoms of ASD and relatively low levels of anxiety and depression, a second with more severe ASD symptoms and similarly low anxiety and depression, and a final group with moderate ASD symptoms and high anxiety and depression. Social motivation was strongest in the first and second groups, and low social motivation was strongly associated with anxiety and depression symptoms. Age and sex did not predict group membership, nor did items measuring social motivation early in development. Implications of these symptom patterns for the assessment and treatment of anxiety and depression in ASD are discussed.

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Profiles of Internalizing Symptomatology and Social Motivation in Youth with ASD

Children and adolescents with ASD frequently suffer from a host of comorbid psychiatric diagnoses, including anxiety disorders, attention-deficit/hyperactivity disorder (ADHD), depressive disorders, and oppositional defiant disorder (Simonoff et al., 2008). A substantial proportion of individuals with ASD meet diagnostic criteria for two or more of these comorbid diagnoses (Simonoff et al., 2008). Although a considerable body of work has explored the presentation of externalizing disorders such as ADHD and oppositional defiant disorder (Gadow, DeVincent, & Drabick, 2008; Lichtenstein, Carlström, Råstam, Gillberg, & Anckarsäter, 2010), the majority of literature on comorbidity has focused on internalizing symptomatology. Internalizing problems, including both anxiety and depression, are the most prevalent category of comorbidity in ASD, limit functional independence and quality of life, persist across developmental stages, and adversely impact outcomes in adulthood (Howlin, Goode, Hutton, & Rutter, 2004; Lugnegård, Hallerback, & Gillberg, 2011; White, Oswald, Ollendick, & Scahill, 2009).

Anxiety in ASD

A rich body of literature has explored the manifestation of anxiety in individuals with ASD, including evaluations of prevalence, underlying mechanisms, and treatment effectiveness. Prevalence estimates vary based across person-level variables including chronological age, severity of ASD symptoms, and cognitive ability, as well as assessment methodology, but reviews and meta-analyses have estimated that 40-60% of individuals with ASD meet diagnostic criteria for one or more anxiety disorders (Lugnegård, et al., 2011; Maddox & White, 2015; van Steensel, Bögels, & Perrin, 2011; White et al., 2009). More specifically, studies suggest that social anxiety, generalized anxiety, and specific phobia are highly prevalent as compared to rates

seen in typically developing populations. In one study, almost one third of children and adolescents presented with specific phobias, while approximately 17% demonstrated significant symptoms of social anxiety and another 17% may have met criteria for obsessive-compulsive disorder (which at the time of publication remained categorized as an anxiety disorder; van Steensel et al., 2011). Another study reported the greatest prevalence for both social anxiety (over 20%) and generalized anxiety disorder (over 20%) in a sample of adults with ASD, with lower figures for panic disorder, agoraphobia, and OCD (Lugnegård et al., 2011). In contrast to these studies suggesting approximately 50% of participants present with *any* anxiety disorder, another prevalence figure established based on clinical interviews for social anxiety disorder reported a prevalence figure of 50% for social anxiety alone (Maddox & White, 2015). Elevated rates of anxiety disorders in adults may be indicative of unremitting anxiety symptomatology that first emerged in childhood or adolescence.

The elevated rates of anxiety seen in individuals with ASD can be attributed to a variety of risk factors, many of which are associated with core ASD symptoms. For example, difficulty with perspective-taking and effective social communication frequently contribute to poor outcomes in social encounters. Indeed, evidence suggests the vast majority of youth with ASD are frequent victims of peer victimization and bullying (Sreckovic, Brunsting, & Able, 2014). The development of significant anxiety symptoms may therefore follow the development of realistic expectations that attempts at peer interactions will be unsuccessful (Mazefsky & Herrington, 2014; Wood & Gadow, 2010). However, development of such an expectation and associated anxiety requires some degree of insight into the fact that one is being evaluated. Such perspective-taking or “theory of mind” is understood to be impaired in individuals with ASD (Hamilton, Brindley, & Frith, 2009). Atypical forms of social anxiety in ASD may therefore

develop as a product of worries that are more diffuse (Kerns & Kendall, 2012). For individuals whose perspective-taking skills are insufficient to develop the fear of negative evaluation that is a hallmark of social anxiety, a broader worry may develop based on the ambiguity and uncertainty associated with social interactions.

Restricted and repetitive behaviors (RRBs), the second core symptom domain of ASD, overlap substantially with symptoms of anxiety. Insistence on sameness, including inflexibility in adherence to routines, is a diagnostic criterion of ASD and relates closely to symptoms of generalized anxiety such as excessive worry about the future and preoccupation with small changes and past events (Kerns et al., 2014). Additionally, compulsive rituals and rules that must be followed with great specificity bear resemblance to obsessive-compulsive symptoms (Kerns et al., 2014). Within the category of RRBs, hyper- or hypo-reactivity to sensory input may confer additional risk for anxiety disorders. These sensory differences can render everyday experiences (e.g., a school fire drill or hearing a vacuum cleaner) aversive for many individuals with ASD. Given the distress associated with these experiences and pervasive worry that one might encounter them unexpectedly, reactivity to sensory stimuli may place youth with ASD at risk for the development of specific phobias (Muskett, Radtke, White, & Ollendick, 2019).

Factors beyond core diagnostic criteria for ASD also contribute to elevated risk for anxiety disorders in this population. Emotion regulation, referring to one's ability to modulate emotions in order to engage in goal-directed behavior, is often impaired for individuals with ASD. These common deficits in emotion regulation ability are strongly associated with the emergence of anxiety disorders. Neural, physiological, and behavioral evidence suggests that individuals with ASD more readily experience hyperarousal and demonstrate diminished capability to engage regulatory processes as compared to typically developing samples (White et

al., 2014). These processes, when paired with cognitive rigidity, information processing differences, and sensory issues associated with ASD, represent one likely pathway to the emergence of anxiety disorders in individuals with ASD (Mazefsky et al., 2013; Rieffe et al., 2011).

Depression in ASD

In recent years, comorbidity literature has seen an increased focus on depressive symptoms. Though one review (Wigham, Barton, Parr, & Rodgers, 2017) identified only 19 articles reporting prevalence figures for depression, a more recent meta-analysis focused on adults with ASD reported prevalence rates derived from 66 articles (Hudson, Hall, & Harkness, 2019). Authors reported lifetime and current prevalence rates of 14.4% and 12.3%, respectively. Age, IQ, and percentage of white participants in the sample were each correlated with increased prevalence of depressive disorders (Hudson et al., 2019). Hollocks and colleagues conducted a meta-analysis examining symptoms of both anxiety and depression, and reported current and lifetime pooled estimates of 23% and 37% for depression, respectively (Hollocks, Lerh, Magiati, Meiser-Stedman, & Brugha, 2019). Both of these studies reported significant heterogeneity in terms of methodology and diagnostic tools used in included studies. This finding is consistent with extant literature reporting limited evidence of validity for depression assessment tools developed and normed on typically developing samples (Gotham, Unruh, & Lord, 2015; Hollocks et al., 2019; Hudson et al., 2019; Stewart, Barnard, Pearson, Hasan, & O'Brien, 2006).

Given the potential impact of depression throughout the lifespan for individuals with ASD (Gotham, Brunwasser, & Lord, 2015), further research into its underlying social and cognitive factors throughout the lifespan remains an urgent need. In typically developing populations, depression is under-diagnosed, contributing to a greater public health problem

because of its impacts on medical conditions such as arthritis, asthma, and diabetes (Cassano & Fava, 2002; Moussavi & Chatterji, 2007). Although no research, to our knowledge, has examined functional impairment associated with depression in youth with ASD, depressive symptoms occurring prior to adulthood are associated with later problems in employment, quality of life, and other indicators of outcome for adults with ASD (Howlin et al., 2004).

Models of Anxiety and Depression

Although depression and anxiety are both prevalent among samples of youth with ASD, existing theoretical models of comorbid anxiety and depression have not yet been thoroughly considered as they may apply to ASD samples. Several etiological frameworks for comorbid anxiety and depression (independently of ASD) were presented in a review on the topic (Cummings, Caporino, & Kendall, 2014). Among the considered frameworks was the tripartite model, which posits that negative affect is a common component of both anxiety and depression, with unique factors of physiological hyperarousal in anxiety and low positive affect in depression (Clark & Watson, 1991). The authors reported that empirical evidence for the tripartite model is mixed, in part due to the heterogeneity of anxiety disorders and in part due to varying results throughout the developmental trajectory (Brown, Chorpita, & Barlow, 1998). Evidence for the behavioral inhibition/behavioral activation (BIS/BAS) approach to comorbid anxiety and depression was also reviewed, with the authors concluding that overlap is present between the tripartite model and the BIS/BAS model, and that the latter merits further study (Cummings et al., 2014). Finally, the authors proposed that a number of pathways may predict the development of comorbid anxiety and depression in typically developing youth, in keeping with the developmental psychopathology concept of equifinality (Cicchetti & Rogosch, 1996). Specifically, the authors proposed that three pathways may be present: one in which youths

present with a predisposition for anxiety, with depression developing later as a product of impairment from anxiety symptoms; one consisting of a shared diathesis for depression and anxiety resulting in concurrent emergence of disorders; and a third pathway in which anxiety may occur as the result of depressive symptoms (Cummings et al., 2014). With regard to specific disorders, evidence has suggested that social anxiety may be linked to depression, and that depression may exacerbate the negative cognitive biases that are already present in social anxiety disorder (Cummings et al., 2014). This final point may be of particular relevance for individuals with ASD, given that they may be more prone to experience and develop cognitive biases due to their social communication deficits (Wood & Gadow, 2010).

Social Motivation and Comorbidity

Although the above models of comorbid anxiety and depression as developed in typically developing populations may be applied to individuals with ASD, alternative ASD-specific models may take into consideration core characteristics of that disorder. Namely, another pathway to depression and social anxiety in ASD may lead through the interaction of social communication deficits and motivation to engage in social interactions. This relationship may be understood through the framework of social motivation theory. Social motivation theory suggests that infants who ultimately develop autistic symptoms demonstrate diminished interest and motivation to engage in social stimuli (e.g., faces, biological motion patterns) early in development. By failing to attend to these stimuli, infants do not develop the specialized neural mechanisms necessary to quickly and efficiently process social information, which ultimately leads to the symptoms seen in ASD (Chevallier, Kohls, Troiani, Brodtkin, & Schultz, 2012).

Social motivation theory is well-supported by an extensive body of literature using EEG, fMRI, and eye-tracking. Collectively, this body of research suggests that infants who go on to

develop ASD attend less to social stimuli than typically developing infants (Klin, Jones, Schultz, Volkmar, & Cohen, 2002). However, more recent work has questioned whether behavioral indices of “social motivation” such as eye-tracking accurately characterize an individual’s true desire to engage in social interaction. Evidence from other behavioral tasks and self-report instruments has suggested that individuals with ASD may demonstrate more social interest—or social motivation—than previously thought (Deckers, Muris, & Roelofs, 2017). Individuals with ASD might therefore experience the desire to engage socially concurrent with deficits in the ability to engage in effective social communication, which is a defining characteristic of ASD (APA, 2013). The juxtaposition of a desire to engage socially and a relative inability to do so effectively might contribute to feelings of isolation, loneliness or social withdrawal. Withdrawal and isolation are well-known predictors of depression, and may also emerge as a product of depressive symptoms in the form of anhedonia and avolitional tendencies (Rich & Scovel, 1987). The interaction of social communication deficits and social motivation may therefore produce feelings of loneliness, and ultimately depression. However, high social motivation is unlikely to be present in all individuals with ASD. For those with ASD and low social motivation, the interaction of motivation and skills deficits would not be predictive of isolation, and depression would be unlikely to result.

Regardless of etiology, diagnoses of anxiety and depressive disorders—even independently of ASD—are pervasive, persistent, and incur a substantial public health cost. A meta-analytic study examining the effects of a variety of anxiety diagnoses on quality of life established that social and generalized anxiety, obsessive-compulsive disorder, and others exert a significant negative impact on quality of life (Olatunji, Cisler, & Tolin, 2007). Individuals with social anxiety are also more likely to experience impairment in daily activities and demonstrate

lower quality of life as compared to controls (Stein & Kean, 2000). In ASD, anxiety and depression have been shown to be associated with lower quality of life and poorer functional outcomes for adults with ASD (Howlin et al., 2004; Smith, Ollendick, & White, 2019). When combined with the impact of core symptoms as well as the comorbid medical conditions that frequently accompany diagnoses of ASD, the effects of anxious and depressive symptoms may be exacerbated. The pervasiveness of internalizing symptoms in ASD and their impact on functioning has led to the development and validation of a variety of interventions. Cognitive-behavioral approaches have predominated, and have demonstrated generally positive effects (Chalfant, Rapee, & Carroll, 2007; Lang, Regester, Lauderdale, Ashbaugh, & Haring, 2010; White et al., 2013; Wood et al., 2009). Far fewer interventions have been developed to address depressive symptoms in individuals with ASD (White et al., 2018). Change in depressive symptoms as a result of intervention has been demonstrated primarily within the context of social skills interventions, wherein addressing comorbid symptomatology was not a primary study aim (Schiltz et al., 2018).

Current Study

Evidence suggests that anxiety and depression are commonplace among individuals with ASD, contribute to overall impairment in functioning and poor outcomes, and are responsive to intervention (White et al., 2018). Desire for social interaction and experienced loneliness have also been associated with comorbid internalizing symptoms in ASD samples (Deckers, Roelofs, Muris, & Rinck, 2014). Identification of patterns of internalizing symptomatology and social motivation may provide support for alternate theoretical approaches to the etiology of anxiety and depression in ASD. Additionally, should meaningful patterns of anxiety, depression, and social motivation emerge, interventions might be tailored to leverage an individual's intrinsic

desire for social interaction to build skills in this domain or address depressive or anxious symptoms. The current study therefore aimed to explore patterns of anxiety, depression, social motivation, and autistic symptomatology in youth with ASD via latent profile analysis (LPA). Consistent with prior work, we hypothesized that a substantial proportion of individuals would be grouped in classes with clinical levels of anxious and depressive symptoms. Further, we predicted that at least one class would emerge in which anxious and depressive symptoms were associated with elevated social motivation. The identification of such a class would provide support for the hypothesis that an interaction between social communication deficits and social motivation may contribute to the development of internalizing symptomatology. Our secondary aim was to examine whether profile membership was associated with age, biological sex, or desire for social interaction during a developmental period when symptoms of ASD frequently emerge.

Method

Participants

Participants included 195 individuals drawn from the National Database for Autism Research (NDAR). Although not all studies providing data to NDAR verify diagnoses using best-practice measures such as the ADOS or ADI-R, all participants included in the current study were required to have been administered Module 3 of the ADOS-2 (Lord et al., 2012). Average age of participants at time of ADOS-2 completion was approximately 10 years ($M = 10.08$ years, $SD = .80$), and participants were 74% male. All participants had an ADOS overall total of seven or greater, which is equal to the “autism spectrum” cutoff on the ADOS-2; the average comparison score corresponded to symptoms of a moderate severity ($M = 7.39$, $SD = 1.74$). Full descriptive statistics can be found in Table 1. Due to the heterogeneous nature of NDAR data

collection, not all participants completed all study measures at the same time point. In order to mitigate potential problems with interpretability of analyses utilizing data that were collected at vastly different time points, we included only participants who had completed all four primary study measures within a two-year window. Participants who had completed measures over a greater span ($n = 10$), ranging from 29-67 months, were excluded.

Measures

Autism Diagnostic Observation Schedule (ADOS-2; Lord et al., 2012). The ADOS-2 is considered a best-practice tool for diagnosis of ASD when used in conjunction with other tools and clinical judgment (APA, 2013; Pugliese et al., 2015). The ADOS-2 offers five separate modules with varying structure and tasks; module administration is determined based on chronological age, use of language, and clinical judgment. Participants included in the current study each completed Module 3 of the ADOS-2, which was designed to be administered to children and adolescents with fluent and complex speech. For the purposes of comparison across modules, total scores on the ADOS-2 (i.e., the sum of Social Affect and Restricted and Repetitive Behavior algorithm items) can be converted into a Calibrated Severity Score (CSS; Gotham, Risi, Pickles, & Lord, 2007). These scores range from 1-10 and are accompanied by qualitative descriptors indicating level of evidence for ASD diagnosis (i.e., minimal-to-no-evidence, low, moderate, and high).

Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001). The CBCL consists of 113 items rated on a three-point scale (0 = not true, 1 = somewhat or sometimes true, 2 = very true or often true) assessing various components of psychopathology. The Withdrawn/Depressed subscale of the CBCL was used as a dimensional measure of depressive symptomatology in the current study. The Withdrawn/Depressed subscale is one of eight empirically based syndrome

scales. The CBCL is among the most broadly used measures of internalizing symptomatology in youth, and substantial evidence supports the sound psychometric properties of the measure (Achenbach & Rescorla, 2001). Additionally, the syndrome scale factor structure of the CBCL has been supported specifically in samples of youth with ASD (Pandolfi, Magyar, & Norris, 2014).

Screen for Child Anxiety Related Disorders (SCARED; Birmaher et al., 1997). The parent-report version of the SCARED was used for the current study. The SCARED includes 39 items, each of which is rated on a three-point scale (0 = not true or hardly ever true, 1 = somewhat true or sometimes true, 2 = very true or often true). The SCARED offers subscales for generalized anxiety, panic disorder and somatic symptoms, separation anxiety, social anxiety, and school avoidance, as well as an overall anxiety score. Clinical cutoffs are available for each of these subscales. The social anxiety subscale was used for the current study. The SCARED has been used extensively in samples of youth with (Keefer et al., 2017; van Steensel & Heeman, 2017) and without ASD (Birmaher et al., 1997; Birmaher et al., 1999; Monga et al., 2000). An evaluation of the psychometric properties of the measure in a sample of youth with ASD has indicated that the factor structure established in non-ASD samples is replicated in ASD, along with strong internal reliability and moderate convergent validity with a structured clinical interview (Stern, Gadgil, Blakeley-Smith, Reaven, & Hepburn, 2014; van Steensel, Deutschman, & Bögels, 2012).

Social Responsiveness Scale (SRS-2; Constantino & Gruber, 2012). The parent-report version of the SRS-2 was used for the current study. The SRS-2 consists of 65 items rated on a 4-point scale (1 = not true, 2 = sometimes true, 3 = often true, 4 = almost always true). In addition to a total score, the SRS-2 includes subscales for social awareness, social cognition, social

communication, social motivation, restricted interests and repetitive behaviors. For the current study, scaled scores for the social motivation subscale were used as an index of interest or desire to engage in social relationships. The social motivation subscale of the SRS-2 has previously been used as an indicator of desire for social interaction (Factor, Condy, Farley, & Scarpa, 2016; Swain, Scarpa, White, & Laugeson, 2015). Because higher scores on the social motivation subscale correspond to greater levels of withdrawal (and not interest in social interaction), this construct will henceforth be referred to as “social amotivation.” A substantial body of evidence also exists to support the sound psychometric properties of the SRS-2 as a continuous measure of ASD severity (Frazier et al., 2013; Wigham, McConachie, Tandos, & Le Couteur, 2012).

Autism Diagnostic Interview, Revised (ADI-R; Le Couteur, Lord, & Rutter, 2003).

Although not included in our primary analyses, select items from the ADI-R were included where available to determine whether indicators of social motivation early in development (i.e., between 4 and 5 years) predicted membership in classes with higher levels of comorbid psychopathology in later childhood. The ADI-R is a standardized, semi-structured interview conducted with caregivers of children and adults and contains 93 items assessing early developmental milestones as well as current behavior in the domains of social interaction, communication and language, and restricted and repetitive behaviors. Items selected as indicators of social interest included item 51 (use of social smile), item 54 (seeking to share enjoyment with others), item 62 (level of interest in same-age peers), and item 63 (response to approaches of other children). Caregivers report on the presence or absence of these behaviors both at the time of the interview and between the ages of four and five. Responses are coded on a four-point scale, with zero corresponding to typical development and three corresponding to

diminished interest in social interaction. Coding for these items was reversed for the current study, with higher scores corresponding to increased social interest.

Procedure

NDAR is one of several research repositories maintained as part of the National Institute of Mental Health Data Archive. All NIH-funded studies using samples that include individuals with ASD are mandated to submit data to NDAR as a condition of funding. The NDAR repository includes over 300,000 participants and includes behavioral, genomic, and neuroimaging data. Because of the heterogeneity of studies submitting to the repository, no standard battery of assessment measures has been administered to all individuals in the database. However, some commonly used measures, such as those used to make ASD diagnoses, are present in a substantial subset of the overall NDAR sample. Data for the current study were assembled using NDAR's Data Dictionary tool based on data available as of June 2019. In order to obtain the broadest possible sample, complete data for all participants in the database with scores on Module 3 of the ADOS-2 (Lord et al., 2012) were downloaded. Therefore, all participants had overall ADOS scores (i.e., Social Affect + Restricted and Repetitive Behaviors). Other included measures (i.e., CBCL, SCARED, and SRS-2) were subsequently merged into the dataset. Due to the variety of data sources, missing data were common. In order to avoid listwise deletion, study participants were retained if they had complete data on all variables of interest or were missing one variable. Participants missing data for two or more primary variables were excluded.

Data Analysis

Given that these data were collected across a range of different studies, some of which were longitudinal and therefore subject to attrition, there were considerable missing data in our

study. Primary variables (ADOS total score, SRS-2 social motivation T-score, SCARED social anxiety score, and CBCL Withdrawn/Depressed T-score) were subject to 17% missing data. However, the vast majority of this missing data was restricted to SCARED social anxiety scores; ADOS scores, SRS-2 scores, and CBCL scores were missing 4% of data. Missing data were accounted for by the use of full information maximum likelihood (FIML) estimation, as is standard practice in MPlus Version 8 (Muthén & Muthén, 2017). Little's missing completely at random (MCAR) test was used to ensure that assumptions were met for the use of FIML (Little & Rubin, 2002), and data were found to meet this standard, $\chi^2(11) = 12.70, p = .31$. In addition to Little's MCAR, we conducted additional analyses to ensure that participants with SCARED parent-reported data did not systematically differ from those missing such data. First, we conducted a series of independent samples *t*-tests to determine whether participants with versus without SCARED data differed on other primary variables. Participants did not differ significantly on the basis of ADOS total scores, $t(193) = -1.40, p = .16$, SRS-2 Social Motivation T-scores, $t(169) = -1.76, p = .08$, or CBCL Withdrawn/Depressed T-scores, $t(202) = -1.77, p = .08$. Then, we conducted a binary logistic regression to determine whether each of these three variables predicted missingness of SCARED scores. None of these three variables emerged as a significant predictor of whether or not participants were missing SCARED data.

CBCL Validity. Given that the CBCL was developed and validated on a non-ASD population, we conducted a confirmatory factor analysis (CFA) to verify that the empirically based syndrome scales derived from the normative sample were replicated in the current ASD sample. Prior work has conducted initial examinations of the psychometric properties of the CBCL for both preschool and school-age populations with ASD, with results suggesting replication of the established factor structure (Pandolfi, Magyar, & Dill, 2009, 2012). To provide

additional evidence for the validity of the broadband Internalizing/Externalizing factor structure, we conducted comparable analyses in our sample. Our CFA consisted of T-scores for anxious/depressed, withdrawn/depressed, and somatic complaints loaded onto an internalizing factor and rule-breaking problems and aggressive behavior loaded onto an externalizing factor; internalizing and externalizing problems were correlated in our model, consistent with prior work (Ivanova et al., 2007; Pandolfi, Magyar, & Dill, 2012). Model fit was assessed through the use of four indices: maximum-likelihood chi-square, root mean square error of approximation (RMSEA), comparative fit index (CFI), and standardized root mean square residual (SRMR). Acceptable values for RMSEA and SRMR were set at .08, and CFI was set at .90 (Kenny, 2015). A nonsignificant Chi-square test, $\chi^2(4) = 3.95, p = .41$, suggested excellent model fit, and RMSEA (<.001), CFI (>.99), and SRMR (.02) also fell within acceptable levels. All factor loadings were statistically significant.

Latent Profile Analysis. Latent Profile Analysis (LPA), conducted using MPlus Version 8 with combination add-on (Muthén & Muthén, 2017), was used to identify subgroups of youth with ASD with varying levels of ASD symptoms, internalizing symptoms, and social amotivation. Adhering to standard practice for this person-centered clustering approach (Nylund, Asparouhov, & Muthén, 2007), we began by specifying two latent classes and sequentially increasing the number of latent classes in order to achieve parsimony. Five model fit indices, as well as theoretical interpretability, were used to identify the best-fitting model. Akaike Information Criterion (AIC), Bayesian Information Criterion, and sample-size adjusted Bayesian Information Criterion (SABIC) were utilized as primary indices; lower values for each of these indicators generally signifies better model fit. The Lo-Mendell-Rubin Likelihood Ratio Test (LMR) was used to evaluate the fit of a model with k classes as compared to the prior model with

$k - 1$ classes; a significant LMR test indicates the greater number of classes provides significantly better model fit. Thus, classes were added to the LPA models until a nonsignificant LMR test was produced. Entropy values were also used to assess model fit; values above .80 are generally regarded as providing good fit (Carragher, Adamson, Bunting, & McCann, 2009). We also considered additional indicators of parsimony. Consistent with methodological guidance using this approach, models were considered to provide acceptable fit when class probabilities were maximized (i.e., $> .70$ average probability of being placed in a specified class and $< .30$ probability of being placed in a different class), and where no class was specified containing less than 5% of the overall sample (Nagin, 2005; Pearcey et al., 2018). Differences among identified classes on each of the four indicators were examined using a series of one-way analysis of variance (ANOVA) tests, with alpha levels Bonferroni corrected for multiple comparisons (i.e., $.05/4 = .0125$). Significant omnibus ANOVAs were followed up by post-hoc testing using Tukey HSD tests. This analytic plan follows guidance suggesting the use of multivariate analysis of variance (MANOVA) tests may result in inflated Type I error rate (Jaccard & Guilamo-Ramos, 2002).

In addition to generating distinct subgroups of youth with ASD and varying levels of anxious and depressive symptoms, we aimed to identify significant associations between class membership probability and other key variables, including age and biological sex and ADI-R items as an index of social motivation at age 4-5. These analyses were carried out using the automatic three-step approach for modeling of auxiliary variables (Asparouhov & Muthén, 2013). The three steps are as follows. First, the latent profile model is generated based only on the chosen indicator variables. Second, a variable for most likely class membership is generated using the latent class posterior distribution from the first step. In the third step, regression is used

to evaluate the relationship between the proposed antecedent variables and the most likely class variable. The three-step model confers an advantage over traditional cluster analysis as well as other regression approaches with LPA models in that 3-step models take into account the probability of misclassification in step 2, rather than simply treating most likely class membership as a categorical variable (Asparouhov & Muthén, 2013). This three-step process was used to determine whether class membership differed on the basis of age (using the BCH command) or biological sex (using the DCAT command). The R3STEP command was used to determine whether class membership could be predicted by scores on ADI-R items indexing social motivation early in development.

Results

Descriptive statistics and bivariate correlations are reported in Tables 1 and 2. Consistent with inclusion criteria, the average Calibrated Severity Score (CSS) on the ADOS-2 corresponded to moderate symptom severity ($M = 7.49$, $SD = 1.72$), though scores ranged from Low (i.e. $CSS = 4$) to High ($CSS = 10$), out of a possible range of 1-10 (Gotham et al., 2007). Levels of depressive symptoms and social amotivation were each elevated by approximately 1-1.5 standard deviations, or 13-17 points, relative to normative samples of typically developing youth (Achenbach & Rescorla, 2001; Frazier et al., 2013). ADOS severity did not correlate with any other variable; correlations among social amotivation, depression, and social anxiety scores were moderate to large and statistically significant.

Model fit statistics are presented in Table 3. AIC, BIC, and SABIC decreased from the single profile to the two profile solution; AIC and SABIC further decreased from the two profile to the three profile solution. A significant LMR test indicated that model fit was significantly improved for the three profile solution as compared to the two profile solution. Entropy

decreased slightly from the two profile to the three profile solution, but remained within acceptable limits (0.77). Additionally, latent class probabilities indicated satisfactory discrimination among classes, with all class membership probabilities greater than 0.85 and incorrect classification probabilities generally lower than .10.

Estimates and standard errors for the three-profile solution are provided in Table 4. Levels of symptomatology characterizing each class below are described relative to the other two classes rather than population-based norms; qualitative descriptors are consistent with terminology specific to each measure (e.g., “moderate” and “high” levels of ASD symptoms were specified by guidelines from CSS scores). Class 1, in which the majority of participants were categorized ($n = 106$), was characterized by moderate ASD severity (Total $M = 11.68$, $SD = .40$)¹ and relatively low levels of anxiety ($M = 3.17$, $SD = .42$), depression ($M = 58.87$, $SD = .76$), and social amotivation ($M = 60.42$, $SD = 1.11$). Class 1 could therefore be considered a “low comorbidity” group, with ASD symptoms unassociated with significant symptoms in other domains. The smallest group, Class 2 ($n = 23$), consisted of individuals with the greatest levels of ASD symptomatology ($M = 19.85$, $SD = .78$); CSS scores for all individuals with this group fell in the high category. The group was otherwise comparable to Class 1 in terms of social anxiety ($M = 2.90$, $SD = 1.76$), depression ($M = 57.63$, $SD = 1.28$), and social amotivation ($M = 61.62$, $SD = 8.55$). Such a pattern suggests that this group can be characterized as a “severe ASD” class. Estimates for ASD symptoms in Class 3 ($n = 66$) fell between moderate and high severity ($M = 12.87$, $SD = .47$), and participants in this class had substantially elevated scores across all domains of comorbidity. Estimates for social anxiety ($M = 10.16$, $SD = .79$), depression ($M = 72.43$, $SD = 1.09$), and social amotivation ($M = 79.60$, $SD = 1.39$) were all above clinical cutoffs

¹ Standard deviations as reported for LPA results represent standard errors of model estimates (Muthén & Muthén, 2017); as such, they differ from standard deviations as reported subsequently in ANOVA results.

on their respective measures. Therefore, this group might best be characterized as a “high comorbidity” class.

The three classes differed significantly from one another with regard to ADOS overall total scores, $F(2, 192) = 84.85, p < .001$. Post-hoc Tukey HSD tests revealed that ASD severity was significantly lower in the low comorbidity class ($M = 11.66, SD = 2.79$) as compared to the severe ASD class ($M = 20.65, SD = 2.04$), $p < .001$. The severe ASD and high comorbidity ($M = 12.87, SD = .47$) classes also differed significantly from one another ($p < .001$). Social amotivation also differed significantly among the three classes, $F(2, 168) = 98.75, p < .001$, with post-hoc Tukey tests indicating significant differences between the low comorbidity ($M = 60.28, SD = 8.43$) and high comorbidity classes ($M = 80.05, SD = 8.88$), $p < .001$, as well as between the severe ASD ($M = 61.09, SD = 9.17$) and high comorbidity classes, $p < .001$. The low comorbidity and severe ASD classes did not differ significantly from one another, $p = .92$. Differences in social anxiety among the three classes were also statistically significant, $F(2, 53) = 44.48, p < .001$. With regard to post-hoc pairwise comparisons, social anxiety scores in the low comorbidity ($M = 3.06, SD = 2.10$) and severe ASD classes ($M = 3.33, SD = 3.39$) were each significantly lower than the high comorbidity class ($M = 10.14, SD = 2.77$), $p < .001$. The low comorbidity and severe ASD classes did not differ significantly ($p = .96$) from one another. Finally, depression scores differed significantly among the three groups, $F(2, 191) = 107.76, p < .001$. Post-hoc Tukey tests revealed a pattern of significance comparable to anxiety scores, with low comorbidity ($M = 58.78, SD = 6.16$) and severe ASD classes ($M = 57.26, SD = 5.38$) not differing from one another ($p = .57$), but with each significantly lower than depression scores in the high comorbidity class ($M = 72.95, SD = 9.45$), $p < .001$.

Following identification of the optimal profile solution, demographic variables were incorporated using the BCH and DCAT commands in MPlus to identify significant associations with class membership. An overall Chi-square test indicated that age was not significantly associated with class membership, $\chi^2(2) = 5.66, p = .06$. Although the overall test indicated no significant difference among the three classes, participants in the severe ASD class were significantly younger than those in the high comorbidity class, $\chi^2(2) = 5.13, p = .02$. Similarly, an overall test examining whether class membership was associated with biological sex fell short of significance, $\chi^2(2) = 4.99, p = .08$. However, the probability of an individual in the severe ASD class being male was significantly greater than the probability of an individual in the low comorbidity class, $\chi^2(2) = 4.92, p = .03$.

Four ADI-R items associated with desire for social interaction at age 4-5 were examined as predictors of ASD severity, anxiety, depression, and social amotivation later in life using the R3STEP procedure in MPlus. Items examined were use of social smile (item 51), ability to demonstrate shared enjoyment (item 54), interest in other same-age peers (item 62), and response to approaches of same-age peers (item 63); each of these items was coded on a four-point scale, reverse-coded from ADI-R codes as typically administered such that higher scores corresponded to greater levels of social interest. None of these four items significantly predicted membership in any of the three classes. Due to the nature of the R3STEP procedure, FIML is not applied to missing auxiliary variable data; rather, listwise deletion is employed. The sample size for the auxiliary analyses using ADI-R items was therefore considerably smaller ($n = 110$) than primary LPA results and prediction of group membership based on age and biological sex.

Discussion

The goal of this study was to identify meaningful subgroups of youth with ASD presenting with varying profiles of ASD symptomatology, anxiety, depression, and social amotivation. Our analyses produced three distinct classes, each with a unique pattern of symptoms. The largest class consisted of youth with low (i.e., subclinical) symptoms of depression and anxiety, moderate symptoms of ASD, and low social amotivation. A second, smaller group was characterized by similarly low levels of anxiety, depression, and social amotivation, but significantly more severe symptoms of ASD as measured by the ADOS-2. Finally, a high psychopathology class emerged which included youth with significantly elevated (i.e., above clinical cutoff) anxiety, depression, and social amotivation symptoms and with moderate symptoms of ASD. Model fit indices suggested satisfactory distinction among these groups, and the specification of additional groups significantly degraded model fit. Class membership did not vary significantly as a function of age or sex.

Each of the three classes emerging in our analyses warrants contextualization within the broader comorbidity literature. First, the low comorbidity class presented with lower levels of anxious and depressive symptoms than have been reported in comparable samples and using similar measures (Mazefsky, Borue, Day, & Minshew, 2014; Skokauskas & Gallagher, 2012; Stern et al., 2014). This class therefore appears to represent the substantial proportion of individuals with ASD who present without clinical levels of depression and anxiety (van Steensel et al., 2011). Consistent with patterns seen in the broader ASD population, this class may have had considerable comorbid symptomatology in domains not assessed in the current study, such as externalizing symptoms (e.g., inattention, behavioral disturbance, aggression). For example, attention-deficit/hyperactivity disorder (ADHD) has been estimated to be comparable

to anxiety in terms of prevalence among youth with ASD (Simonoff et al., 2008), and may have been prevalent among this “low comorbidity” class.

The second group also demonstrated levels of anxiety, depression, and social amotivation falling below clinical cutoffs for their respective measures. In contrast to the low psychopathology group, however, this group presented with significantly greater ASD severity. All participants in this class had the maximum CSS score (i.e., CSS = 10). The simplest interpretation of this class would suggest that comorbid anxiety and depression are less prevalent among individuals with more severe autistic symptoms. However, examinations of the relationship between ASD symptom severity and comorbid anxiety have tended to suggest that more severe ASD symptoms are associated with *increased* anxiety (Wood & Gadow, 2010). While some individuals in this class likely do have lower levels of anxiety relative to peers with ASD, another interpretation might argue that symptoms of anxiety and depression are more difficult to capture in children with more severe symptoms of ASD. Such an interpretation is consistent with the notion of “diagnostic overshadowing,” in which ambiguous symptoms that might be indicative of either ASD or anxiety are attributed to the former on the basis of a more severe clinical presentation (Kerns et al., 2015; MacNeil, Lopes, & Minnes, 2009). Atypical presentations of anxiety have been shown to be common in youth with ASD (Kerns et al., 2014; Wood & Gadow, 2010). Behaviors not immediately and obviously associated with anxiety (e.g., externalizing and oppositional behavior, engaging in restricted and repetitive behaviors) might be interpreted and reported by parents as manifestations of ASD or externalizing symptoms. ASD severity as indexed by ADOS severity scores has previously been shown to correlate negatively with verbal ability (Gotham et al., 2007; Hus & Lord, 2014). Although updated algorithms have resulted in smaller associations between these constructs (Pugliese et al., 2015), youth severely

impacted by ASD symptoms may still have more difficulty reporting anxious symptoms than their peers with greater verbal abilities (Mazefsky, Kao, & Oswald, 2011; White, Schry, & Maddox, 2012). Yet another interpretation of this class might suggest that youth with more elevated ASD symptomatology may have less insight into their emotions, resulting in correspondingly lower parent report of symptoms. Although omnibus Chi square tests were nonsignificant, pairwise comparisons indicated that the severe ASD class was younger and more likely to be male. If similar effects reach the level of statistical significance in future work, such a finding would represent a departure from literature suggesting that females with ASD present with more severe symptoms of ASD as well as internalizing symptoms (Solomon, Miller, Taylor, Hinshaw, & Carter, 2012).

Finally, the third class emerging in our analyses presented with elevated internalizing symptoms. Social anxiety, withdrawn/depressive symptoms, and social amotivation all exceeded their respective measures' clinical cutoffs. Indeed, the proportion of individuals in this class (34% of the total sample) is consistent with reported rates of anxiety across several reviews and meta-analyses (Mazefsky, Conner, & Oswald, 2010; Simonoff et al., 2008; van Steensel et al., 2011; van Steensel & Heeman, 2017; White et al., 2009). Rates of depressive symptoms comparable to those seen here have also been identified in recent work (Hollocks et al., 2019; Hudson et al., 2019). In this high comorbidity class, social amotivation (also associated with a preference for spending the majority of one's time alone) was significantly higher than that seen in either of the other classes. This pattern might be interpreted as inconsistent with the proposed social motivation model of depression and anxiety, wherein depression and anxiety result from a mismatch between an individual's interest in social interaction (high) and one's ability to navigate such interactions successfully (low). The current pattern of results is, however,

consistent with core symptoms of depression, in which depressed mood would be associated with broader anhedonic and avolitional tendencies (APA, 2013). The emergence of this class therefore maps on to prior work that has identified low social motivation alongside depressive symptoms (Gotham et al., 2015). In that work as well as the current study, it remains unclear whether social motivation (or lack thereof) is a product or a cause of depressive symptoms. Notably, social amotivation was significantly higher among individuals with elevated internalizing symptoms compared to the severe ASD group; ADOS severity scores and social amotivation were also uncorrelated ($r = .02$). These results may suggest that social amotivation as indexed by the SRS-2 captures withdrawal associated with internalizing symptoms more so than social amotivation assumed to accompany ASD based on social motivation theory.

Of note, no class emerged from our analyses with the predicted pattern of elevated symptoms of depression and anxiety and low amotivation scores. This result may be explained in part by the association of amotivation with depressive symptoms, as described above. Individuals with significant symptoms of anxiety and depression may have experienced a sufficient number of social interactions with adverse outcomes such that their interest in such interactions has diminished considerably. This conclusion is consistent with proposed etiological models of anxiety in ASD samples (Wood & Gadow, 2010). Anxiety (i.e., avoidance) and depression (e.g., amotivation, anhedonia) may sap premorbid (i.e., prior to onset of the secondary mood/anxiety problem) desire for social interaction, resulting in the pattern seen in the high psychopathology group. However, factors related to the sample and measures used in the current study may have contributed to our failure to identify a group with strong social motivation and significant internalizing symptoms. The age of our sample may not have aligned with developmental processes associated with emergence of internalizing symptoms. Anxiety has been shown to

increase throughout adolescence (Costello, Copeland, & Angold, 2011), and our sample with a mean age in middle childhood ($M = 10.08$ years) may have been too young to reflect these symptoms. A larger sample may also have yielded more diverse symptom presentations, including elevated internalizing symptoms and low social amotivation.

Auxiliary analyses conducted as part of the current study also indicated that age and sex did not vary significantly among the three identified classes, and that social motivation-oriented ADI-R items did not predict membership in any identified class. Although overall comparisons suggested that age did not vary among classes, the high comorbidity class was significantly older than the severe ASD symptom class. Similarly, sex did not differ significantly in an overall comparison, though males were more prevalent among the severe ASD class than the low comorbidity class. The pattern of results with regard to sex and ASD severity is inconsistent with the body of literature indicating that diagnosed girls tend to be more severely affected in terms of ASD symptoms (Mayes & Calhoun, 2011; Werling & Geschwind, 2013). The association of greater chronological age with higher levels of psychopathology is consistent with accepted models of developmental psychopathology. Internalizing symptoms typically increase prior to and during adolescence, with a common age of onset during middle childhood (Costello et al., 2011; Kessler et al., 2005).

Several factors may have contributed to our finding that social motivation-related ADI-R items did not predict class membership. The validity of the selected ADI-R items as an index of social motivation is a primary consideration. The items (i.e., use of social smile, seeking to share enjoyment with others, level of interest in same-age peers, and response to approaches of other children) were selected as face valid indicators of interest in social interaction. Each item was rated on a four-point scale and used as an independent predictor, with no composite score

calculated. These behavioral indicators of motivation may not constitute a valid reflection of social interest in 4-5 year old children. Another interpretation is that these items do index social interest, but that interest varies throughout the developmental trajectory and/or manifests in functionally different ways as children age (Chevallier et al., 2012).. The ADI-R requires parents to rate their child's behavior both currently as well as during the developmental period in which symptoms of ASD frequently emerge (i.e., 4-5 years old). Parent recall of highly specific behaviors, particularly for those parents with older children, may be subject to questions of validity based on the ways in which items are phrased (Jones et al., 2015). A final interpretation might suggest that the selected items do serve as a valid index of social interest, but that levels of interest do not predict psychopathology.

The results of the current study provide meaningful insights into varying presentations of comorbidity in ASD. Nevertheless, results need to be interpreted in the context of the study's limitations. Foremost among these limitations is the validity of assessment tools to measure key constructs. Although measures used to index ASD severity, depression, and anxiety are all well-validated, an interview-based measure of comorbid symptoms such as the Anxiety Disorders Interview Schedule (ADIS; Silverman & Albano, 1996) may have improved our ability to measure specific symptom domains over broader child psychopathology measures such as the CBCL. Perhaps more crucially, results suggested that the social motivation subscale of the SRS-2 likely does not provide a valid index of desire for desire to engage in social interaction. Although the SRS-2 has previously been used as an index of social motivation (Factor et al., 2016; Swain et al., 2015), the strong correlation between this subscale and the Withdrawn/Depressed subscale of the CBCL in our sample suggest the SR-2 may be capturing internalizing symptoms rather than true social interest. Behaviorally-based measures as well as

questionnaires explicitly inquiring about particular social encounters may provide better indices of desire to engage in social interactions in individuals with ASD (Deckers et al., 2014).

Aside from questions of measurement, assessment time points and the cross-sectional nature of our data represent additional limitations of our study. The proposed pathway to depression and anxiety is by definition longitudinal, with psychopathology emerging over time as a product of motivation to interact, social encounters with adverse outcomes, and increasing awareness of the influence of one's social communication deficits. Assessment of ASD symptoms, depression, anxiety, and social motivation within a two-year window was necessary given the nature of the data, but likely obscured nuanced patterns of developing symptoms. Additionally, measurement of these constructs during middle childhood, as opposed to adolescence or adulthood, may have limited our ability to detect phenomena that play out somewhat later in the developmental trajectory.

Our results also offer several avenues for future study. The low and high comorbidity classes identified align with established models of comorbidity in ASD, both in rates of clinically significant symptoms and in severity (Hollocks et al., 2019; van Steensel et al., 2011; van Steensel & Heeman, 2017; White et al., 2009). The assessment of internalizing symptoms in individuals with severe ASD symptoms also warrants further study. Although the severe ASD class was characterized by relatively lower symptoms of anxiety and depression than the high comorbidity class, additional work is required to determine whether this group is representative of the proportion of the ASD population without significant comorbidity. An alternative interpretation might suggest that certain measures used in the current study were not reflective of true symptomatology due to known issues associated with assessment of anxiety and depression in ASD (e.g., diagnostic overshadowing, discrepant reporting). Our findings based on analysis of

auxiliary variables predicting group membership also warrant further examination. Behaviors indicative of social motivation, as well as demographic factors such as age and sex, were not found to predict group membership in the current study. However, evidence suggesting that internalizing symptoms vary based on age and sex, both in the broader population and ASD, would suggest that our work should be replicated in attempts to determine how these variables should be considered in the assessment and treatment of anxious and depressive symptoms in ASD (May, Cornish, & Rinehart, 2014; Oswald et al., 2016). Perhaps the most crucial future direction is the exploration of varying trajectories of anxiety, depression, and social motivation longitudinally. The model upon which the current study is based rests upon the assumption that depression and anxiety emerge as a product of high social motivation, rendering a prospective study necessary in order to examine this causal relationship.

The distinct classes of symptoms identified in the current study carry implications for the assessment and treatment of anxiety and depression in youth with ASD. Internalizing symptoms remain a primary contributor to diminished functional independence and quality of life for individuals with ASD (van Heijst & Geurts, 2015). Although considerable progress has been made, much work remains in refining and disseminating tools to assist clinicians aiming to conduct efficient and accurate differential diagnosis of ASD and internalizing symptoms. In a similar manner, interventions developed to address anxiety and depression in ASD, primarily through cognitive behavioral frameworks, have demonstrated promising effects. The identification of mechanisms of change as well as moderators of treatment effectiveness of these interventions have been proposed as priorities for future research (White et al., 2018). Through our identification of distinct subgroups, our work may provide clues aiding in the identification of individuals most likely to benefit from treatment. Should social motivation emerge as a risk

factor for the development of internalizing symptoms, highly motivated individuals may be identified as strong candidates for social skills interventions designed to minimize the gap between interest and ability in social interactions. Application of tailored treatment to the individuals who are most likely to respond may pave the way for amelioration of symptoms that interfere with the achievement of positive outcomes, including meaningful peer relationships, education, employment, and quality of life for children, adolescents, and adults with ASD.

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Table 1. *Descriptive Statistics*

	<i>n</i>	<i>M</i> (<i>SD</i>)	Range
ADOS Overall Total (Social Affect + RRB)	195	13.13 (4.11)	7-17
ADOS-2 Calibrated Severity Score	195	7.49 (1.72)	4-10
CBCL Withdrawn/Depressed T-Score	194	63.42 (9.45)	50-91
CBCL Total Problems T-Score	194	63.65 (8.41)	42-80
SCARED Parent Report – Social Anxiety Score	46	4.86 (3.89)	0-14
SCARED Total	56	19.68 (14.25)	0-56
SRS Total T-score	170	74.91 (12.48)	33-113
SRS Social Motivation T-score	171	67.20 (12.73)	42-103
Age in years at completion of ADOS	195	10.08 (.80)	5.1-15
Gender	<i>n</i>	%	
Male	145	74	
Female	50	26	

Table 2. *Correlation Table for Variables included in LPA*

	1.	2.	3.	4.
1. ADOS Total	--			
2. SRS-2 Social Motivation (<i>n</i> = 195)	.02	--		
3. SCARED Social Anxiety (<i>n</i> = 56)	-.04	.51** (<i>n</i> = 53)	--	
4. CBCL With/Dep (<i>n</i> = 194)	-.01	.58** (<i>n</i> = 170)	.38** (<i>n</i> = 55)	--

Note. * $p < .05$ (2-tailed), ** $p < .01$ (2-tailed)

Table 3. *LPA Model Fit Statistics*

# classes	AIC	BIC	SA-BIC	LMR	Entropy	Profile Prevalence		
						1	2	3
1	4204.49	4230.67	4205.33					
2	4105.87	4148.42	4107.24	104.64*	.79	.66	.34	
3	4097.66	4156.58	4099.56	17.54 (ns)	.77	.53	.13	.34

Note. * $p < .05$ (2-tailed).

Table 4. *Latent Profile Class Probabilities and Model Estimates*

Class	Class Probabilities			Model Estimates and Standard Errors			
	1 (<i>n</i> = 106)	2 (<i>n</i> = 23)	3 (<i>n</i> = 66)	ADOS	SRS	SCARED	CBCL
1	0.90	0.04	0.04	11.68 (.40)	60.42 (1.11)	3.17 (.42)	58.87 (.76)
2	0.13	0.85	0.02	19.85 (.78)	61.62 (2.56)	2.90 (1.76)	57.63 (1.28)
3	0.06	0.01	0.91	12.87 (.47)	79.60 (1.39)	10.16 (.79)	72.43 (1.09)

Figure 1. *Inter-class Differences for LPA variables*