Published in final edited form as:

Res Dev Disabil. 2018 October ; 81: 65–72. doi:10.1016/j.ridd.2018.04.005.

Trajectories of Cognitive Development in Toddlers with Language Delays

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Abstract

Background: Toddlers with early language delays (LD) are at risk for developmental difficulties, including autism spectrum disorder (ASD). However, little is known about early cognitive skill acquisition in this population.

Aims: To explore heterogeneity in cognitive development in toddlers with significant LD (n=30) or typical development (n=61), and how this relates to 36-month outcomes (ASD, non-ASD delays, or no delays).

Methods: Growth mixture modeling of nonverbal and verbal mental age (NVMA, VMA) scores from the Mullen Scales of Early Learning was conducted with data from 18, 24 and 36 months.

Results: A two-class NVMA solution was selected (Age Appropriate, 82%, Delayed, 18%); class membership was related to the no delay outcome, and although the proportion of toddlers with ASD in the Age-Expected class was 17% compared to 50% of toddlers with non-ASD delays, this difference was not statistically significant. The best-fitting model for VMA included three classes: Age Appropriate (66%), Delay Catch-Up (23%), Delayed (11%); class assignment differed by outcome. Children in the Delay Catch-Up class were more likely to have non-ASD delays compared to ASD, while the reverse was true in the Delayed class.

Conclusions: Cognitive development in toddlers with LD is heterogeneous, and delayed verbal trajectories relate to later ASD diagnosis.

1. Introduction

The importance of the early developmental course of children who are later diagnosed with autism spectrum disorder (ASD) cannot be overstated (Rogers, 2009). Apart from population-based methods, any prospective study of infants and toddlers who will be

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Declarations of Interest: None

diagnosed with ASD requires that the researchers select some group of children who are more likely than the average child to develop ASD.

During the last 10 years when research using this high-risk design has accelerated, the population that has garnered the most attention is the younger siblings of children diagnosed with ASD. Prospective research on these infants, who have heightened genetic risk for developing the disorder, has critical implications for improving understanding of pathways to later outcomes (Szatmari et al., 2016). Importantly, however, there are limitations to generalizations of the findings from studies of infant siblings. Primarily, the unavoidable parental vigilance associated with having an older child already diagnosed with ASD may bias these studies.

Thus, complementary studies of children at risk for ASD due to factors other than familial genetic transmission alone are needed. Along with other risk groups, including infants with neonatal problems (Karmel et al., 2010) and infants already identified based on concerns about possible ASD (Lord, Luyster, Guthrie, & Pickles, 2012), toddlers with early language delays are good candidates for this purpose (Guthrie, Swineford, Nottke, & Wetherby, 2013). Toddlers with language delays are at increased risk for persistent developmental difficulties, and are diagnosed with ASD at a higher-than-average rate (Miniscalco, Nygren, Hagberg, Kadesjo, & Gillberg, 2006). Given that language delay is a commonly reported first concern of parents whose children are later diagnosed with ASD (Chawarska et al., 2007; Coonrod & Stone, 2004; Hess & Landa, 2012), language delay is a particularly relevant construct for creating a high-risk sample.

Nevertheless, the bulk of our knowledge about the early development of children later diagnosed with ASD comes from the infant sibling literature. A commonly explored potential predictor of interest in these toddlers at risk or already diagnosed is cognitive ability (Chawarska, Klin, Paul, Macari, & Volkmar, 2009; Jones, Gliga, Bedford, Charman, & Johnson, 2014). Research utilizing developmental measures that serve as a proxy for cognitive skills, such as the Mullen Scales of Early Learning (MSEL; Mullen, 1995), has helped elucidate the progression of verbal and nonverbal cognitive skill acquisition. For example, cognitive abilities in high-risk siblings have been shown to diverge from low risk infants by approximately 12 to 14 months (Landa & Garrett-Mayer, 2006; Landa, Gross, Stuart, & Bauman, 2012; Landa, Gross, Stuart, & Faherty, 2013; Ozonoff et al., 2010; Ozonoff et al., 2014). Rates of cognitive growth in infants later diagnosed with ASD leading up to the third year of life are variable, but often delayed (Brian et al., 2014; Estes et al., 2015; Landa & Garrett-Mayer, 2006; Landa et al., 2012). Despite the variability in developmental trajectories among the approximately 20% of high-risk infants who later receive an ASD diagnosis (Ozonoff et al., 2011), cognitive disparities compared to healthy controls tend to increase over time (Brian et al., 2014; Estes et al., 2015; Landa & Garrett-Mayer, 2006; Landa et al., 2012).

Importantly, research has also revealed significant heterogeneity in early cognition and its progression in these high-risk infant siblings. For example, one study documented differences in cognitive development between infants with earlier versus later diagnoses of ASD (Landa et al., 2013). Other studies have quantified this heterogeneity using latent

variable methods, providing further evidence that cognitive development in infant siblings at risk for ASD is not monolithic. Two studies using similar statistical methodology found evidence for three types of trajectories: advanced, stable, and delayed (also called declining, whether it be general delay or specific to language/motor) (Brian et al., 2014; Landa et al., 2012). In both of these studies, the children who later received an ASD diagnosis were more frequently in the stable or declining classes versus the advanced trajectory, suggesting that at least among infant siblings of children with ASD, these trajectories of cognitive development may indicate higher likelihood of later ASD diagnosis.

These studies provide initial, clinically meaningful evidence about the presence of heterogeneity and patterns of early development in infants at increased genetic risk for ASD. However, as described, the limitations inherent to studying infant siblings require that complementary evidence be assembled. In this study, we sought to describe prospectively the development of cognitive ability in children with early language delay, who at 36 months would achieve age-appropriate development, exhibit non-ASD delays, or be diagnosed with ASD. Thus, we utilize growth mixture modeling (GMM), a method of identifying latent subpopulations in longitudinal data, to objectively quantify and describe the heterogeneity in MSEL nonverbal and verbal cognitive trajectories in toddlers with early language delay.

We hypothesized that the heterogeneity in verbal and nonverbal cognitive development of our combined sample of toddlers with language delay (LD) and typical development (TD) would be best explained by more than one trajectory class. We expected that this would include an age-appropriate class and likely two classes with different patterns of delays, consistent with prior findings on trajectories in infant siblings (Landa et al., 2012). Since prior research in at risk samples has not specifically described relationships between nonverbal and verbal cognitive growth as measured by the MSEL, we also report the relationship between nonverbal and verbal MSEL class membership, which we expected to be strongly related. Finally, we expected that membership in both delayed verbal and nonverbal MSEL trajectory classes would predict ASD outcomes, as demonstrated in the infant sibling data (Brian et al., 2014; Landa et al., 2012).

2. Methods

2.1 Participants

Ninety-one toddlers participated in a longitudinal study examining language delay as a risk factor for outcomes such as ASD conducted across two sites: the National Institute of Mental Health and University of Utah. The Institutional Review Boards at both sites approved this study, and all families gave written consent for study participation. Participants included toddlers with LD ($n=30$) or TD ($n=61$). Study recruitment was based on suspicion of LD as a risk factor for ASD and drew from pediatric practices, early intervention providers, and child development clinics. TD toddlers were recruited through flyers posted in community settings (e.g., doctors' offices, preschools) and word-of-mouth. Toddlers were initially evaluated at 12 or 18 months of age (± 2 months) to determine study eligibility. Participants who met inclusion criteria (described below) were evaluated at 18, 24, and 36 months. Given the limited number of participants with 12-month data, the focus

of the present study is on the 18, 24 ($n_{LD}=28$, $n_{TD}=59$), and 36-month ($n_{LD}=24$, $n_{TD}=56$) intervals. Participant characteristics are shown in Table 1.

Based on inclusion criteria, all participants were born full term (≥ 36 weeks) and lived in households where English was the primary language. LD was determined upon study entry (12 or 18 months) using the Mullen Scales of Early Learning (MSEL; Mullen, 1995) and defined as receptive and expressive language scores in the “very low” range, or two standard deviations below the mean. No participants with LD had motor or other medical impairments deemed responsible for delays (e.g., cerebral palsy, known genetic disorder). Toddlers with TD had nonverbal and verbal scores on the MSEL of less than 1.5 standard deviations below the mean (T-scores greater than 35). Additional inclusion criteria for the TD group were no motor or medical impairment that would interfere with study participation, no known genetic disorders, and no sibling diagnosed with ASD.

2.2 Measures

Developmental assessment for all participants at all study intervals included administration of the Mullen Scales of Early Learning (MSEL; Mullen, 1995). Four subscale domains were administered: fine motor, visual reception, expressive language, receptive language. Age equivalents were derived to calculate nonverbal mental age (NVMA: average of the fine motor and visual reception age equivalent scores) and verbal mental age (VMA: average of the expressive and receptive age equivalent scores). The fine motor domain measures a range of abilities related to the use of hands and fingers and manual dexterity. The visual reception domain involves visual perceptual ability. The receptive language domain assesses understanding of verbal instructions, memory for commands, and auditory-spatial and auditory quantitative concepts. The expressive language domain measures vocalizations and utterances, as well as verbal responses to tasks and the expression of underlying concepts.

The Autism Diagnostic Observation Schedule, Second Edition (ADOS-2), a semi-structured, interactive observational measure, was administered by research-reliable doctoral level clinicians to LD participants at each interval and to TD participants at the 18 and 36 month intervals. The ADOS-2 includes opportunities for play and joint interaction and is designed to elicit behaviors relevant to a diagnosis of ASD. The Autism Diagnostic Interview, Revised (ADI-R; Le Couteur, Lord, & Rutter, 2003), a semi-structured clinical interview typically conducted with a parent or caregiver, was administered to all toddlers in the LD group at 36 months. Both measures are considered gold-standard assessment tools for the evaluation of ASD.

2.3 Outcome Classification

At 36 months, participants were classified as having ASD (determined by meeting diagnostic criteria, regardless of cognitive functioning), non-ASD developmental delays, or no delays by determination of an expert doctoral-level clinician after reviewing diagnostic criteria for ASD. For all three outcome groups, clinician judgment was informed by the results of the standardized tests administered as part of this study. For the 11 toddlers who did not have 36month data available, data from the most recent time point (the 24-month

interval for seven toddlers, the 18-month interval for four toddlers) were used to classify outcome. Outcome criteria were defined as follows:

2.3.1 ASD.—Best estimate diagnosis of ASD was based on the ADOS-2 and the ADI-R, criteria from the Diagnostic and Statistical Manual for Mental Disorders, 5th edition (American Psychiatric Association, 2013), and clinical judgment of expert doctoral-level clinicians.

2.3.2 Non-ASD Delays.—Toddlers who did not meet criteria for ASD but were judged to have remaining delays by clinicians were classified as non-ASD delay. These delays could include delays in verbal (receptive and/or expressive language) or nonverbal (cognitive and/or fine motor) development, as well as non-ASD social communication delays, so were not determined by specific scores.

2.3.3 No Delays.—Toddlers who did not meet criteria for ASD or non-ASD delays were classified in this group.

2.4 Statistical Approach

Growth mixture modeling (GMM) is a latent variable method to parse heterogeneity in a sample. It is an extension of conventional latent growth modeling, which treats heterogeneity in the parameters as explained by an unobserved (latent) class variable. We explored four different parameterizations of growth mixture models for the combined sample of toddlers with LD and TD. The simplest, the latent class growth curve analysis (LCGA), allows only the mean values of the intercept and slope terms to vary among classes. Subsequent models relaxed (a) within-class constraints on the variance of the intercept and slope factors (GMM1), (b) within-class constraints on the covariance of the intercept and slope factors (GMM2), (c) between-class constraints on the variance of the intercept and slope factors (GMM3), and (d) between-class constraints on the covariance of the intercept and slope factors (GMM4). Within each parameterization, models with up to four classes were enumerated and compared using the following relative fit indices: the loglikelihood, the Bayesian information criterion, the adjusted Bayesian information criterion, Aikake's information criterion, and the consistent Aikake's information criterion. Bayes' factor and the approximate weight of evidence criterion were used to assist in interpretation of information criteria. Finally, the Vuong-Lo-Mendell-Rubin likelihood ratio test was used to assess the degree of improvement in model fit with additional classes. Maximum Likelihood Estimation was used, which uses the available data from each case, rather than listwise deletion.

The best candidate models were further evaluated based on their classification quality and degree of distinction between classes, including entropy, average posterior probability, odds of correct classification, modal class assignment proportions, and separation of each parameter allowed to vary between classes. Models were also evaluated for robustness to slight changes in model specification. The best solution was selected based on fit indices and interpretability. Finally, given the high level of entropy of the resulting models, diagnostic outcome (ASD, non-ASD delays, no delays) was evaluated as a distal outcome using chi-

square tests of independence (or Fisher's Exact Test when the expected cell size was less than five).

To avoid excluding children without 36-month data from outcome analyses, we used the last observation carried forward approach. Given that the diagnostic outcome category was generally stable at 36 months from 18 months (68 of 80 children) and 24 months (73 of 80 children), this approach was selected to minimize bias. We also performed sensitivity analyses including only children who had data collected at 36-months. All growth mixture model analyses were completed in MPlus version 7.4 (Muthén & Muthén, 2012); other analyses and data management were performed in SAS/STAT version 9.3 and SPSS version 25. Mplus codes for GMM are included in Supplemental Information.

3. Results

3.1 Outcome at 36 months

Outcome data for all participants classified as LD or TD at 18 months are as follows. Among toddlers with early LD, 40% (n=12) met criteria for ASD and 37% (n=11) met criteria for non-ASD delays, while 23% (n=7) were classified as No Delay. Of the 11 toddlers with early LD and non-ASD delay outcomes, eight had receptive or expressive language T-scores on the MSEL ≥ 35 , one met clinician judgment of non-ASD social communication concerns, and two had Fine Motor T-scores on the MSEL ≥ 35 . One participant who was initially enrolled in the TD group met criteria for non-ASD delays at 36 months due to clinician judgment of non-ASD social communication concerns and fine motor delay (MSEL Fine Motor T-score=32). All other TD toddlers were classified as No Delay at outcome.

Of the 11 toddlers for whom we did not obtain 36-month outcome data, six (55%) were classified as No Delay, four (36%) had non-ASD delays, and one (9%) was classified as ASD. Among the 80 children who did complete the final visit, 61 (76%) were classified as No Delay, eight (10%) had non-ASD delays, and 11 (14%) were classified as ASD.

3.2 Quantification of the heterogeneity in developmental trajectories

Based on the relative fit indices, candidates were selected from among the 20 available models for each MSEL domain (see Table 2 for candidate models, results from all models are shown in Supplemental Table S1). Classification quality was assessed for each of the seven candidate models (see Supplemental Table S2). Classification quality was unacceptable for the NVMA GMM3 2-class, and class sizes were too small for the NVMA LCGA 3-class and the VMA GMM1 4-class, so these models were discarded. Finally, separation of the classes was calculated for the remaining models (see Supplemental Table S3). Based on this information, the best models were the LCGA 2-class model for NVMA (Age-Expected, 82%, and Delayed, 18%), and the LCGA 3-class model for VMA (Age-Expected, 66%, Delay Catch-Up, 23%, and Delayed, 11%) (see Figure 1 for illustration of trajectories).

3.3 Trajectory Class and Outcome Classification

NVMA class assignment was related to outcome (overall Fisher's Exact Test, $p < .001$); without exception, toddlers with a No Delay outcome were classified in the Age-Expected NVMA trajectory (see Table 3). While the proportion of participants assigned to the Age-Expected NVMA trajectory was lower among toddlers with ASD outcome (17%) than among toddlers with non-ASD delays (50%), this difference did not reach statistical significance (Fisher's Exact test, $p = .19$). Therefore, while NVMA class membership was associated with whether a child was in the No Delay outcome group, membership in the Delayed NVMA trajectory class was not uniquely related to ASD or non-ASD outcomes in this sample. Similar results were obtained in sensitivity analyses excluding participants without 36-month follow-up data (data not shown).

VMA class assignment was also significantly related to outcome (overall Fisher's Exact Test, $p < .0001$). Children with a No Delay outcome were primarily assigned to the Age-Expected VMA trajectory class (88%), with 12% classified in the Delay Catch-up VMA class, a distribution that differed significantly from that observed in both the ASD (Fisher's exact, $p < .0001$) and non-ASD delay (Fisher's exact, $p < .0001$) groups (see Table 3). In contrast to NVMA, however, the two delay outcomes were uniquely related to VMA trajectory. The majority (83%) of children with non-ASD delay were assigned to the Delay Catch-up VMA class, compared to only 25% of children who received an ASD diagnosis (Fisher's exact test, $p = .0028$). Stated alternatively, membership in the Delayed VMA trajectory class was uniquely associated with an ASD diagnosis (90%), as compared to the Delay Catch-up VMA trajectory class (23% diagnosed with ASD). Similar results were obtained in sensitivity analyses excluding participants without 36-month follow-up data (data not shown).

3.4 Joint Distributions of Trajectory Classes

The joint distributions of most likely class assignment are shown in Table 4. VMA and NVMA class membership were strongly related (Fisher's Exact Test, $p < .0001$). Children in the more advanced class(es) in one domain were generally assigned to the more advanced class(es) in the other domain. A large portion (67%) of toddlers who started with low VMA scores and then caught up (VMA Delay Catch-Up Class) had age-expected NVMA score trajectories.

4. Discussion

We utilized a person-centered, latent variable approach to explore cognitive development and trajectory heterogeneity in a study of toddlers with early language delay followed prospectively for outcomes such as ASD. Among toddlers recruited for language delay at 12 or 18 months, 40% were diagnosed with ASD, 37% showed non-ASD delays, and 23% were categorized as having no delays at 36 months of age. Although a delay in language itself is not pathognomonic for ASD (Buschmann et al., 2008), prior studies that have included ASD as a possible outcome indicate that up to 23% of those with early language delay are later diagnosed with the disorder (Miniscalco et al., 2006). The slightly higher rate in this study may relate to the nature of study recruitment and inclusion criteria, with significant delays in

both receptive and expressive language required. Alternatively, given the relatively small sample, it may simply be a result of normal variation.

This is the first study we are aware of to use growth mixture modeling to describe cognitive growth in toddlers with early LD. Consistent with previous studies of toddlers at increased genetic risk for ASD (Brian et al., 2014; Landa et al., 2012), our expectation that the heterogeneity in cognitive trajectories would be better explained by multiple classes was supported. We found evidence for more heterogeneity in verbal development than nonverbal development; VMA trajectories were age-expected, catch-up, and delayed, while NVMA classes were only age-expected and delayed. This is, perhaps unsurprising given that children were recruited based on language delay; this may be considered something like regression to the mean.

In addition to differences in study population, this study differs from the work of Landa et al. (2012) and Brian et al. (2014) in sample size, which inherently limits the ability to identify classes in GMM. Nevertheless, the delayed classes to which the LD toddlers were most commonly assigned had a growth pattern similar to the developmental slowing class found by Landa et al. (2012) and the declining class found by Brian et al. (2014), in which MSEL scores grew slowly over time across domains. Further, consistent with these previous studies, we found that the delayed VMA trajectory was uniquely related to an ASD outcome (versus non-ASD delay). Children eventually diagnosed with ASD were less likely than those not diagnosed with ASD to belong to the catch-up class, indicating that the delayed trajectory of VMA in children with language delay may be predictive of later ASD diagnosis. NVMA trajectory was nominally associated with outcome—50% of toddlers who had a non-ASD delay were assigned to the average NVMA class, compared to only 17% of the toddlers who received an ASD diagnosis ($p=.19$). Given the small sample size, this may be attributed to Type II error. Thus, our findings suggest that similar patterns of cognitive development are manifest in toddlers with early language delay and at-risk infant siblings, and that those developmental patterns relate similarly to eventual ASD outcome. While more research is needed to compare directly toddlers with significant LD to other at-risk populations, including infant siblings later diagnosed with ASD, the present study contributes an important piece of evidence suggesting similarities between the risk groups.

Finally, we examined the extent to which patterns of verbal and nonverbal cognitive development co-occurred in our sample. As expected, there was a strong association between VMA class membership and NVMA class membership. Further, age-appropriate nonverbal skills were present in a large portion of toddlers with early LD who caught up and eventually reached age-expected language levels. This suggests a relationship between nonverbal cognition and improvement in language skills in toddlers with early LD, which is consistent with prior studies indicating a link between nonverbal cognition and later language skills in young children (Oliver, Dale, & Plomin, 2004; Thurm, Lord, Lee, & Newschaffer, 2007).

4.1 Limitations

The primary limitation of this study is the small sample size, particularly in the LD group. This limited the evaluation of more complex models, and as with any statistical endeavor,

increased the probability of Type II error. Additionally, some data were missing (11 of 91 participants did not complete the final study visit), and although this was addressed using last observation carried forward and maximum likelihood estimation, it is possible these missing data biased the results. Certainly, evaluations beyond 3 years of age would also have increased the statistical power and clinical significance of outcome groups identified.

Further, while the MSEL is a widely used measure of verbal and nonverbal development in young children, with research supporting its validity (Swineford, Guthrie, & Thurm, 2015), it has limitations. For example, the amount of test information (i.e., the number of items used) at a given age range is limited, reducing its accuracy in capturing the nuances of developmental skills.

In this cohort, mothers in the TD study group were significantly more likely to have at least some graduate-level education than mothers in the LD group. Though the difference in maternal education is likely not impactful here (both groups highly educated, but the difference is between at least some college and at least some graduate school), it is possible that the notably high education levels in the TD group may affect the generalizability of our results. Future research comparing toddlers with LD and TD should include samples with increased heterogeneity in familial education and socioeconomic background.

4.2 Implications and Future Directions

This is the first study to examine cognitive development and associated heterogeneity using growth mixture modeling in a sample of toddlers with early language delays followed prospectively for outcomes such as ASD. Given the range of neurodevelopmental outcomes described in children with early LD (Miniscalco et al., 2006), findings from this study provide important information about the diversity of cognitive trajectories in toddlers with early LD. These results are clinically significant; understanding how and when early changes in cognitive development occur may inform approaches for developmental surveillance of toddlers with early LD, which is particularly important given the relationship between patterns of early development and outcomes such as ASD. The trajectories outlined in this study provide useful information regarding the timing of nonverbal and verbal skill acquisition in individuals with early LD that may inform the development and implementation of early intervention approaches. Findings additionally lend support to prior research linking impaired cognitive development to ASD in other at-risk infants.

Importantly, future research should extend the present findings and examine predictors of trajectory class membership. One population-based latent class growth mixture model analysis of MSEL data in infants exists; while it uncovered much heterogeneity as well as early risk factors for membership in delayed trajectory classes (Nishimura, Takei, Tsuchiya, Asano, & Mori, 2016), the results do not provide information about children at risk specifically for ASD. Examining predictors of developmental trajectories in samples of individuals at-risk for neurodevelopmental disorders may inform understanding of early vulnerabilities that may relate to patterns of development over time and associated outcomes. Further, examining early predictors of cognitive trajectories may help elucidate why some individuals with early delays “catch up” while others do not.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgements

We thank the participants and their families, as well as the staff of the Pediatrics and Developmental Neuroscience Branch.

Funding: This work was supported by the National Institute of Mental Health Intramural Research Program (ZIA MH002868).

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Highlights

- In a study of toddlers with early language delays, growth mixture models revealed heterogeneity in cognitive development.
- The best-fitting models comprised two nonverbal and three verbal classes.
- Membership in the delayed verbal cognitive trajectory class was related to ASD diagnosis.

Figure 1.
Trajectories of verbal (Panel A) and nonverbal (Panel B) MSEL scores. MSEL=Mullen Scales of Early Learning.

Table 1.

Participant Characteristics

Characteristic	LD (N=30)	TD (N=61)
Male, <i>n</i> (%)	21 (70%)	35 (57%)
Age at 18-month visit, <i>M</i> ± <i>SD</i> (months)	18.77 ± 1.40	18.51 ± 0.97
Maternal Education, <i>n</i> (%) ^a		
High school	2 (7%)	2 (3%)
At least some college	15 (50%)	18 (30%)
At least some graduate school	12 (40%)	41 (67%)

^aMissing maternal education data for one participant in the LD group. Mothers in the TD group were significantly more likely to have at least some graduate-level education ($p=.02$).

Note: Groups did not differ at any visit in age (18 months: $p=.36$; 24 months: $p=.35$; 36 months: $p=.28$) or sex (18 months: $p=.25$; 24 months: $p=.36$; 36 months: $p=.43$).

Table 2.

Relative Fit Indices for Candidate Models

Model	Parameters	Loglikelihood	AIC	BIC	ABIC	Vuong-Lo-Mendell-Rubin LRT p-value
<i>NVMA</i>						
LCGA 2-class	8	−690	1397	1417	1391	0.04
GMM3 2-class	12	−666	1357	1387	1349	0.01
LCGA 3-class	11	−676	1375	1402	1368	0.04
<i>VMA</i>						
GMM3 2-class	12	−751	1526	1556	1518	0.00
LCGA 3-class	11	−771	1563	1591	1556	0.02
LCGA 4-class	14	−757	1542	1577	1533	0.06
GMM1 4-class	16	−746	1524	1564	1514	0.01

Note. Loglikelihood is an index of the probability of the data as a function of the parameters; the algorithm attempts to maximize this. AIC = Akaike's Information Criterion, smaller values are better. (A)BIC = (adjusted) Bayesian Information Criterion, smaller values are better. Vuong-Lo-Mendell-Rubin LRT = likelihood ratio test, small p-value indicates better fit for this number of classes versus a model with one fewer class.

Table 3.

Trajectory Class Assignment by Outcome

36-Month Outcome	VMA Class Assignment				NVMA Class Assignment		
	Delayed	Delay Catch-Up	Age-Expected	Total	Delayed	Age-Expected	Total
ASD	9	3	0	12	10	2	12
Non-ASD Delay	1	10	1	12	6	6	12
No Delay	0	8	59	67	0	67	67
<i>Total</i>	<i>10</i>	<i>21</i>	<i>60</i>	<i>91</i>	<i>16</i>	<i>75</i>	<i>91</i>

Table 4.

Joint Distributions of Trajectories

NVMA Class	VMA Class			<i>Total</i>
	Delayed	Delay Catch-Up	Age-Expected	
Delayed	9	7	0	<i>16</i>
Age-Expected	1	14	60	<i>75</i>
<i>Total</i>	<i>10</i>	<i>21</i>	<i>60</i>	<i>91</i>