The Administrative Prevalence of Autism Spectrum Disorders in Nevada School Districts: A Pooled Time Series Analysis, 1996-2004

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Abstract

Objective: To examine the administrative prevalence of Autism Spectrum Disorders (ASD) in all seventeen school districts in Nevada during the period of 1996 to 2004.

Methods: Normalized administrative prevalence rates (per 1,000 children ages 6-17) for ASD, Mental Retardation (MR), Learning Disability (LD), and Speech and Language Impairment (SLI) were calculated. Covariates for board certified pediatricians per 1,000 students, Federal special education funding per student, and other measures of school resources were employed. Models were estimated with pooled Ordinary Least Squares (OLS) regression with panel corrected standard errors. A separate analysis compared pooled OLS results to results from Latent Growth Curve models (LCGM)

Results: The average administrative prevalence of ASD in Nevada school districts increased from .56 per 1,000 in 1996 to 2.37 per 1,000 in 2004. The upward trajectory of ASD prevalence during the time series was not associated with declines in MR, LD or SLI prevalence. Federal funds distributed partly for detection of disabilities was associated with ASD prevalence ($p<0.01$) (results were not due to endogeneity). The concentration of pediatricians in each school district, changes in the regulatory definition of ASD, and real salaries for personnel were shown to have no effect. The results of the pooled OLS models were robust when compared to the Latent Growth Curve models.

Keywords: Epidemiology; Disabilities; Autism; Prevalence; Diagnostic Substitution

Introduction

Since the late 1990s, public health officials and researchers have drawn attention to the growth in the administrative prevalence of autism spectrum disorders (ASD) in U.S. schools.[1] The interpretation of trends in administrative prevalence has been the subject of controversy in the scholarly literature, however. While recognizing the potential limitations of administrative reporting data, some analysts claim that the prevalence trends from schools are suggestive of the increase in the disease burden of ASD among children in the U.S. (Newschaffer, Falib & Gurney 2005). Other scholars question this interpretation, claiming that the increase of administrative prevalence in ASD is due to "diagnostic substitution," a process wherein children formally misclassified as having mental retardation (MR), learning disabilities (LD), speech and language impairment (SLI), or other disabilities were shifted to the autism category after its creation in the early 1990s (Shattuck 2006; National Research Council 2001: 25). According to this hypothesis, the increase in the administrative prevalence of ASD reflects the offsetting declines in the reporting of other disability counts by special education officials.

The debate concerning the interpretation of administrative prevalence of ASD in U.S. schools has been subject to certain methodological limitations. Since the hypothesis concerning diagnostic substitution partly involves a claim about misdiagnosis (National Research Council 2001), it would seem important to adjust for variables that might mediate the capacity of school officials or professionals to identify (retroactively or contemporaneously) the misclassification of autistic children. Certainly, school districts with a greater concentration of professional expertise might be more effective in identifying autistic children and in correcting inaccurate classification. School districts that receive more

[1] For a review of the recent debate about the causes of ASD, see Gillberg (2005); Larsson et al (2005); Reichenberg et al (2006).

funding may also have more resources for identifying disabled children and for in-service training so that school officials better understand the differences between ASD and other developmental disorders. Yet, although research has found that wealth and funding characteristics of districts (or states) may play a role in the detection of ASD (Palmer, Blanchard, Jean & Mandell 2005; Tuman, Roth-Johnson, & Vecchio 2006), the effects of these influences have not been modeled in studies that examine diagnostic substitution and ASD prevalence (Shattuck 2006). Similarly, to our knowledge, previous research has not adjusted for relative differences in the concentration of professional expertise across school districts.

The extant literature on administrative prevalence of ASD has also been limited by neglecting to examine changes in the administrative definition of autism used by school officials. In recent years, some states implemented new special education regulations that broadened the definition of autism to include atypical autism, Asperger's, and Pervasive Developmental Disorder-Not Otherwise Specified (PDD-NOS). Because changes in administrative regulations might be expected to influence prevalence rates (Newschaffer, Falib & Gurney 2005; Shattuck 2006), it is important to control for such changes in a research design. Finally, virtually no research on prevalence has been completed on Nevada. The one previous study of ASD prevalence in Nevada covered students in only one grade, across only two school districts, for a single year, 1998 (Chang, Crothers, Lai & Lamm 2003).

In this study, we attempt to address gaps in the literature on administrative prevalence in Nevada. Utilizing a data set that pools observations from all seventeen school districts in Nevada for the period 1996 through 2004, we investigate changes in the administrative prevalence of ASD among school children ages 6 through 17. The study has several objectives. First, we test the hypothesis concerning diagnostic substitution and ASD prevalence, while adjusting for other social and economic factors across school districts, including the concentration of professional expertise, funding, and district size. Second, the study attempts to provide a more fine-grained extension of previous research (Palmer, Blanchard, Jean, & Mandell 2005) regarding the relationship between funding and detection of ASD. In particular, we focus on the relationship between ASD prevalence and Federal special education funds that are specifically tied to the identification of disabled children. Third, we attempt to gauge whether changes in the administrative definition of autism in the state regulations for special education eligibility have had any effect on the administrative prevalence over time.

Methods

Prevalence Measures

Normalized administrative prevalence rates (per 1,000 students ages 6 through 17) were calculated for ASD, MR, SLI and LD for each school district and year. Data used for the numerators for ASD, MR, SLI and LD rates were collected for the period of 1995 through 2004 from records in the Nevada State Department of Education, Office of Special Education, Elementary and Secondary Education, and School Improvement Programs (1994-2007). The records include annual school district counts (as of December 1 of each year of the series) from all seventeen Nevada districts as reported to the Nevada State Department of Education; each report lists the number of children in each district and year, by category of disability, that receive special education services pursuant to the Individuals with Disabilities Education Act. For the purposes of reporting and providing services, officials must determine the child's primary disability; thus, for example, a child may not be reported in the ASD and LD categories simultaneously, but only in one disability category each year. Data for the Developmental Delay (DD) category were not collected because our study covers children ages 6 through 17, and the Nevada state regulations prohibit schools from reporting a child age 6 or older in the DD category (State of Nevada 2007). In addition, children ages 3-5 and 18-21 were excluded due to incomplete coverage of school enrollment data (used for the denominator to calculate prevalence rates) for children in those age ranges; however, pooling the remaining observations for children ages 6-17 was reasonable as no discernible shift could be detected in ASD counts between the ages of 11 and 12 (Laidler 2005).

Because complete county level data for all years in the study were not available from the U.S. Census Bureau for normalization of the data, we employed total student enrollment (sum of public and private) in grades 1 through 12, in each district and year, as the denominators to calculate the normalized administrative prevalence rates of ASD, MR, SLI and LD. This technique has been

used in previous studies where the school district is the unit of analysis (Harcourt 2006). School enrollment data were collected from Nevada State Department of Education (Nevada State Board of Education 1994-2005).

Covariates Employed in the Regression Models

We include a number of covariates in the models to adjust for differences across districts that might influence the detection of autism and the process of diagnostic substitution. First, to assess the effects of levels of expertise in the medical community, we employed a measure for the number of board-certified pediatricians (per 1,000 children enrolled grades 1 through 12) in each district. Because new diagnostic criteria for autism were introduced in 1994 with the DSM-IV, we have included observations only for those pediatricians who were board certified after 1994. The pediatrician data have been lagged by one year to account for the lengthy process involved in referring a child for private evaluation, school evaluation and determination of eligibility for public services. The source for this is the American Board of Pediatrics (American Board of Pediatrics 2006).

Second, as a proxy measure for diagnostic capacity within school districts, we included a covariate for the mean inflation-adjusted (real) salary of all licensed personnel for each district for each year, lagged by one year (Palmer, Blanchard, Jean & Mandell 2005). The data include mean real salaries for school psychologists, speech pathologists, occupational therapists, special education teachers, and regular teachers. Higher salaries in districts may be associated with licensed personnel who have more years of service and education, on average. The salaries data were obtained from the Nevada State Board of Education (1994-2005) and the inflation data (for conversion to real salaries) were obtained from the U.S. Department of Labor, Bureau of Labor Statistics (2007).

Third, to tap the effects of funding in each district, we employed a measure for the natural log of total Part B funding per enrolled student given to each district, adjusted for inflation and lagged by one year. Federal Part B funding is used partly to identify children who might have a disability and for special education in each district. We expect a lagged effect on prevalence because funding in the present year pays for advertising to raise awareness about disability and for evaluation; the advertising campaigns do not immediately lead to evaluation, and multiple evaluations may be sought. Therefore investments made in Part B "child find" activities in the current year may not influence disability counts until the following year. The Hausman test indicated that there is no endogeneity problem between Part B funding and ASD prevalence (i.e., that ASD prevalence is a *determinant* of Part B funding). Data for Part B funding, for each district and year, were obtained from unpublished records in the Nevada State Department of Education (Nevada State Department of Education, Office of Special Education, Elementary and Secondary Education & School Improvement Programs 1994-2007) and normalized with school enrollment data (Nevada State Board of Education 1994-2005). The deflator for the Part B funding measure (to adjust for inflation) was obtained from the U.S. Department of Labor, Bureau of Labor Statistics (2007).

Fourth, because larger school districts may have a concentration of resources that might facilitate changes in diagnoses, we included a covariate for school district size based upon total enrollment (public and private) in grades 1 through 12. To avoid distortion caused by extreme values in the enrollment data for large districts, we employed the natural log of school enrollment in the regression model reported in Table 3. However, diagnostic tests for the substitution models (Appendix A, Table 2) suggested that the log of enrollment was highly collinear with the covariate for pediatricians. To avoid multicollinearity, in the substitution regression models (Table 2), we employed a recoded ordinal variable for school enrollment: 0-999 students = "1," 1,000-4,999 students = "2," 5,000-9,999 students = "3," and >10,000 students = "4."

Finally, we included a covariate to control for changes in the administrative definition of autism used by Nevada school officials. In the year 2000, the Nevada Board of Education amended the administrative definition of autism to include "…autistic disorder, [A]sperger's disorder, atypical autism, pervasive developmental disorder [PDD], and other disorders that share the characteristics described [in the definition of autism]" (Nevada Administrative Code 2007, Chapter 388; Alred 2007). Anecdotal evidence suggests that prior to implementation of the changed definition, school eligibility teams may have already certified and counted children with Asperger's disorder and Pervasive Developmental Disorder-Not Otherwise Specified (PDD-NOS) as eligible for special education. Nevertheless, the statistical

effect of the change in the definition of autism in Nevada has not been assessed systematically. Since the administrative change in the definition of ASD did not take effect until several months into the year 2000, we assume that any effect would not have been present on ASD counts until the following year, 2001. To capture this effect, we include a dummy variable, coded "1" for the year 2001, and "0" for all other years in the time series. An alternative coding rule – to code all years after 2000 as "1," and the year 2000 and prior years as "0" – could not be implemented because it produced results that were too collinear with the covariates for funding and salaries.

Estimation Methods

The dataset for this study pools observations of prevalence from seventeen Nevada schools districts over a nine-year period. As such, it can be described as a pooled cross-sectional time-series. Such data may exhibit heteroscedasticity, correlation of the errors terms across units (Cook-Weisberg Chi-Square test for heteroscedasticity in LD, MR, SLI and ASD models, $p<.001$). To address this issue, we employed linear regression (Ordinary Least Squares, or OLS) with "panel corrected" standard errors, which has been shown to correct for heteroscedasticity and contemporaneous correlation in the data (Beck & Katz 1995). In addition, pooled cross-sectional time-series data are prone to the problem of autocorrelation, correlation of the error terms across time. To control for autocorrelation, we include a lagged dependent (endogenous) variable as a covariate in all models (Beck & Katz 1996). Estimation of the model with a lagged endogenous covariate successfully controls for autocorrelation.

The practical effect of using lagged endogenous covariates to adjust for autocorrelation is the loss of one year from the time series explained by each model. Data for ASD prevalence is available for 1995, but data for the denominators used to calculate all prevalence measures are not available prior to 1995; accordingly, lagged endogenous covariates begin in 1995, which implies that the first year of observations explained by the models begins in 1996. Truncation of the time series to 1996 to 2004, does, however, have the advantage of holding constant the standard diagnostic criteria employed by school assessment teams. The Diagnostic and Statistical Manual-IV (DSM-IV) criteria for ASD were revised in mid-1994, became diffused the following year, and have remained constant throughout the remainder of the time-series (Szatmari 1997; Alred 2007).

In the Appendix, we present the results of an additional analysis that compares the results of the pooled OLS model to (1) results from models where data are first-differenced and expressed as change over time, and (2) results from a Latent Growth Curve Model (LCGM). The sensitivity analysis suggests that results of the pooled OLS are robust. In addition, the Appendix present tests for autocorrelation (estimation with, and without the lagged endogenous covariates), multicollinearity, and diagnoses of unit effects.

Results

Trends in ASD Administrative Prevalence

Table 1 presents the trend in the average administrative prevalence rate of autism spectrum disorder (ASD) across all seventeen school districts in Nevada. As one can see from the data, the mean ASD prevalence rate increased from .56 per 1,000 in 1996 to 2.37 per 1,000 in 2004. Prevalence, on average, increased during the entire study period, and after adjusting for autocorrelation, the change per year remained statistically significant (the coefficient for "year" = .07, panel-corrected standard error = .03, $p<0.04$; coefficient for the lagged dependent variable, ASD Prevalence $_{t-1}$, = 0.76, panel-corrected standard error = .12, $p<0.001$)

Table 1 .Mean Administrative Prevalence of ASD (per 1,000), Nevada School Districts, 1995-2004

1995	.27
1996	.56
1997	.81
1998	.59
1999	.84
2000	1.31
2001	1.40
2002	1.74
2003	2.16
2004	2.37

The increase in ASD prevalence was not confined to a small number of school districts. More than eighty percent of Nevada school districts experienced an increase in autism rates. Moreover, nearly all (sixteen out of seventeen) school districts reported prevalence for some years during the time series. Esmeralda School District, which enrolls fewer than 80 students, was the only district that reported zero prevalence during all years of the study. Given that the upper bound of ASD prevalence is estimated in recent epidemiological studies to be one in 150 (Centers for Disease Control and Prevention 2007), we would not necessarily

expect to detect autism in a district as small as Esmeralda.

Diagnostic Substitution and ASD Prevalence

Next, we discuss the results for the diagnostic substitution models. It should be recalled here that the diagnostic substitution hypothesis suggests that increases in ASD prevalence are associated with the downward trajectory of MR, LD and SLI prevalence. Given that ASD was *increasing* during the time series, to test the hypothesis of diagnostic substitution in a statistical regression design, MR, LD and SLI prevalence rates must be defined as *endogenous* variables that are regressed on ASD prevalence (defined as the exogenous variable). If diagnostic substitution is present, then one expects a negative association between the coefficient for ASD prevalence and MR, LD or SLI prevalence (i.e., a negative and significant coefficient for ASD prevalence would imply that for every one-unit increase in ASD prevalence, there is a statistically significant decline in MR, LD and SLI prevalence, on average, during the time series – which would be suggestive of diagnostic substitution).

Table 2 (reported in Appendix A) presents the findings for reduced and full models for MR prevalence. In the reduced model, only the lagged endogenous variable (MR prevalence $_{t-1}$) and ASD prevalence are employed as covariates; the full model is estimated with a lagged endogenous variable, and with a covariate for ASD prevalence and several other covariates of interest. As one can see from the data, the results were stable in both the reduced and full models. In both trials, the coefficient for the lagged dependent variable, the prevalence of MR in the previous year, was statistically significant ($p<0.001$). This suggests a strong association in the prevalence of MR from year-to-year among school districts in this sample. After controlling for the effects of autocorrelation, neither the reduced or full models in Appendix A, Table 2 provided evidence for the effects of diagnostic substitution. As the data in Table 2 demonstrate, the coefficient for ASD prevalence in both the reduced and full trials was unexpectedly positive but failed to achieve statistical significance ($p>0.05$). Thus, for every one-unit change in ASD prevalence there was no significant effect, on average, on MR prevalence, a finding inconsistent with the hypothesis of diagnostic substitution. The coefficients for the other covariates in the model, prevalence of board certified pediatricians, real salaries of licensed personnel, district size, and real Part B funding per capita (logged), all failed to achieve statistical significance.

Table 2 (Appendix A) also presents the results for the LD and SLI models. As with the MR model, we estimated both reduced and full diagnostic substitution models for LD and SLI. The results were extremely similar to those reported in the MR trials. In both the reduced and full models for LD and SLI, the coefficients for the lagged dependent variables, LD and SLI in the previous year, were statistically significant ($p<0.001$). Likewise, in both the reduced and full LD and SLI models, the coefficients for ASD prevalence were positive but not statistically significant ($p>0.05$). In the LD model, the coefficient for district size was negative and statistically significant, which suggests that higher rates of LD prevalence are concentrated in smaller districts ($p<0.05$). With the exception of district size, however, the coefficients for other covariates in the full models for LD and SLI were not statistically significant ($p>0.05$). Overall, the findings for the MR, LD and SLI models are strongly suggestive that increases in administrative prevalence of ASD were not due to diagnostic substitution.[2]

[2] In separate trials, we also estimated pooled cross-sectional time-series models (full and reduced) for Multiple Impaired (MI), Other Health Impairmed (OHI), and Traumatic Brain Injury (TBI); the same covariates as in Appendix A, Table 2, were employed. Consistent with our results for Mental Retardation, Speech and Language Impairment, and Learning Disabilities, the results from these trials indicated that the coefficient for ASD prevalence had no effect ($p>0.05$) on MI, OHI and TBI prevalence for children ages 6-17, which suggests, again, that the upward trajectory of ASD prevalence was not associated with declines in MI, OHI or TBI prevalence. In addition, we examined the rates of change for MI, OHI and TBI prevalence in a latent growth curve model (as in Appendix B), with no other covariate than time. The rates of change in MI, OHI, and TBI were positive but not statistically significant ($p>0.05$).

Table 3: Administrative Prevalence of ASD across School Districts, 1996-2004

Lagged Endogenous Covariate $_{t-1}$	.76*** (.12)
Board Pediatricians per 1,000 $_{t-1}$	.01 (.31)
Real Salaries $_{t-1}$	-.000007 (.00003)
Real Part B Funding per student (log) $_{t-1}$	.49** (.17)
District Size (log) $_{t-1}$	.08**
Change in Administrative Definition of Autism	-.18 (.13)
Constant	-2.12* (1.22)
N=153	
Wald Chi-Square	316.10***
Adjusted R^2	.59

Note: Entries are unstandardized OLS regression coefficients; panel corrected standard errors reported in parentheses below regression coefficients.
***p<.001; ** p<.01; p<.05

Characteristics of School Districts and ASD Prevalence

We also examined whether the social and economic characteristics of school districts in Nevada were associated with detection of autism (Table 3). As expected, the coefficient for ASD prevalence in the previous year was large, positive and statistically significant. During the period in question, there was a strong association between levels of ASD prevalence from year to year, and estimation of the model with the lagged endogenous covariate successfully controlled for the effects of autocorrelation in the data set.

In addition, the coefficient for the natural log of inflation-adjusted Part B funding per student in each district, lagged by one year, was positive and statistically significant. Thus, the model in Table 3 provides support for the supposition that higher levels of federal special education funding may help to detect ASD in the school-age population. Given that a portion of Part B funds is to be used for raising awareness, for evaluation, and for identifying children who may be at risk of being disabled, it is not surprising that there was a positive association, on average, between (lagged) funding levels per student and prevalence across Nevada school districts from 1996 through 2004.

The coefficient for district size, as measured by the natural log of enrolled students (public and private) in each school district, was also positive and statistically significant (p<0.01). Consistent with the findings of a study of Texas schools (Palmer, Blanchard, Jean & Mandell 2005), the results from Nevada suggest that the even after adjusting for many factors, the larger the district, the higher the ASD administrative prevalence during the time series. To demonstrate this, in separate trials we estimated the model in Table 3 with two additional covariates (suggested by Palmer, Blanchard, Jean & Mandell 2005) that might mediate the effect of school district size: (1) annual assessed property values per student in each district (lagged by one year) (State of Nevada, Department of Taxation 1994-2005), and (2) annual poverty rate among children ages 5-17 in each county (lagged by one year) (U.S. Census Bureau 1997-2004). In this trial, the coefficients for property values and poverty were not statistically significant, the coefficient for school enrollment remained positive and significant (p<0.01), while results for all other covariates in the full model remained completely consistent with results in Table 3. Thus, even when wealth and poverty were controlled for with other covariates, school enrollment is positively associated with ASD prevalence. This suggests that the effects of district size are not due to the variation in income levels or the local tax base across school districts. A reasonable interpretation is that some of this association between enrollment size and prevalence might be due to the presence of advocacy organizations in larger districts. We elaborate more on this point in the conclusion of the paper.

Beyond funding and district size, the model does not provide support for the claim that greater concentration of professional expertise at the district level leads to better detection. The coefficient for board-certified pediatricians per 1,000 students (lagged) was small and failed to achieve statistical significance (p>0.05). This shows that school districts that had a greater density of board certified pediatricians were not more likely to have higher autism rates. To further assess the effects of medical expertise, in a separate trial we removed the covariate for board-certified pediatricians, and then replaced it (in the full model) with a covariate for the number of pediatricians per 1,000 students, lagged by one year, in each district and year (State of Nevada, Board of Medical Examiners 2006). The coefficient for this variable also failed to achieve statistical significance (p>0.05). Likewise, the coefficient for real mean salaries for licensed personnel in each district was also not statistically significant (p>0.05).

Finally, changes in the administrative definition of autism did not have an effect on prevalence. The coefficient for the administrative change variable was unexpectedly negative, but its effects were not significant (p>0.05). This suggests that ASD prevalence rates during the year when the administrative definition of ASD changed were not significantly different from the

average prevalence in other years in the study. However, given that the change in the administrative definition of autism was implemented only in 2001, it is possible that the effects of that change will be measurable in a study with a longer time series. Certainly, future research should assess this possibility.

Discussion

In this study, we have attempted to improve understanding of the administrative prevalence of ASD in Nevada. The results indicate that districts that are larger and which receive more federal special education funding might do a better job of detecting autism among school-age individuals in the local population. At the same time, the findings of the research do not indicate that the dramatic increase in autism rates in Nevada were due to diagnostic substitution. Indeed, the upward trajectory in ASD administrative prevalence during the study period was not associated statistically with declines in MR, LD and SLI. Similarly, concentration of professional expertise, and changes in the administrative definition of autism, do not appear to have any significant effect on ASD prevalence rates across school districts in Nevada.

The findings of the study give rise to several implications about how future research on ASD prevalence might be refined and extended. One issue has to do with understanding the relationship between enrollment size and ASD prevalence. The effects of district size may be attributed to several factors that co-vary with population, including the presence of more educated parents in larger districts. Nevertheless, it is reasonable to suppose that some of the effect from enrollment levels may be due to the cross-sectional variation in density levels of advocacy organizations (Fiedler & Swanger 2000). Complete data are not available for all years, but recent evidence suggests that advocacy organizations – including ones that focus on autism – are concentrated in Nevada's three largest schools districts, Clark, Washoe and Carson City (e.g., the organization Parents Educating Parents). However, in smaller districts, advocacy organizations have few members and no professional staff or offices. To the extent that such organizations raise awareness in the general population and assist parents in obtaining services for children (Tuman, Roth-Johnson & Vecchio 2006), their concentration in larger districts may have the effect of consistently improving detection of ASD, with attendant consequences for the growth in prevalence rates. Despite the paucity of data on disability organizations, future researchers should attempt to assess the effects of such organizations while also investigating other possibilities for the association between school enrollment per district and ASD prevalence.

A second implication concerns the effects of funding on the detection of ASD among school children. Although previous research has suggested the importance of local school revenues for detecting ASD, our study is the first to demonstrate a meaningful effect of Federal Part B funds; given that some Part B funds are specifically intended for use in identification of disabilities among school children, this is an important finding. Yet, the data on Part B funding per student in Nevada suggest that there is an imbalance in the distribution of these funds across districts. Our results suggest that efforts to correct some of the imbalance in the distribution of Part B funds across districts – either through changes in the funding formula or through supplementary funds targeted for surveillance of disabilities in each district – might improve detection of ASD in the school age population in some areas of Nevada.

More broadly, the findings of this study contribute to the debate over the relationship between "better diagnosis" and ASD. The Centers for Disease Control and Prevention (Centers for Disease Control and Prevention 2007) has suggested that better diagnosis is implicated in the upward trends in ASD prevalence. Operationalizing and measuring the concept of better diagnosis is inherently problematical, but the putative effects of better diagnosis should be associated with diagnostic substitution. Given that the behaviors associated with moderate to severe forms of ASD are visible to even the casual observer, it seems unlikely that school officials failed to notice children with ASD in the past. Rather, as suggested by the substitution hypothesis, children with ASD might have been misdiagnosed (and misclassified) as having MD, LD, SLI, or some other childhood disorder. Presumably, as the professional medical community improved its capacity to diagnose ASD, a shift in the diagnostic classification of ASD students should have occurred as diagnoses became more accurate. To the extent that our findings add to an accumulating body of evidence from state-level studies that do not find

an effect for diagnostic substitution (Blaxill, Baskin & Spitzer 2002; Gurney, et al 2003), we would suggest that claims about better diagnosis should be scrutinized more carefully.

A number of limitations should be noted. As with other studies that examine administrative prevalence, the analysis is limited by the lack of individual level data. A research design employing data from individual records would improve the analysis of diagnostic substitution, but such data are frequently not available for a cross-sectional time-series study. In addition, because the disability counts used for the numerators for prevalence measures are derived from school district reporting, the data likely represent an underestimate of the true disease burden among the school age population in Nevada (Yeargin-Allsop et al 2003). For many reasons, parents of disabled children may remove their children from public schools in order to provide them with private, home-based programs that are not covered by special education services; these children are not included in the school district counts. Finally, although state regulations in Nevada establish a uniform procedure and criteria for diagnosis, the degree of heterogeneity in diagnosis across districts may be greater than in individual-level studies employing strict diagnostic criteria and checks for inter-coder reliability.

Despite these limitations, administrative prevalence data provide researchers and policy makers with the most complete, longitudinal picture of how ASD might have been changing in recent years. Inasmuch as our results do not support claims that trends in ASD administrative prevalence within Nevada are due simply to diagnostic substitution, we remain confident that the administrative data can be combined usefully with single-year estimates from epidemiological surveillance studies to study the geographical and over-time variation in autism prevalence within states.

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Appendix A. Table 2: Diagnostic Substitution and ASD in Nevada School Districts

Covariates	Dependent Variable: MR Prevalence (Reduced Model)	Dependent Variable: MR Prevalence (Full Model)	Dependent Variable: LD Prevalence (Reduced Model)	Dependent Variable: LD Prevalence (Full Model)
Lagged Endogenous Covariate $_{t-1}$	.62*** (.09)	.60*** (.10)	.94*** (.04)	.87*** (.08)
ASD Prevalence	.15 (.14)	.15 (.20)	.44 (.89)	1.18 (1.02)
Board Certified Pediatricians per 1,000 $_{t-1}$		-.70 (.54)		5.78 (4.03)
Real Salaries $_{t-1}$		.00006 (.0001)		.00003 (.0007)
Real Part B funding per student $(\log)_{t-1}$		.005 (.49)		-3.27 (2.53)
District Size $_{t-1}$		.22 (.16)		-4.53* (2.16)
Constant	1.60*** (.48)	-.46 (3.75)	5.64 (4.24)	31.73 (24.19)
N	153	153	153	153
Wald Chi-Square	46.98***	101.91***	428.26***	4803.24***
Adjusted R^2	.47	.46	.87	.87

Note: Entries are unstandardized OLS regression coefficients; panel corrected standard errors reported in parentheses below regression coefficients. ***p<0.001; **p<0.01; p<0.05

	Dependent Variable: SLI Prevalence (Reduced Model)	Dependent Variable: SLI Prevalence (Full Model)
Lagged Endogenous Covariate$_{t-1}$	.67*** (.11)	.67*** (.11)
ASD Prevalence	.11 (.59)	.13 (.73)
Board Certified Pediatricians per 1,000 $_{t-1}$		.82 (1.73)
Real Salaries $_{t-1}$		-.0002 (.0003)
Real Part B funding per student $(\log)_{t-1}$		-.03 (1.54)
District Size $_{t-1}$		-.65 (.59)
Constant	6.56** (2.41)	13.75 (9.47)
N	153	153
Wald Chi-Square	38.69***	49.66***
Adjusted R^2	.44	.43

Appendix B. Further Comment on the Statistical Analysis

1. Autocorrelation and the Lagged Dependent Variable as a Covariate: In the reduced MR, LD and SLI models (Table 2), estimation without a lagged dependent variable as a covariate yielded a pooled autocorrelation parameter, rho, of 0.70, 0.90 and 0.61, respectively, suggesting high degrees of autocorrelation. Estimation of the reduced models in Table 2 with lagged dependent variables reduced the degree of autocorrelation considerably (in MR, LD and SLI reduced models estimated with lagged dependent variables, rho is 0.27, 0.25, and 0.10, respectively). The same pattern is evident when the full models for MR, LD and SLI (Table 2) are estimated without a lagged dependent variable (rho = 0.74, 0.80, and 0.67, respectively); however, when a lagged dependent variable is used, rho falls to 0.29, 0.17, and 0.09, respectively. Likewise, in the ASD model in Table 3, estimation without a lagged dependent variable yields a rho of 0.60; when the ASD model is estimated with a lagged dependent variable, rho falls to 0.04.

The beta for ASD prevalence $_{t-1}$ is 0.70 in the ASD prevalence model in Table 3; standardized regression coefficients for the natural log of Part B funding and the log of enrollment are .18 and .11, while betas for all other variables <0.03. When the lagged dependent variable is removed in the model in Table 3, the Adjusted R^2 drops to 0.05 and the results are compromised by autocorrelation and model mis-specification. The betas for the lagged dependent variables in the MR, LD and SLI trials (Table 2) are similar.

2. Unit Effects. Because this data set pools observations across school districts and years, the results may be sensitive to "unit effects." To diagnose for unit effects, we followed advice by Stimson (Stimson 2005). All models (Tables 2-3) were estimated and summed residual and residual variance ratios for each school district were calculated. Next, we inspected summed residuals and variance ratios approaching the threshold (i.e., variance ratios four times the mean of each dependent variable). In the ASD model, none of the district dummy variables reached the critical threshold and results were consistent with Table 3. In the MR, LD, and SLI models, only a small number of the district dummy variables approached the threshold. Next, we estimated reduced and full models for MR, LD and SLI with dummy variables from those districts that had reached the threshold. None of the coefficients for the district dummy variables achieved statistical significance (p>0.05) in the MR, LD and SLI models while results remained consistent with Table 2. This suggests that the pooled OLS models are not distorted by the failure to model unit effects.

3. Multicollinearity. To diagnose for multicollinearity, we examined tolerance and Variance Inflation Factor (VIF) statistics. No covariate had an individual VIF score approaching the suggested threshold of 10 (maximum VIF scores were 2.55 and 2.52, respectively, for lagged LD prevalence and district size in the LD model; individual VIF scores > 1.8 for all other covariates in the LD, MR, SLI and ASD models). The average VIF score for each model was within tolerance (mean VIF model scores for MR, LD, SLI and ASD were 1.37, 1.78, 1.36, and 1.33, respectively, well below the suggested threshold of five). We also regressed all covariates on all others in each model (a high adjusted R^2 might indicate multicollinearity problems). We then estimated models without the most collinear variables (maximum adjusted R^2 = 0.62 for district size on other covariates in the LD model) and compared the results. The results were completely consistent with those reported in Tables 2-3, indicating that results are not distorted by multicollinearity.

4. Generalized Least Squares as an Alternative Method: The estimation approach we employ is more reliable than Generalized Least Squares (GLS) (and Weighted Least Squares [WLS]). Using Monte Carlo simulations, Beck and Katz (1995) demonstrate that OLS with panel-corrected standard errors (PCSE) produce more statistically conservative estimates compared to GLS. GLS yields "over-confident" standard errors in data sets where the number of years pooled is smaller than the number of units (i.e., school districts) – as in this data set. Therefore, GLS (and WLS, a variant) will tend to inflate the statistical significance levels of covariates. PCSEs perform better than White's robust standard errors to control for heteroscedasticity (Beck & Katz 2004).

5. Pooled OLS with first-differenced dependent variables and covariates. Prevalence can be analyzed with data that are "first-differenced" (year-to-year change). Estimation with the first-

differenced data (with and without lagged endogenous covariates) produced results that were completely consistent in the substitution models (as measured by signs of coefficients and levels of statistical significance). The results of the first-differenced ASD model were consistent with results in Table 3, with the exception of the lagged dependent variable ($p>0.05$; first-differencing reduced autocorrelation).

6. Latent Growth Curve Model Results. Trials with latent growth curve models (LGCM) were also estimated (Shattuck 2006; Palmer, Blanchard, Jean & Mandell 2005). A two-level random coefficient LGCM was estimated with no other covariate than time (origined to the first year of observations); rates of change in MR, LD, SLI and ASD prevalence were examined (Rabe-Hesketh, Pickles & Skrondal 2001). School districts were modeled as the level 2 unit of analysis; the program Generalized Linear Latent and Mixed Models (GLLAMM), STATA v. 10, was used. The rates of change for MR, LD and SLI were -.06, 1.58, and -.13, respectively, but none of the rates of change was statistically significant ($p>0.05$). By contrast, the rate of change for ASD (.11) was statistically significant ($p<0.01$) (The component representing the covariance between intercept and slopes was insignificant, $p>0.05$. The log-likelihood suggested no model misspecification). Given that the growth trajectories of MR, LD and SLI were not declining significantly while ASD was increasing, it is unlikely that diagnostic substitution occurred (see Shattuck 2006).

Next, we employed a two-level random coefficient (LGCM) estimator for MR, LD SLI and ASD prevalence (Tables 2-3). The same covariates as in Tables 2-3 were employed, adjusting for time-invariant and time-varying covariates (on estimation of random coefficient models with covariates fixed at initial levels, covariates that vary over time, and lagged covariates, see Wan, Zhang, & Unruh 2006; Alwin & Wray 2006). In the MR, LD, SLI and ASD trials, we used enrollment data at 1996 levels as the time-invariant covariate, while all other covariates (as in Tables 2-3), lagged by one year, were allowed to vary over time. In all trials, the coefficient for ASD prevalence failed to achieve significance ($p>0.05$), indicating no significant inverse association between ASD and MR, LD, and SLI prevalence; the results for all other covariates (as measured by sign and statistical significance levels) were completely consistent with the results in Table 2. Results of the random coefficient estimation for the ASD model were also completely consistent with results in Table 3.

Given the high degree of autocorrelation in the data, the LGCM results may be biased. Recent studies using Monte Carlo simulations demonstrate that when AR1 processes are present, the parameters of the LGCM are biased and not statistically conservative (Sivo, Fan & Witta 2005). To assess this possibility, all LGC models were re-estimated with a lagged endogenous covariate to control for autocorrelation (on this technique in LGC models, see Beck & Katz 2007; Wan, Zhang, & Unruh 2006; Bollen & Curran 2004). The coefficients for the lagged dependent variables were positive and significant ($p<0.01$), suggesting the effects of autocorrelation, while signs and statistical significance levels of the coefficients for other covariates in each model were consistent with the previous LGCM results. Still, even when an AR1 process is modeled within a LGCM, *residual* autocorrelation may still cause bias in parameter estimates (Sivo, Fan & Witta 2005).