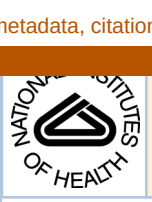



Published in final edited form as:

*Am Sociol Rev*. 2011 April 1; 76(2): 320–346. doi:10.1177/0003122411399389.

# Socioeconomic Status and the Increased Prevalence of Autism in California

**Marissa D. King**[a] and **Peter S. Bearman**[b]

[a]Yale University

[b]Columbia University

## Abstract

The prevalence of autism has increased precipitously—roughly 10-fold in the past 40 years—yet no one knows exactly what caused this dramatic rise. Using a large and representative dataset that spans the California birth cohorts from 1992 through 2000, we examine individual and community resources associated with the likelihood of an autism diagnosis over time. This allows us to identify key social factors that have contributed to increased autism prevalence. While individual-level factors, such as birth weight and parental education, have had a fairly constant effect on likelihood of diagnosis over time, we find that community-level resources drive increased prevalence. This study suggests that neighborhoods dynamically interact with the people living in them in different ways at different times to shape health outcomes. By treating neighborhoods as dynamic, we can better understand the changing socioeconomic gradient of autism and the increase in prevalence.

### Keywords

neighborhoods; health gradient; autism; socioeconomic status; inequality

Autism is a disorder characterized by impairments in communication, social interaction, and repetitive behaviors. Over the past 40 years, the measured prevalence of autism has multiplied roughly 10-fold. While progress has been made in understanding some of the factors associated with increased risk and rising prevalence, no one knows with certainty what causes autism or what caused autism prevalence to rise so precipitously. There is, however, a growing awareness among scholars that focusing solely on individual risk factors such as exposure toxicants, prenatal complications, or parental education is insufficient to explain why autism prevalence rates have increased so stunningly. Social and institutional processes likely play an important role. For example, changes in diagnostic criteria and an influx of resources dedicated to autism diagnosis may be critical to understanding why prevalence rates have risen. Increased awareness and social influence have been implicated in the rise of autism and a variety of comparable disorders, where social processes mimic the effects of contagion (Christakis and Fowler 2007; Liu, King, and Bearman 2010; Pescosolido and Levy 2002). Studies have examined the contribution of changes in diagnostic criteria and diagnostic substitution to rising autism prevalence rates, but the importance of institutional factors, resources for diagnosis, and greater awareness have not been systematically assessed. The sociological literature on health and inequality, however,



**Corresponding Author:** Peter Bearman, Paul F. Lazarsfeld Center for the Social Sciences, CB3355, Columbia University, New York, NY 10027, psb17@columbia.edu.

provides substantial motivation for exploring how individual- and community-level effects operate to shape the likelihood of an autism diagnosis.

This article examines why autism prevalence has increased in the aggregate by exploring why autism prevalence has historically been uneven across different communities. Three processes could explain these phenomena, both the unevenness and the increase. First, the risk associated with a specific factor could increase over time. If this were the case, the size of the population at risk would remain constant but an increase in risk would drive the increase in prevalence. Second, risk factors could contribute to increased prevalence by becoming more prevalent in the population. Third, the characteristics of places could drive the rate of autism diagnoses. Put another way, otherwise similar children living in different areas could be at significantly different risk for autism, not because of their own characteristics, but because of the characteristics of the communities in which they reside. If sociologically salient community characteristics matter for autism diagnosis, we should observe a strong temporal patterning of risk at the community level, such that increases and decreases in risk associated with community characteristics map onto changing prevalence rates. In this article, we use a large geographically and temporally sensitive multilevel data structure to understand dynamics underlying the increased prevalence of autism by examining changes in patterns of risk over nine birth cohorts.

This study makes two contributions to the empirical literature on autism by providing the first multilevel analysis of risk factors for autism and by identifying critical social factors that map onto changes in prevalence rates. In doing so, we present a more dynamic framework for understanding the way in which individual and neighborhood characteristics interact over time to shape health outcomes. The majority of studies that examine how neighborhoods and institutions shape inequalities in health are static (Diez Roux and Mair 2010; Freese and Lutfey forthcoming;Sharkey 2008). Here, we demonstrate that communities and institutions do not passively exert the same influence over time; rather, neighborhoods have different effects on different individuals and different effects at different moments in the autism epidemic. Paying closer attention to temporality when trying to understand health inequalities can provide important insight into factors that may mitigate or exacerbate the relationship between socioeconomic status and health outcomes. By showing that the socioeconomic gradient for autism changed in the course of a decade, this work suggests that socioeconomic health gradients can and do change.

## Socioeconomic Status, Fundamental Cause, and Health Gradients

It is a sociological truism that social status affects one's life chances, including one's health. The more resources people have, the less likely they are to experience disease or early mortality. This generates the negative socioeconomic (SES) health gradients that have been identified for a broad array of conditions ranging from infant mortality to heart disease (Pamuk et al. 1998). The persistence of health gradients across time and in different contexts has given rise to the idea that SES status itself is a fundamental cause of health (Link et al. 1998; Link and Phelan 1995). Within this framework, gradients are thought to be robust because they operate through a multitude of micro-mechanisms (Link and Phelan 2002; Lutfey and Freese 2005), such that if access to one health resource becomes saturated or blocked, numerous other pathways are available through which the SES health gradient can be expressed. Health gradients are believed to arise and persist because actors with more resources can devote more of those resources to their health. When technological and medical advances create opportunities for better health, educated and wealthy individuals are disproportionately able to exploit those opportunities. Therefore, much of fundamental cause theory relies on purposive agents' use of resources (Freese and Lutfey forthcoming).

Progress in understanding how health disparities can change has been stymied by an incomplete conceptualization of the mechanisms by which social and environmental contexts shape health outcomes and how these can change over time. With regard to the fundamental cause literature, the bulk of attention has been devoted to theorizing about how and why health gradients should endure. Relatively little attention has been given to empirically assessing whether, and under what conditions, health gradients can change. However, the advent and diffusion of new technologies has transformed socioeconomic gradients for cholesterol (Chang and Lauderdale 2009) and cancer screening (Link et al. 1998). Autism is a particularly important case in this regard because there has not been a technological innovation in the autism realm akin to cholesterol lowering statins or cancer identifying mammograms.[1] Autism diagnoses have always been based solely on presentation. Because technological change has had no impact, if we identify changes in the autism gradient, broader social processes are likely implicated.

## Socioeconomic Status, Neighborhoods, and Health Gradients

It is well established that where people live has an important effect on their health. Neighborhood context, independent of the individuals composing the neighborhood, is associated with all-cause mortality and a host of negative health outcomes (Kawachi and Berkman 2003).[2] Neighborhoods can influence health, independent of the individuals who compose them, through the physical environment, the social environment, and by structuring access to medical care and services (Adler and Newman 2002; Roberts 1997). The physical environment can affect health directly and indirectly. Directly, it can expose residents to toxicants. Indirectly, it can structure opportunities to engage in healthy behaviors, for instance by providing access to parks and playgrounds (for a review, see Kaczynski and Henderson 2008). Studies exploring how the social environment may affect health outcomes have examined factors such as a lack of social cohesion or disorganization, which tend to increase stress and isolation, thereby adversely impacting inhabitants' health and well-being (Sampson, Morenoff, and Gannon-Rowley 2002). Aside from stress-mediated mechanisms, networks can influence health outcomes by shaping how individuals identify and treat (or do not treat) health problems (Pescosolido 1992). By structuring opportunities for social interaction through architecture or the quality of their institutions, neighborhoods can affect health outcomes through network-based mechanisms. Finally, neighborhoods provide differential access to health-related services, such as sanitation, hospitals, and primary-care providers (Matteson, Burr, and Marshall 1998).

To date, research examining the role of neighborhoods and social context in shaping health outcomes has been largely static and devoid of adequate mechanisms. Yet, neighborhoods and institutions can interact with, exacerbate, and mitigate the relationship between individual SES and health over time (Auchincloss and Diez Roux 2009; Freese and Lutfey forthcoming). Inert conceptualizations of the role of neighborhoods are not confined to studies of health outcomes, but pose a problem for understanding stratification processes more generally (Sharkey 2008). While a few longitudinal studies examine how individual health outcomes are shaped by neighborhoods over the life course (Carson et al. 2007; Pollitt et al. 2008), little research analyzes how neighborhoods themselves may have different effects over time. This study contributes to efforts to address this shortcoming in the literature by allowing the effects of neighborhoods to vary across cohorts. By dynamically conceptualizing how neighborhoods shape health outcomes and tracking them

[1]However, see Eyal and colleagues (2010) for an important historical analysis of the role of therapies in the construction of the autism spectrum.

[2]These outcomes include low birth weight, systolic blood pressure and serum cholesterol, depression, gonorrhea, and violence (see Kawachi and Berkman [2003] for a summary of previous studies).

over time and space, we gain insight into the changing socioeconomic gradient for autism and the rising autism prevalence rates.

### Roadblocks and a Roadmap

It follows that combining insights from perspectives emphasizing the role of individual resources in inducing health inequalities with a dynamic conceptualization of the way that neighborhoods influence health outcomes increases our ability to understand how gradients arise, persist, and change. We examine how the socioeconomic gradient for autism changes over time. We then turn to the mechanisms that might account for the gradient. Rather than focusing on technology, we examine differences in the expression of ES across the autism spectrum.[3] If ascertainment accounts for the autism gradient, a positive SES gradient should be strongest for less severe cases. This follows from the idea that less severe cases are harder to identify and diagnose, because the symptoms on which a diagnosis is based are less prominent. Because a disproportionate share of the increased caseload in California arises from the high-functioning tail of the distribution (Liu, King, and Bearman 2010), a stronger SES effect for less severe cases, which have disproportionately contributed to the increase, would allow us to link changing patterns of identification and treatment to rising prevalence rates. We discover that over the course of a decade, a positive SES gradient for autism appears and then begins to reverse, a transformation driven by changing patterns of identification and treatment. Before we take these steps, however, we first consider what we know—and do not know—about the causes of rising autism prevalence.

## AN INTRODUCTION TO THE DETERMINANTS OF AUTISM

Despite a growing body of research, the etiology of autism remains unknown and highly uncertain. At the biological level, studies examining parental, prenatal, perinatal, and obstetric risk factors have identified a host of conditions associated with an increased risk of autism. Prenatal and perinatal factors associated with an increased risk of autism include parental age (King et al. 2009), low birth weight (Eaton et al. 2001), low Apgar score (Eaton et al. 2001), fetal distress (Glasson et al. 2004), multiple births (Croen, Grether, and Selvin 2002), small for gestational age (Hultman and Sparen 2004; Larsson et al. 2005), and birth order (Durkin et al. 2008). However, there is considerable inconsistency across studies, casting doubt on which of these factors are truly associated with increased autism risk (for a review, see Kolevzon, Gross, and Reichenberg 2007). Autism surely has a genetic component, although how important it is remains unclear. On one hand, the largest twin study to date estimates a concordance rate of 47.5 (95 percent CI: 41.6–53.4), so there is indirect evidence of substantial heritability (Liu, Zerubavel, and Bearman 2010). On the other hand, molecular genetic research has not yet identified genetic causes for the vast majority (85 to 98 percent) of all autism cases (Abrahams and Geschwin 2008).

Environmental toxicants have also been highlighted as a potential cause. Currently, five chemicals are known to cause neuro-developmental disorders, including autism.

[3]The diagnostic criteria for autistic disorder outlined in the Diagnostic and Statistical Manual of Mental Disorder (DSM) delineates 12 criteria in three domains: (1) social interaction (e.g., lack of social or emotional empathy), (2) communication (e.g., inability to initiate or sustain conversation), and (3) restricted and stereotyped patterns of behavior (e.g., persistent preoccupation with parts of objects). For an autism diagnosis, a minimum of two criteria within the social interaction domain and one each from communication and stereotyped behaviors must be met. Additionally, symptoms must be present before a child is 3 years old. Autism, however, is part of a larger group of autism spectrum disorders that includes Asperger's Syndrome (AS) and Pervasive Developmental Disorder-Not Otherwise Specified (PDD-NOS). AS and PDD-NOS require fewer symptoms to be present for a diagnosis. Social deficits are common across the spectrum, although there are large differences in symptomatic severity and intensity. Persons with the most severe forms of autism may lack speech and often have comorbid intellectual disabilities. People with Asperger's syndrome generally do not have speech or communication delays. PDD-NOS is a subthreshold diagnostic category for children who do not meet the diagnostic criteria for autism or Asperger's Syndrome. (See Johnson and colleagues [2007] for a more lengthy description of parts of the spectrum.)

Additionally, another 200 chemicals are known to have neuro-toxic effects in adults (Grandjean and Landrigan 2006). Thus, there is strong reason to believe that chemical exposure might be implicated in the autism epidemic. However, empirical studies investigating the role of environmental toxicants have been limited, and the two ecological studies that do exist (Palmer et al. 2005; Windham et al. 2006) cannot causally disentangle aggregated and individual exposures or directly assess exposure.[4]

Turning to potential social sources of the epidemic, autism is difficult to diagnose because there are no known biological markers and the symptoms are hard to assess, especially among persons with cognitive impairments. Diagnoses are based solely on clinical presentation and parental interviews. This fact has led some scholars to suggest that diagnostic substitution plays a significant role in the increasing prevalence of autism (Shattuck 2006; Wing and Potter 2002). Evidence in support of the diagnostic substitution hypothesis arises from a few recent studies that show increased autism rates accompanied by concurrent declines in the prevalence of mental retardation and other developmental disabilities (Bishop et al. 2008; Coo et al.2008; Shattuck 2006).

Independent of diagnostic substitution, some scholars argue that changes in diagnostic practices lie behind the increased prevalence of autism. Of course, these scholars note that changing diagnostic practices and procedures accompany and are implicated in a process of diagnostic substitution. Since Kanner first described autism in 1943, diagnostic standards, practices, and procedures have changed considerably (Fombonne 2001). Research shows that changes in diagnostic criteria prompt changes in diagnostic status, specifically from a sole diagnosis of MR to a diagnosis of autism-MR (King and Bearman 2009). Drawing on a dataset of persons diagnosed with autism in California, King and Bearman estimate that diagnostic accretion and substitution may account for close to one-quarter of the increase in the California caseload. Hence, there is some evidence that changes in diagnostic standards and diagnostic substitution are driving some component of the observed increase in autism prevalence.

Scholars have also investigated parental social characteristics, such as socioeconomic status, education, and occupation for possible correlations with autism. This literature is largely inconclusive (Croen, Grether, and Selvin 2002; Larsson et al. 2005; Palmer et al. 2005). Early studies identified consistent associations between parental education or socioeconomic status and autism (Finegan and Quarrington 1979), while more current studies tend to find little to no association between parental education, income, or wealth and autism (Larsson et al. 2005). However, a recent study by Durkin and colleagues (2010) using area-based measures of socioeconomic status found that prevalence of autism increased with SES in a dose-response manner.

Resources may matter because obtaining an autism diagnosis can be extremely difficult. In obtaining a diagnosis and services for their children, parents often confront a dizzying institutional maze and spend considerable resources navigating through it. Some communities do not have qualified diagnosticians. Accordingly, community resources—including screening resources, service availability, educational spending levels, the number of school-based health centers, and the number of pediatricians in a community—have been tied to autism (Barbaresi et al. 2005; Mandell and Palmer 2005; Palmer et al. 2005). As with studies of potential environmental toxicity, the absence of a multilevel design makes it impossible to disentangle whether community resources have an independent effect or are acting as proxies for aggregated individual-level effects.

[4]The idea that vaccines cause autism has been popular but is not substantiated in the scientific literature.

The growth in attention and resources devoted to autism has been almost as astounding as the rising prevalence rates. Autism was the first specific disorder to have a Senate hearing focused exclusively on it (Insel 2007). Between 1997 and 2006, funding for autism research by the National Institutes of Health increased five-fold from $22 million to $108 million (Singh et al. 2009). The Centers for Disease Control and Prevention's funding of autism activities increased from $2.1 million in 2000 to about $16.7 million in 2005 (Government Accountability Office 2006). And an additional $1 billion was committed to autism with the signing of the Combating Autism Act in 2006. The increase in resources devoted to autism research and treatment has far out-paced increasing prevalence rates. Between 1994 and 2004 in California, expenditures by the Department of Developmental Services (DDS), the agency responsible for serving persons with developmental disabilities in California, increased 160 percent. During the same period, the agency's caseload increased by 61 percent (DDS 2007). In California, as in the rest of the country, there has been a dramatic increase in resources available to screen for and to treat autism.

What effect, if any, this influx of resources has had on prevalence rates remains unknown. More basic questions about the importance of parental and community resources for autism diagnoses remain unanswered. The literature continues to debate whether autism has a socioeconomic gradient. Accordingly, whether infusions of resources into communities to diagnose and treat autism have coincided with changes in the autism gradient and how, if at all, this may relate to prevalence rates present an important unsolved puzzle.

## DATA AND METHODS

To make progress in answering these questions, we need to work with population-based data that allow us to simultaneously model individual and social contextual factors over time. This study examines birth and diagnostic records for all children born in California between 1992 and 2000. We analyzed 4,906,926 birth records from this period. Of these, we could match 18,731 to children with a diagnosis of autism. To identify autism cases, we combined birth records obtained from the California Birth Master Files and the California Department of Developmental Services. The DDS coordinates diagnoses, services, and support for persons with developmental disabilities living in California. The agency provides services to patients with autistic disorder (ICD-9-CM code 299.0).

Our goal is to estimate a series of multi-level models examining the association between individual- and community-level SES measures and the probability of an autism diagnosis across consecutive birth cohorts. The analysis begins with the birth cohort of 1992, the first year the DDS began maintaining electronic records. To ensure that all children had ample time for case ascertainment, the analyses end with the birth cohort of 2000. All children were followed from the time of birth until June 2006. We allowed differential ascertainment times because the age of diagnosis for autism has been consistently falling, from a mean of 5.9 years (± 2.9) among the 1992 birth cohort to 3.8 years (± .9) for the birth cohort of 2000.[5]

### Individual-Level Variables

To obtain demographic, prenatal, and perinatal information for persons with and without autism, we electronically linked records from the DDS and Birth Master Files using probabilistic and deterministic matching algorithms developed by Campbell (2004).

[5]The differential ascertainment times for each cohort have no effect on our results or our estimates of prevalence. As Palmer and colleagues (2005) and Hertz-Picciotto and Delwiche (2009) demonstrate in detail, by the 2000 cohort the vast majority of children are diagnosed by age 6.5 years. In addition, a series of robustness checks (available from the authors upon request) further demonstrate that ascertainment bias arising from a declining follow-up period across cohorts do not affect our results.

Matches were made based on first, middle, and last name; date of birth; race; zip code at birth; and sex. We manually reviewed uncertain matches. On average, 81 percent of DDS files were linked to birth records. The linkage rate increased over time from 79 percent (1992) to 86 percent (2000).[6]

We extracted data on parental education and whether a child's birth was paid for with Medi-Cal (California's Medicaid program) from the matched files. These variables serve as proxies for socioeconomic status. Roughly 40 percent of births in California are paid for with Medi-Cal, so the program serves a considerable portion of the population. Data on paternal education was missing for approximately 8 percent of birth records. Maternal education was rarely missing. To avoid potential biases arising from missing data, we used the maximum education level of either parent as the child's parental education. To assess whether having missing data on the father has any association with risk of autism, we included a dummy variable for missing paternal information.

From the birth certificates, we also obtained the following variables: birth weight, parental age, duration of gestation, whether the child was admitted to the neonatal intensive care unit (NICU), and parity. We calculated parental age similarly to parental education to avoid problems arising from missing data. We examined birth weight categorically, with birth weight greater than or equal to 2,500 grams considered normal. Gestational age of fewer than 35 weeks was considered pre-term. If a new-born had a normal birth weight and gestational age but was admitted to the NICU, the baby likely had poor presentation at birth. When these conditions were met, we recorded "low Apgar proxy" as 1, otherwise zero. We used parity to generate a dummy variable for first live birth.

## Community-Level Data

The birth data files contain the zip code for residence at birth, which makes it possible to nest individuals within the communities in which they were born. California had 1,620 zip codes in 1992, this rose to 1,677 numerical zip codes in 2000. We eliminated zip codes with fewer than 10 births per year because neighborhood estimates based on small numbers of residents may be unreliable.[7] To examine the importance of community resources for autism diagnoses, we constructed five community-level variables using zip codes as the unit of analysis. (See Table A1 in the Appendix for a more detailed list.) From the 1990 and 2000 Censuses, we extracted the median property value of the zip code (logged) and the educational attainment for persons over 25 years. We categorized educational attainment as the percent of persons in the zip code with a college degree or higher. We used linear interpolation to obtain data for intermediate years. Average property value and educational attainment correlate at $r < .35$; it is thus unlikely that problems arise from multicollinearity.

To test for other community factors that might influence the likelihood of an autism diagnosis, we aggregated data on the number of autism advocacy organizations, the number of pediatricians, and the number of child psychiatrists operating in a zip code at the time of the cohort's birth. To obtain data on the number of autism advocacy organizations, we searched Guidestar's tax records for all organizations that identified "autism" as their primary code under the National Taxonomy of Exempt Entities. Research organizations have a different classificatory status. To identify less formal organizations, which may not request 501(c)3 status, we conducted exhaustive Internet searches. For organizations with local chapters, we recorded each chapter as its own entity. For each organization, we

[6]Given the fairly exhaustive linking criteria and the typical patterns of immigration into California, DDS files that could not be linked likely belong to children who were born outside of California and later moved to the state.

[7]We ran robustness checks to see if not excluding zip codes or excluding zip codes with greater numbers of births influences results. The results (available from the authors upon request) show that inclusions and exclusions do not significantly change our findings.

recorded a founding date, a dissolution date if applicable, and an address. We obtained data on the number of pediatricians and child psychologists in a zip code from Medical Marketing Services, who license data from the American Medical Association. We measured all variables at the time of a cohort's birth to mitigate problems arising from endogeneity.

### Severity

To compare how risk is shaped by severity at first diagnosis, we constructed severity measures using social and communication scores that are contained within the DDS's Client Development and Evaluation Report. We additively converted nine items into an index, with each item equally weighted: peer interaction, non-peer interaction, friendship formation and maintenance, participation in social activities, unacceptable social behaviors, word usage, receptive language, and expressive language.[8] Cronbach's alpha for these items exceeds .75. Severe cases are those above the mean evaluation score, less severe cases are those below the mean. The distribution of severity scores is fairly symmetrical. Accordingly, the number of severe and less severe cases is roughly equal. The severity score is the score at time of entry into the DDS. Because a person entering the DDS at age 3 years will have a lower score than someone entering at age 5 years—all else being equal, 5-year-olds are more developed than 3-years-olds—we mean-centered severity scores by birth year and age at entry.

### Multilevel Analysis

To simultaneously consider the association between individual- and community-level factors over time, we ran a multilevel model for each birth cohort in HLM 6 (Raudenbush, Bryk, and Congdon 2004). The dependent variable in the logistic regression is whether a child received an autism diagnosis. Multilevel models take into account the hierarchical nature of the social world. Here we consider individuals nested within neighborhoods, captured by zip code. All of the individual-level variables are grand mean centered, except parental education, which is group mean centered because there is systematic variation in the mean parental educational level across zip codes. All level-two, or community, variables are grand mean centered. We include two cross-level interactions to understand how the risk associated with an individual-level variable varies depending on the nature of the characteristics of the community in which the individual lives. We examine how the association between parental education and autism varies depending on the percent of college graduates in the community, as well as how the effect of being a Medi-Cal beneficiary on autism varies depending on the median property value in a community. In summary, the modeling strategy allows us to examine how individual- and community-level risk factors for autism vary across time and with each other. The multilevel models use an unstructured covariance matrix. Because an unstructured covariance matrix does not impose any constraints and we have sufficient statistical power, this is the most conservative choice. We calculated predicted probabilities of diagnosis from the models in HLM. After reporting findings from analyses by birth cohort, with an autism diagnosis as the dependent variable, we then focus on level of severity.

## RESULTS

The prevalence of autism among the 1992 through 2000 California birth cohorts increased considerably, from 29 per 10,000 in 1992 to 49 per 10,000 in 2000. Figure 1 shows this trend: rapid growth and then saturation. Note that the *x*-axis reports birth cohorts. Children

[8]Because each item has a different range, we used (Σ((item score/maximum possible score)/(total number of items)))*100 to calculate the severity score.

born in 1992 are largely diagnosed by 1998, whereas children born in 2000 are largely diagnosed by 2005.

Just as there is significant variation in the probability of diagnosis over time, we also observe significant variability in the probability of diagnosis between zip codes. The estimated variance of $\beta_{0k}$, $\tau_{t00}$, is statistically significant at the $p < .01$ level in all of the unconditional models, except for the model for the 1995 birth cohort ($p = .09$). (See Part A in the online supplement for a complete table [http://asr.sagepub.com/supplemental].) The observed variability in prevalence rates is consistent with the identification of statistically significant autism clusters in California, in which the risk for autism is four times greater than the risk in other parts of the state (Mazumdar et al. 2010). The observed variance remains statistically significant in the individual and neighborhood models. Only in the complete multilevel model is the variance reduced to statistical insignificance, indicating that our model accounts for the observed variability in diagnoses between zip codes.

As we turn to examine the risk factors associated with autism, recall that a factor could account for the increased prevalence of autism by meeting one of two criteria. One possibility is that a risk factor may become more prevalent in the population. Alternatively, the population at risk could remain fairly constant but an increase in the risk of a factor could drive the increase in prevalence. If this were true, we would expect to see the risk increase or decrease in concert with prevalence rates.

## Individual-Level Factors

We first turn to the individual-level factors examined in our model. Table 1 summarizes the results for each of the multilevel models by birth cohort.[9] Odds ratios and 95 percent confidence intervals for each variable appear under the year for each birth cohort. As Table 1 shows, Medi-Cal, birth order, normal birth weight, being male, paternal age, and paternal education are consistently associated with autism risk across all birth cohorts.

Being a first-born child, a boy, or having older or more educated parents increased the risk for autism. Increasing parental age may increase risk of autism as a result of the increasing likelihood of de novo mutations that occur with age (Liu, Zerubavel, and Bearman 2010). Alternatively, parental age could be an indirect proxy for elements of socioeconomic status not captured by education and property values, as parents of higher SES tend to delay childbirth (Baldwin and Nord 1984). Conversely, being on Medi-Cal, our measure of income status, is associated with a decreased risk of an autism diagnosis. Being male is associated with a four-fold greater risk of autism diagnosis. The gender gap in autism prevalence (mirrored in ADHD) remains unexplained (Baron-Cohen, Knickmeyer, and Belmonte 2005).[10] Birth weight in excess of 2,500 grams reduced the likelihood of an autism diagnosis in the majority of cohorts, suggesting that low birth weight increases risk for autism. Premature birth, in which the gestational age was fewer than 35 weeks, is associated with increased risk of autism in three out of nine years, reaching statistical significance only in later years. This could be due to the increased survival rate among premature births. The consistent birth-order effect indicates that first-born children are at greater risk for autism. Possible mechanisms to account for this finding involve the concentration of fat-soluble chemicals in maternal milk and tissue, slower development of

[9]We will return to the results presented in this table throughout the following sections. The results will also be depicted graphically section by section to aid with interpretation.

[10]There are many explanations for disproportionate male incidence of autism; the most well known is Baron-Cohen and colleagues' (2005) argument that autism is an extreme form of the male brain that emphasizes systematizing functions over empathetic functions. This explanation, and many others, is largely a re-description of maleness and autism.

immune systems due to fewer exposures to infections, and the statistical effect of parents who stop having children after having a child with autism (Durkin et al. 2008).

Turning to the temporal variability and periodicity of individual risk factors, we observe that the majority of individual-level factors have a consistent effect size. As Figure 2 demonstrates, the odds ratios for parental age are largely invariant across birth cohorts, ranging from 1.03 (95 percent CI: 1.03, 1.04) to 1.04 (95 percent CI: 1.03, 1.05). Parental education is similarly stable, varying from a high of 1.15 (95 percent CI: 1.13, 1.16) to a low of 1.08 (1.07, 1.10). Recall that parental education is group mean centered; therefore, odds ratios represent the effect of parental education relative to the average level of parental education within a child's neighborhood. Having more education relative to one's neighbors is associated with an increased risk of autism diagnosis.

Only the effects of Medi-Cal reveal meaningful periodicity. Among the birth cohorts of 1993 until 1995, Medi-Cal receipt reduced the odds of an autism diagnosis by roughly 20 to 40 percent. Accordingly, autism had a positive socioeconomic gradient. Individuals with greater resources were more likely to receive an autism diagnosis. Early in the epidemic, individuals relying on Medi-Cal for health care likely did not have the resources necessary to acquire an autism diagnosis. Note that the strength of the positive SES gradient waned among later cohorts as diagnoses became increasingly prevalent and knowledge about the disorder diffused widely. By the 2000 birth cohort, the odds ratio for Medi-Cal was .97 (.90, 1.03), suggesting that the positive socioeconomic gradient was disappearing. With the exception of Medi-Cal, the individual-level variables in our model do not exhibit periodicity in risk consistent with changing prevalence rates.

Similarly, there is little variation in the prevalence of any of the risk factors in the population. While average parental age and the proportion of births that were premature or had poor presentation all increased, these increases are not large enough to make even a minimal contribution to increases in autism prevalence. (See Table A1 in the Appendix for a summary of demographic changes across cohorts.) Accordingly, it is unlikely that the increasing prevalence of an individual-level risk factor in the population accounts for the observed increase in autism prevalence.

## Neighborhood Wealth and Autism

The effect of community wealth on the chance of acquiring an autism diagnosis is of central interest. Figure 3 plots the odds ratios for logged median property values over time and demonstrates that the effect of community wealth varies with changes in prevalence rates. The grey line tracks the percent change in prevalence over the previous period (or the rate of increasing prevalence). The effect size of community property values appears in black. The correlation between the odds ratio for property value and changes in prevalence across the cohorts is .71. These results indicate that the economic composition of a community matters most when prevalence rates are rising. As diagnoses became more common and the rate of increase slowed, the importance of community wealth declined.

Turning to how prevalence rates vary by levels of individual and community economic resources, we now consider cross-level interactions. Figures 4a and 4b plot the predicted probability of an autism diagnosis for children who do and do not receive Medi-Cal in communities with very high (top 90 percent) and very low (bottom 90 percent) median property values. The *y*-axis can be thought of as a prevalence rate. Figure 4a reports the probability of an autism diagnosis for children whose birth was paid for by Medi-Cal and who reside in a neighborhood in the bottom decile of the neighborhood property distribution (dashed line) compared with the "same" child residing in a neighborhood in the top decile (solid line) of the property value distribution. A child born on Medi-Cal and residing in the

wealthier neighborhood was, on average, close to 250 percent more likely than his counterpart living in a poorer neighborhood to be diagnosed with autism. This can be seen by comparing the difference in prevalence rates between the solid and dotted lines in Figure 4a. Moreover, the importance of neighborhood context increased over time, as seen by diverging prevalence rates in wealthier and poorer neighborhoods.

By contrast, for a child whose birth was not paid for by Medi-Cal (see Figure 4b), moving from the bottom decile (dashed line) to the top decile (solid line) of the property value distribution increased the probability of diagnosis by 190 percent, on average, over the same period. Although community resources matter, they matter less than if the child received Medi-Cal.

Finally, the predicted probabilities reveal an important temporal pattern that yields insight into changes in the socioeconomic gradient for autism. Recall that autism initially had a strong positive socioeconomic gradient. Children born to wealthier and more educated parents living in wealthy neighborhoods had the highest probability of obtaining an autism diagnosis. However, the diagnostic rate among this group was stagnant after 1994. Diagnoses among wealthier individuals in wealthier neighborhoods appear to have hit a ceiling around 40 per 10,000. By contrast, prevalence rates among children whose birth was paid for by Medi-Cal living in these same neighborhoods experienced consistent increases from 20 per 10,000 in 1992 to 46 per 10,000. Similarly, the probability of diagnosis for children living in the poorest neighborhoods increased steadily across cohorts, although at a slower rate. In summary, neighborhood resources matter tremendously for autism prevalence rates. However, they matter much more for children born to parents with fewer economic resources. The trends presented here add further support to the notion that the socioeconomic gradient for autism has begun to reverse. In wealthy communities, the socioeconomic gradient for autism has flattened; a different picture arises from poorer communities.

### Neighborhood Educational Attainment

Educational attainment in a neighborhood had a small effect on the likelihood of an autism diagnosis. In contrast to the results for economic resources, educational resources primarily operate at the individual level, as reported in Figures 5a and 5b. Comparing Figure 5a, which shows the probability of diagnosis for a child whose parents are at the 25th percentile of education relative to their neighbors, with Figure 5b, which plots the probability of diagnosis for a child whose parents are in the top 75th percentile of education, we see that diagnoses are consistently more likely for children born to relatively more educated parents. However, the overall effect of education at the parental or community level appears to be relatively weak.

### Health Care Resources

Of course, property values are likely acting as proxies for more meaningful mechanisms. One way to address more proximate mechanisms is to try to identify variables that property value may capture. We consider three variables—the number of pediatricians, child psychiatrists, and autism advocacy organizations in each community—to try to understand whether access to health care or exposure to organizations committed to increasing awareness about autism might afford a more detailed understanding of variability in autism prevalence rates across time and communities. None of these variables are substantively important for the likelihood of an autism diagnosis. As Table 2 shows, while the number of pediatricians and child psychologists are statistically significant in many years, their effect size is small. Autism organizations are occasionally negatively associated with the likelihood of diagnosis. Supplementary analyses, which included breast cancer advocacy

organizations, suggest that this relationship is likely due to ecological factors, rather than the presence of autism organizations themselves. Furthermore, inclusion of all the supplementary variables has very little effect on other measures of socioeconomic status included in the model. This suggests that whatever socioeconomic status is capturing, it is not the availability of health care providers or advocacy organizations.

## Severity

While we gained little by trying to measure health care resources in a community, insight into possible mechanisms that could account for the changing autism gradient does arise by looking at whether there are differences in socioeconomic status by severity. More severe cases are easier to identify, because the symptoms upon which a diagnosis are made are less ambiguous. It follows that if differential ascertainment and diagnostic capacity underlie the SES gradient, we should be able to observe this by comparing SES gradients for more and less severe cases. When autism cases are split by severity, a striking pattern is revealed. Less severe cases are disproportionately found in wealthier and more educated neighborhoods. Figure 6 shows the ratio of less severe to more severe cases with respect to neighborhood wealth and education. Recall that we split severity at the mean, so there are relatively equal numbers of more and less severe cases in the population. If severity were independent of neighborhood context, we would expect the ratio to be roughly one to one. This is not the case. The most diagnostically ambiguous cases, those that are the hardest to identify and diagnose because the symptoms that provide a basis for diagnosis are less pronounced, are disproportionately found in educated and wealthy neighborhoods. Thus, the observed SES gradient for autism is at least partially driven by identification and ascertainment.

Odds ratios obtained from multilevel models examining the probability of a more or less severe diagnosis add further support to the assertion that differential ascertainment may be driving the socioeconomic gradients, and hence increased prevalence. Figure 7 presents these results graphically. (See Part B of the online supplement for complete tabular results.)

Odds ratios for more severe cases are shown in black, those for less severe cases are shown in grey. As Panel A shows, the effect of Medi-Cal is always stronger for less severe cases; that is, the grey line (odds ratio for less severe cases) always appears below the black line (odds ratio for more severe cases). By 1997, odds ratios for Medi-Cal for more severe cases generally exceed one, suggesting that the socioeconomic gradient for severe cases may be reversing and becoming negative. Parental education, shown in Panel B, is positively associated with the likelihood of both a more severe and a less severe diagnosis. Odds ratios are higher, however, for less severe cases, relative to more severe cases, consistent with our observations for income.

Turning to community-level factors, we find a positive, although generally insignificant, effect of community educational attainment for less severe cases. Neighborhood property value matters for case identification for more and less severe cases. This is consistent with the overall importance of property value identified with all autism cases. Of note, however, is the declining significance of property value for more severe cases after the 1994 cohort. This pattern is almost identical to the one observed when we consider all autism cases together. In summary, if less severe cases are more difficult to identify, then differential ascertainment capacity at the community level may be one mechanism through which SES effects operate. The stronger effects of socioeconomic status on less severe cases provide support for this idea.[11] Recall that the majority of the increased caseload in California arose

[11]While environmental toxicants could be responsible, the toxicant would have to be differentially associated with less severe symptoms and disproportionately present in highly educated, wealthy neighborhoods. This seems unlikely.

disproportionately from an increased number of less severe cases. Patterns of ascertainment and identification underlie not only the autism gradient but also the increased prevalence of autism in California.

Results for neighborhood education and community resources provide some clues about the type of information that may be required for identification and ascertainment. These results suggest that specialized, granular knowledge is likely required for diagnosis. When specialized knowledge is costly to obtain or difficult to access, the financial resources available in a community will be more important than a community's general education level. This appears to be the case with autism, because access to a highly trained specialist—not just any specialist—is often critical to getting a diagnosis. Similarly, because granular information is more easily transmitted through embedded social relations (Uzzi 1996), the diffusion of information about autism through communities likely occurs around foci of repeated parental interactions, rather than through more traditional institutions, as shown by Liu, King, and Bearman (2010).

## CONCLUSIONS

### Limitations and Robustness Checks

This study has several limitations. First, we interpolated the property value data used in the analysis from the 1990 and 2000 Censuses because data were not available for all zip codes for intermediate years. To test the accuracy of the property value interpolation, we used the average sales price per zip code per year (RAND 2010). Sales data and interpolated property value are highly correlated. The multilevel analysis with the alternate property value specification yields similar results, which increases our confidence in the interpolation. (See Part C of the online supplement for results of this analysis.)

Two selection issues arose in the analyses. Because our data focus solely on children born in California, one might wonder whether these children differ from children who were born elsewhere and later moved to the state. While it is impossible to test this with the available data for the general population, for the population with autism we found no statistically significant differences for property value and race, the only variables for which we have data on children moving into the state. Similarly, we wanted to ensure that persons covered by Medi-Cal who reside in wealthier areas are not different from persons on Medi-Cal residing in poorer areas. To test this, we compared available demographic characteristics for persons on Medi-Cal living in wealthy communities (top decile of property value) to the rest of the Medi-Cal population. We examined variables in our analysis and additional variables, including parental age, education, race, immigration status, and the number of prenatal visits. The differences observed are substantively unimportant. The maximum parental age difference between the two groups is two years and a single year of education separates the two groups. Race, immigration status, and the number of prenatal visits also show minimal differences between the two residential groups. While selection is possible based on unobserved characteristics, the lack of obvious selection based on standard sociological variables increases our confidence that our findings would not be considerably altered by possible omitted individual-level variables.

Another potential concern in our analyses is that omitted variable bias or inadequate controls in our individual-level analysis could bias our estimates of neighborhood effects. We took a tripartite approach to assessing the possible effects of incomplete controls for individual-level socioeconomic status. Our first robustness check, which we believe is a strong test, added the median income in the block group in which a child resided as an additional individual-level control. This was only possible for cohorts born after 1996, because the point level address data needed to assign individuals to block groups is not available for

earlier cohorts. For available cohorts, we grand mean centered the block group income variable. Inclusion of median block group income as an individual-level covariate does not alter our results (see Part D in the online supplement). Our second set of analyses incorporates all proxies for socioeconomic status available from the birth certificates that we did not previously use, such as race and whether a child's mother was born outside of the United States. Addition of these covariates does not alter our results and the variables are generally insignificant (see Part E in the online supplement). Finally, we conducted a move analysis to see whether children born in the same neighborhood but who later moved to neighborhoods with different median property values have different probabilities of diagnosis (Mazumdar et al. 2010). By using propensity score matching to match on individual-level socioeconomic status and neighborhood of origin, we observe that upwardly mobile children were at higher risk of diagnosis. If the analysis is stratified by Medi-Cal receipt, the mobility effects are stronger for children whose birth was paid for with Medi-Cal. These results are consistent with the results reported earlier in our text (Mazumdar et al. 2010). Taken together, these supplementary analyses indicate that Medi-Cal enrollment, parental education, and having a missing father adequately control for individual-level socioeconomic status and that our results are robust.

Several additional limitations remain. First, we calculated all community SES measures using zip code of residence at birth. While we know where a child diagnosed with autism lived at the time of diagnosis, we do not know residence at all times for all children born in California. This makes an analysis comparable to the one based on residence at diagnosis impossible. However, the birth clusters identified by Mazumdar and colleagues (2010) overlap with diagnostic clusters, suggesting that geographic mobility does not present a large problem for our analysis.

A second limitation is that our study is restricted to the state of California. California has a well-established agency through which it provides diagnoses, services, and support to persons with autism. As a result, one may expect higher prevalence rates and less inequality than in other states where diagnostic capacity and autism awareness may be lower. Against this background, and given the striking inequality observed here—especially during the height of the epidemic—California may provide a conservative case. Future research is needed to determine whether a similar pattern of a waxing and waning SES gradient can be identified elsewhere. Just as community characteristics within California are an important part of the prevalence story, state-to-state variation has also been shown to be significant (Shattuck 2006); whether these variations are due to factors examined in this study is an important area for future research. Finally, while we have assembled evidence that at least part of the observed SES gradient for autism and increased prevalence is due to changing patterns of identification and treatment, we cannot rule out the possibility that toxicants or other factors may also be implicated. They may be.

## Comment

We find that the socioeconomic gradient for autism has begun to reverse. It is reasonable to ask whether this gradient—or the strong positive relationship between community resources and autism—fueled the epidemic. As prevalence rates began to increase at a slower rate and diagnoses became less rare among groups with high socioeconomic status, the community-level SES gradient began to fade. Yet, the community gradient did not weaken for all individuals. It persisted among individuals with fewer economic resources. Over time, however, a lack of resources presented less of an obstacle to diagnosis. Individuals who would have been less likely in previous cohorts to be diagnosed with autism due to their parents' socioeconomic status or the communities they lived in became more likely to be diagnosed.

This finding is consistent with the idea of maximally maintained inequality, brought to bear in the literature on socioeconomic health gradients by Lutfey and Freese (2005) and first introduced by Raftery and Hout (1993) to explain class differentials in educational attainment. Here, class differences in enrollment become less salient as enrollment expands to the point at which a system is able to be less selective. Applied to the socioeconomic gradient for autism, we find that it began to wane as knowledge about the disorder and the capacity for ascertainment saturated the entire population and enrollment expanded to the point where the health care system became less selective.

This article examines how, when, and why resources matter for an autism diagnosis. We show that individual and community resources mattered differently at different points in the evolution of the disorder. Higher levels of parental education and parental economic resources were consistently associated with an increase in the likelihood of diagnosis. By contrast, economic resources within a community mattered most when prevalence rates were rapidly increasing. As the epidemic gained strength, a strong neighborhood SES gradient appeared and then weakened as prevalence rates stabilized. However, neighborhood effects did not disappear for everyone. At the end of our observation period, neighborhood SES effects were negligible for children born to educated parents and children not receiving Medi-Cal. By contrast, neighborhood effects remained strong for children born to parents with fewer economic resources.

A comparison of more and less severe cases provides insight into a mechanism that might be driving the autism epidemic. The expression of SES was strongly positive for less severe cases. By contrast, the strength of SES effects for more severe cases was more moderate. The most likely explanation is that the rapid upswing of measured autism prevalence was driven by diagnostic dynamics and knowledge diffusion in wealthy and highly educated communities. Over time, these community effects spilled over into less affluent areas. This finding, combined with the importance of community resources for rising autism prevalence, provides a framework for understanding the autism epidemic as constituted, in significant part, as an “epidemic of discovery” (Grinker 2007).

Within this framework, it is easy to see how neighborhoods can dynamically shape health outcomes. Rather than passively structuring advantages and disadvantages for residents in the same way, the importance of neighborhood socioeconomic status changed over time. The fingerprints left behind by these changes point to the importance of diagnostic resources and ascertainment capacity for rising autism prevalence. By devoting greater attention to temporal and contextual variability in when and how neighborhoods matter, future research might uncover the conditions under which neighborhoods are likely to exacerbate or mitigate health inequalities.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

## Acknowledgments

We thank Christine Fountain, Noam Zerubavel, Ka-Yuet Liu, Diana Dakhallah, Bruce Link, the editors of the *ASR*, and the anonymous reviewers for helpful comments on previous drafts.

**Funding**

This research is supported by the NIH Director’s Pioneer Award program, part of the NIH Roadmap for Medical Research, through grant number 1 DP1 OD003635-01.

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## Biographies

**Marissa D. King** is Assistant Professor of Organizational Behavior at Yale University's School of Management. Her current research examines patterns of antidepressant, stimulant, and antipsychotic utilization. In general, her research analyzes the spatial and temporal dimensions of innovation and diffusion. To understand how large-scale phenomena arise from local behavior, she has studied cases ranging from the rise in autism prevalence during the past decade to the organizational foundations of the antislavery movement in the late-nineteenth century.

**Peter S. Bearman** is the Cole Professor of the Social Sciences at Columbia University and the Director of the Paul F. Lazarsfeld Center for the Social Sciences.

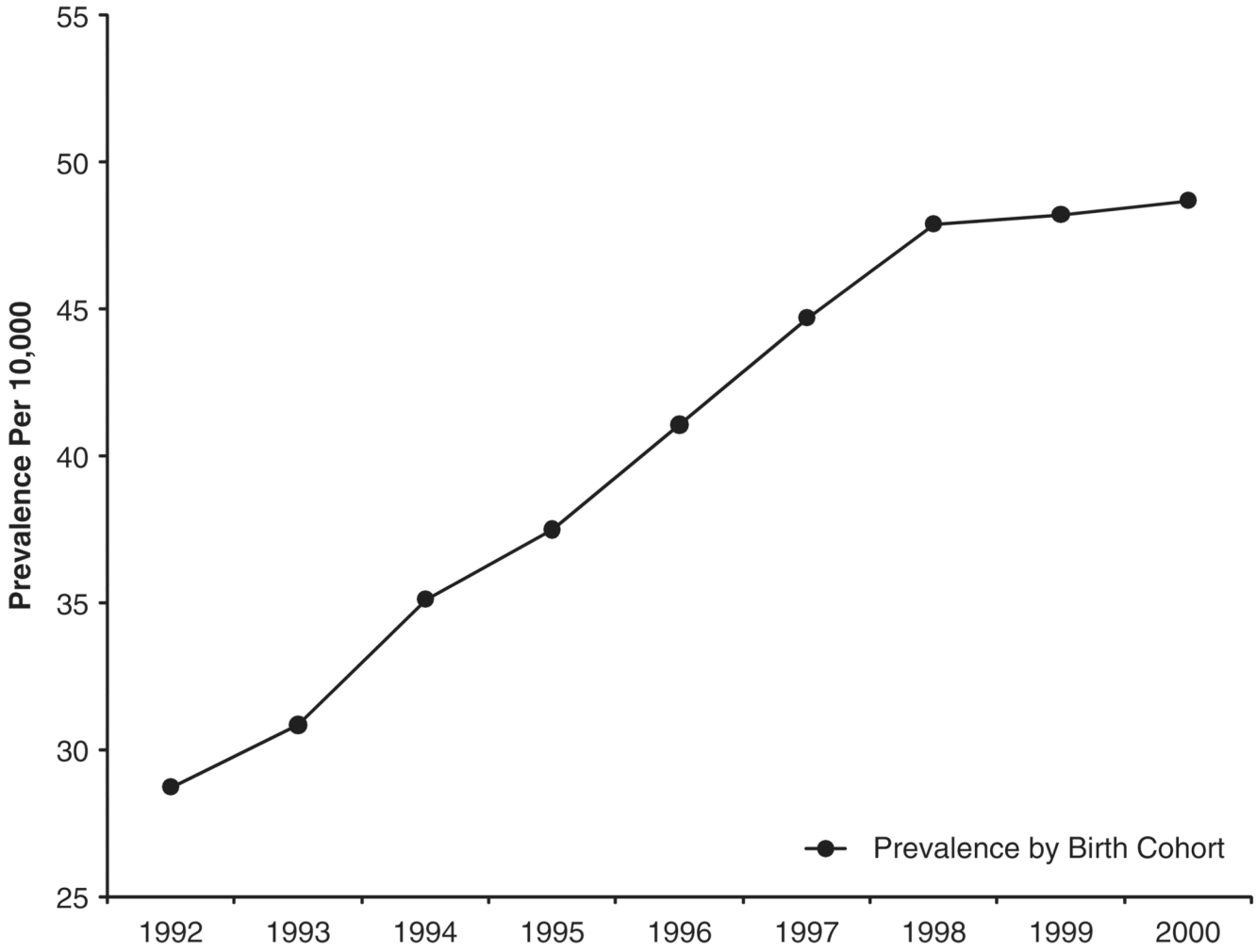


**Figure 1.**
Autism Prevalence by Birth Cohort

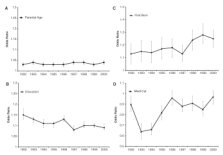

**Figure 2.**
Graphic Representation of the Odds Ratios and Confidence Intervals Obtained for Select Individual-Level Variables
*Note:* The multilevel models used to obtain the point estimates for each birth cohort adjust for all of the individual- and community-level covariates and their interactions listed in Table 1.

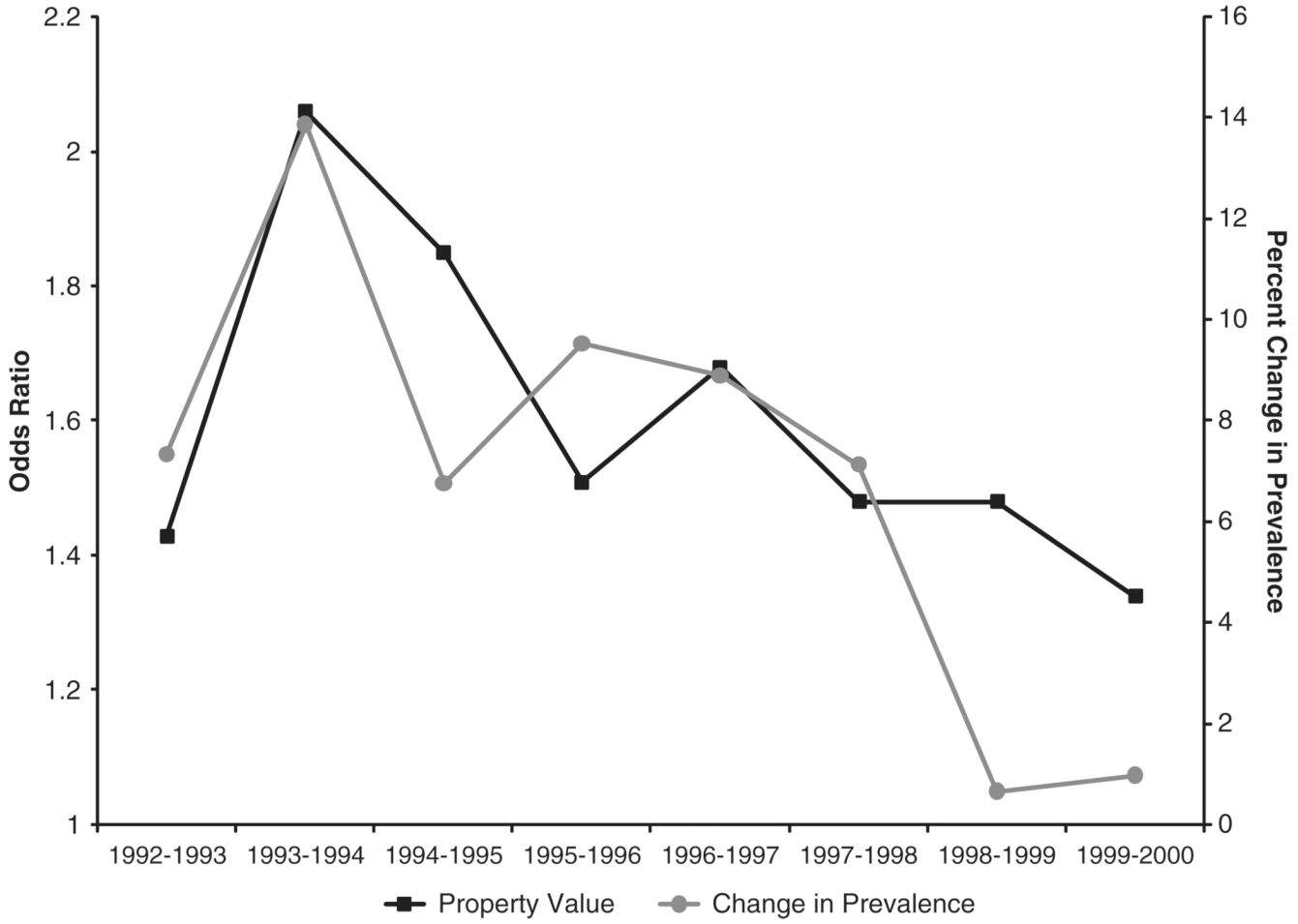


**Figure 3.**
Comparison of Odds Ratios for Property Values and Changes in Prevalence Rates
*Note:* The black line tracks the changing effect size of logged property values at the zip code level across successive birth cohorts. The grey line tracks changes in prevalence rate.

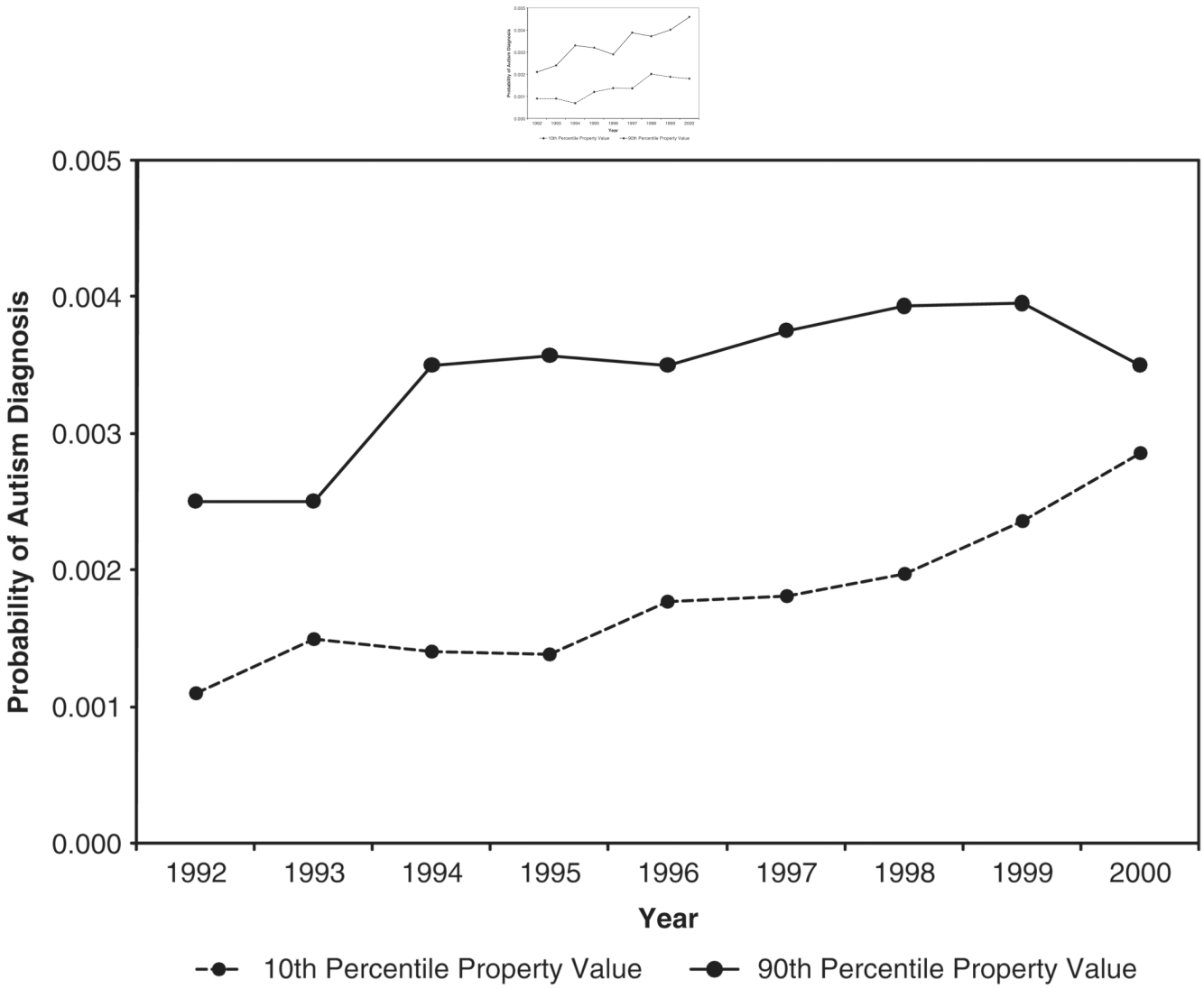


**Figure 4.**

**a.** Community Effect of Property Value on Medi-Cal Recipients

*Note:* Figure shows the probability of diagnosis for children receiving Medi-Cal residing in communities at the bottom decile (dashed line) compared with the top decile (solid line) of property value.

**b.** Community Effect of Property Value on Non–Medi-Cal Recipients

*Note:* Figure shows the probability of diagnosis for children not receiving Medi-Cal residing in communities at the bottom decile (dashed line) compared with the top decile (solid) of property value.

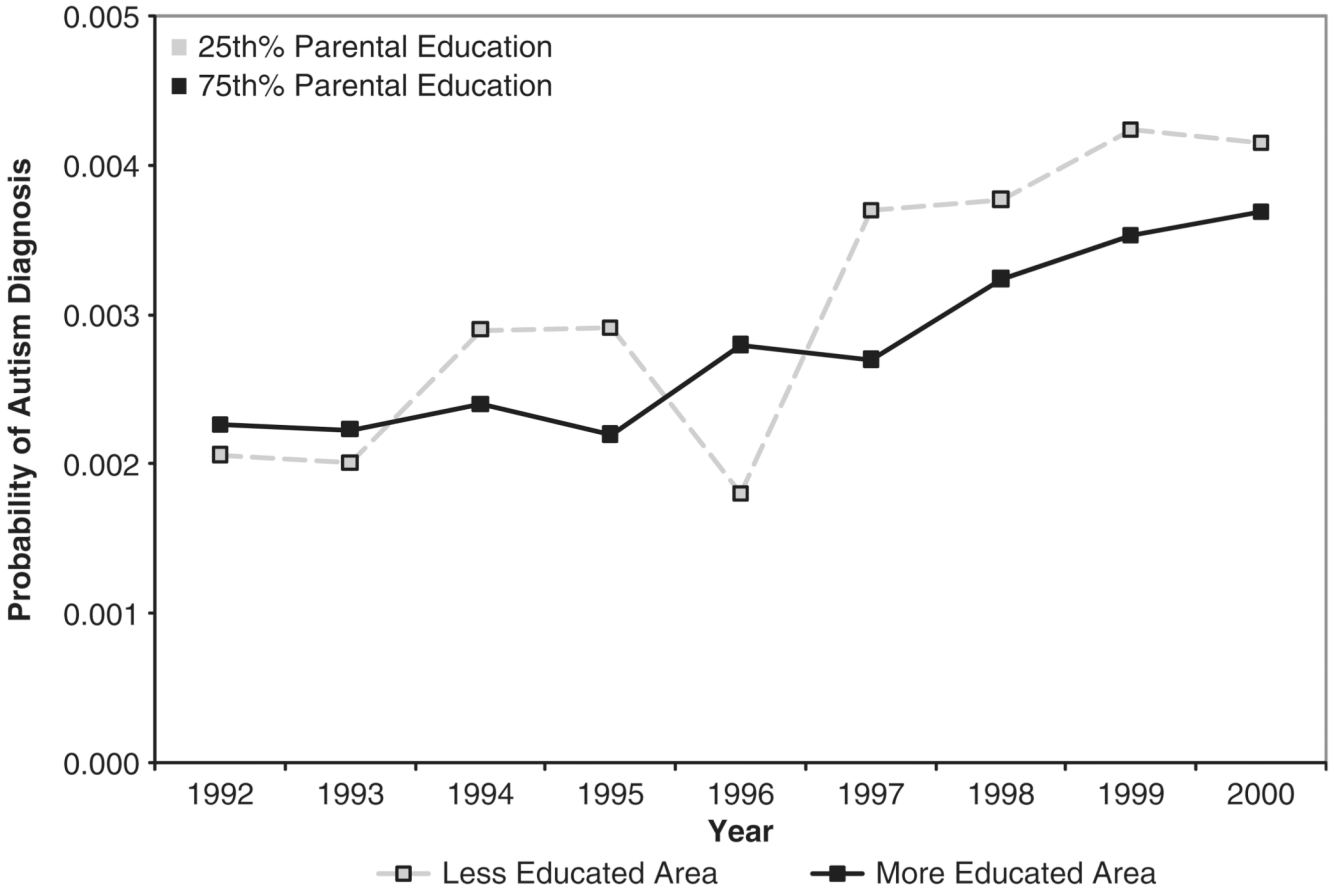


**Figure 5.**

**a.** Community Effect of Education on Children Born to Parents with Less Education

*Note:* Figure shows the probability of diagnosis for children whose parents are at the 25th percentile of education residing in areas with a lower percentage of college graduates (dashed line) compared with a higher percentage of college graduates (solid line).

**b.** Community Effect of Education on Children Born to Parents with More Education

*Note:* Figure shows the probability of diagnosis for children whose parents are at the 75th percentile of education residing in areas with a lower percentage of college graduates (dashed line) compared with a higher percentage of college graduates (solid line).

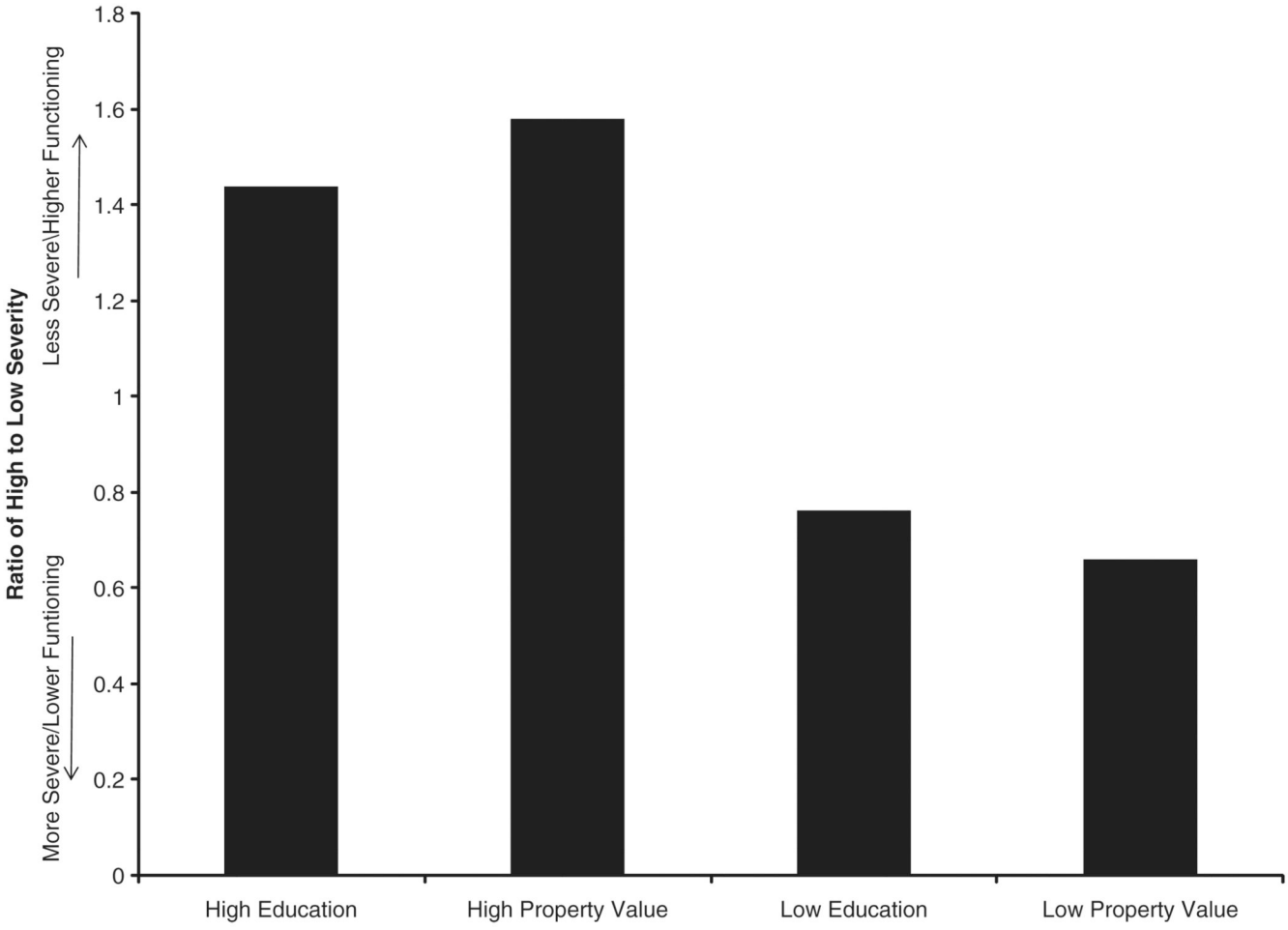


**Figure 6.**
Ratio of Less Severe to More Severe Cases by Community Composition
*Note:* High education communities are in the top decile of education; high property value communities are in the top decile of property value. Low property value and low education are the bottom decile.

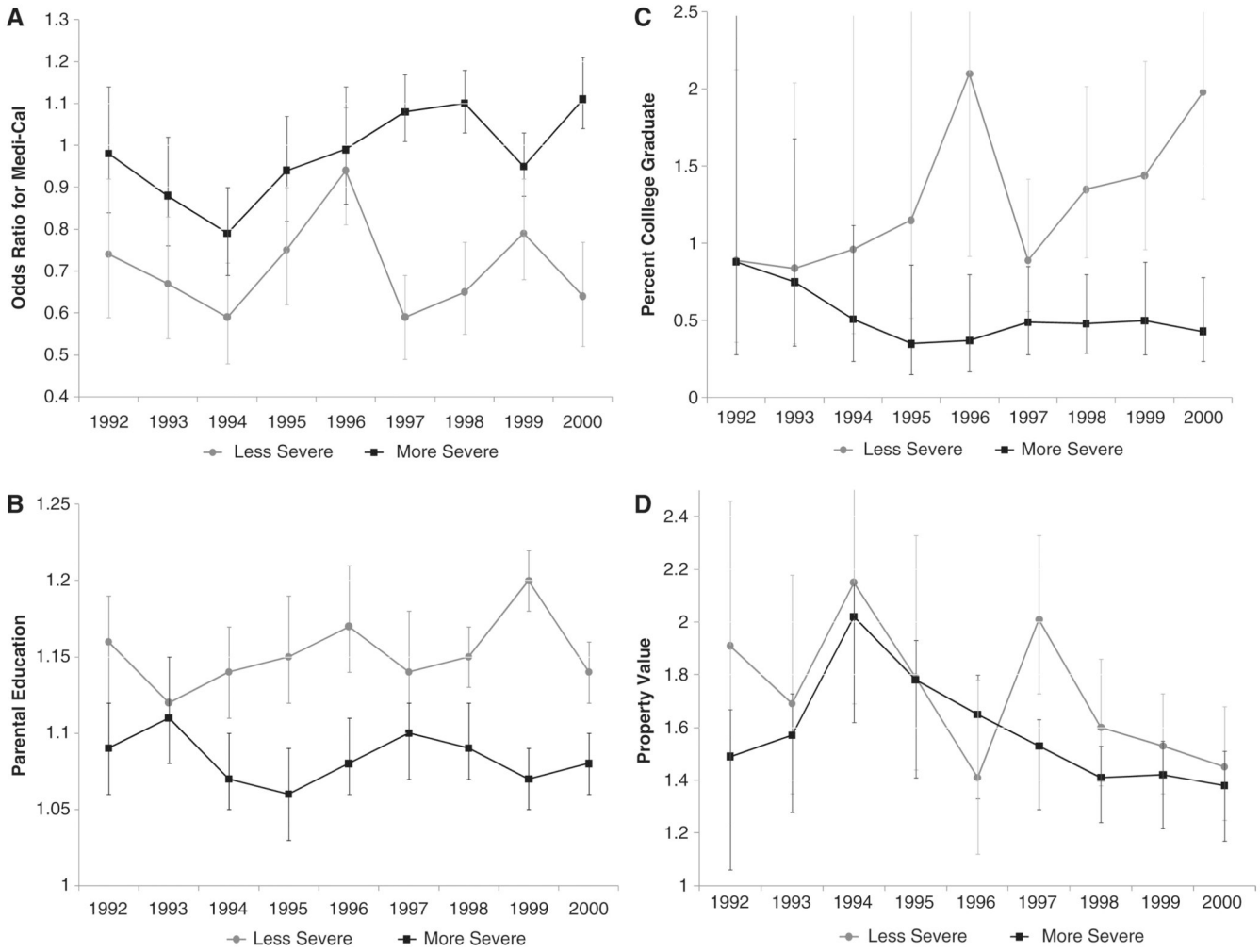


**Figure 7.**
Odds Ratios for Socioeconomic Variables for More Severe and Less Severe Cases

**Table 1**

Risk Factors for Autism

| | 1992 | | 1993 | | 1994 | | 1995 | | 1996 | | 1997 | | 1998 | | 1999 | | 2000 | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **OR** | **95% CI** | **OR** | **95% CI** | **OR** | **95% CI** | **OR** | **95% CI** | **OR** | **95% CI** | **OR** | **95% CI** | **OR** | **95% CI** | **OR** | **95% CI** | **OR** | **95% CI** |
| Intercept | .002 | (.001,.002) | .002 | (.002,.002) | .002 | (.002,.002) | .002 | (.002,.002) | .003 | (.002,.003) | .003 | (.003,.003) | .003 | (.003,.003) | .003 | (.003,.003) | .003 | (.003,.004) |
| *Individual Level* | | | | | | | | | | | | | | | | | | |
| Medi-Cal | **.90** | **(.83,.98)** | **.64** | **(.57, .72)** | **.66** | **(.61,.72)** | **.82** | **(.75, .89)** | **.96** | **(.89,1.03)** | **.88** | **(.83,.95)** | **.91** | **(.84,.98)** | **.85** | **(.79,.93)** | .97 | (.90,1.03) |
| First Born | **1.13** | **(1.05,1.23)** | 1.15 | (1.07, 1.24) | **1.14** | **(1.06,1.23)** | **1.17** | **(1.09, 1.26)** | **1.18** | **(1.10,1.25)** | **1.13** | **(1.07,1.20)** | **1.24** | **(1.16,1.32)** | **1.28** | **(1.21,1.37)** | **1.25** | **(1.18,1.32)** |
| Premature | 1.01 | (.91,1.13) | .79 | (.87,1.24) | 1.07 | (.96,1.20) | 1.07 | (.97, 1.19) | .98 | (.89,1.08) | **1.11** | **(1.02,1.21)** | **1.14** | **(1.04,1.24)** | 1.09 | (.97,1.17) | **1.23** | **(1.13,1.33)** |
| Normal Weight | **.82** | **(.70,.96)** | **.70** | **(.60,.81)** | 1.03 | (.87,1.21) | **.84** | **(.73, .98)** | **.73** | **(.64,.83)** | **.74** | **(.66,.83)** | **.77** | **(.68,.86)** | **.80** | **(.70,.91)** | **.84** | **(.75,.95)** |
| Male | **4.13** | **(3.80,4.50)** | **4.03** | **(3.72,4.37)** | **4.61** | **(4.25,5.00)** | **4.14** | **(3.82, 4.49)** | **4.81** | **(4.48,5.16)** | **4.53** | **(4.25,4.84)** | **4.07** | **(3.79,4.38)** | **4.23** | **(3.96,4.53)** | **4.25** | **(3.99,4.54)** |
| Low Apgar | 1.13 | (.80,1.59) | 1.23 | (.90,1.69) | 1.30 | .98,1.72) | 1.28 | (.96, 1.72) | .92 | (.71,1.20) | 1.17 | (.93,1.48) | 1.21 | (.96,1.53) | .96 | (.75,1.24) | **1.35** | **(1.08,1.68)** |
| Max Parental Age | **1.03** | **(1.03,1.04)** | **1.04** | **(1.04,1.05)** | **1.03** | **(1.02,1.03)** | **1.03** | **(1.03, 1.04)** | **1.03** | **(1.03,1.04)** | **1.04** | **(1.03,1.04)** | **1.04** | **(1.03,1.04)** | **1.03** | **(1.03,1.04)** | **1.04** | **(1.03,1.04)** |
| Education | **1.15** | **(1.13,1.16)** | **1.13** | **(1.11,1.14)** | **1.11** | **(1.09,1.13)** | **1.11** | **(1.09, 1.12)** | **1.13** | **(1.12,1.15)** | **1.08** | **(1.07,1.10)** | **1.10** | **(1.09,1.12)** | **1.10** | **(1.09,1.12)** | **1.09** | **(1.08,1.11)** |
| Missing Father | .99 | (.84,1.16) | **1.42** | **(1.23,1.64)** | 1.15 | (.96,1.40) | 1.12 | (.96, 1.31) | **1.20** | **(1.06,1.35)** | **1.25** | **(1.13,1.38)** | 1.03 | (.92,1.17) | .98 | (.87,1.12) | **1.11** | **(1.00,1.24)** |
| *Neighborhood Level* | | | | | | | | | | | | | | | | | | |
| Logged Property | **1.64** | **(1.38,1.96)** | **1.43** | **(1.24. 1.67)** | **2.06** | **(1.82,2.34)** | **1.85** | **(1.62, 2.10)** | **1.51** | **(1.34,1.71)** | **1.68** | **(1.49,1.88)** | **1.48** | **(1.33,1.65)** | **1.48** | **(1.33,1.65)** | **1.34** | **(1.21,1.49)** |
| Percent College Graduate | .84 | (.46,1.53) | 1.24 | (.74,1.67) | .65 | (.42,1.01) | **.56** | **(.35, .90)** | .87 | (.57,1.34) | .70 | (.46,1.04) | .85 | (.59,1.24) | .82 | (.57,1.19) | .99 | (.69,1.43) |
| *Cross Level* | | | | | | | | | | | | | | | | | | |
| Medi-Cal × Property | .90 | (.74,1.10) | **1.44** | **(1.18, 1.74)** | **1.53** | **(1.28,1.83)** | 1.07 | (.90, 1.28) | **1.29** | **(1.12,1.49)** | **1.19** | **(1.03,1.36)** | 1.00 | (.85,1.17) | 1.15 | (.99,1.34) | **1.52** | **(1.34,1.71)** |
| Education × Percent College | **.83** | **(.75,.93)** | .91 | (.82,1.02) | **.85** | **(.78,.94)** | .92 | (.83, 1.03) | .99 | (.90,1.08) | **.86** | **(.81,.93)** | **.90** | **(.82,.99)** | **.89** | **(.83,.96)** | **.91** | **(.85,.98)** |
| *Random Effects* | *Variance* | *SD* | *Variance* | *SD* | *Variance* | *SD* | *Variance* | *SD* | *Variance* | *SD* | *Variance* | *SD* | *Variance* | *SD* | *Variance* | *SD* | *Variance* | *SD* |
| Intercept | **.10** | **.32** | .11 | .32 | .13 | .36 | .09 | .30 | .15 | .38 | .19 | .44 | .15 | .39 | .12 | .35 | .17 | .42 |
| Medi-Cal Slope | .03 | .16 | .07 | .26 | .03 | .18 | .03 | .18 | .07 | .25 | .05 | .22 | .04 | .19 | .08 | .28 | .05 | .22 |
| Education Slope | **.003** | **.05** | .001 | .04 | .001 | .03 | .001 | .04 | .001 | .04 | .001 | .02 | .002 | .05 | .002 | .04 | .001 | .03 |

*Note:* Odds ratios for factors examined for a potential association with autism risk appear in the column labeled OR. An odds ratio over one indicates an increased risk for autism. An odds ratio of less than one suggests that the variable is associated with a decreased risk for autism. The 95 percent confidence intervals appear in parentheses. Factors that are statistically significant at the $p \leq .05$ level appear in bold. The year heading for each column indicates the birth cohort that the results pertain to. The estimated variance of $\beta_{0k}$, $\tau_{00}$ is statistically significant in the unconditional model for all years at $p < .001$ except 1995.

**Table 2**

Risk Factors for Autism Including Health Care Resources

| | 1992 | | 1993 | | 1994 | | 1995 | | 1996 | | 1997 | | 1998 | | 1999 | | 2000 | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **OR** | **95% CI** | **OR** | **95% CI** | **OR** | **95% CI** | **OR** | **95% CI** | **OR** | **95% CI** | **OR** | **95% CI** | **OR** | **95% CI** | **OR** | **95% CI** | **OR** | **95% CI** |
| Intercept | .002 | (.002, .002) | .002 | (.002, .002) | .002 | (.002, .002) | .002 | (.002, .002) | .002 | (.002, .003) | .003 | (.003, .003) | .003 | (.003, .003) | .003 | (.003, .003) | .003 | (.003, .004) |
| *Individual Level* | | | | | | | | | | | | | | | | | | |
| Medi-Cal | **.89** | **(.82, .97)** | **.72** | **(.66, .80)** | **.66** | **(.59, .74)** | **.81** | **(.74, .88)** | .95 | (.88, 1.02) | **.85** | **(.80, .92)** | **.89** | **(.83, .97)** | **.85** | **(.78, .93)** | **.96** | **(0.90, 1.02)** |
| First Born | **1.13** | **(1.05, 1.23)** | **1.15** | **(1.07, 1.24)** | **1.14** | **(1.05, 1.25)** | **1.17** | **(1.09, 1.26** | **1.18** | **(1.10, 1.26)** | **1.13** | **(1.06, 1.20)** | **1.23** | **(1.17, 1.32)** | **1.28** | **(1.21, 1.37)** | **1.24** | **(1.18, 1.32)** |
| Premature | 1.01 | (.91, 1.13) | .97 | (.86, 1.10) | 1.07 | (.94, 1.23) | **1.07** | **(.961.20)** | .98 | (.88, 1.08) | **1.11** | **(1.01, 1.21)** | **1.14** | **(1.05, 1.24)** | 1.07 | (.97, 1.17) | **1.22** | **(1.13, 1.32)** |
| Normal Weight | .82 | (.70, .96) | **.70** | **(.60, .82)** | 1.03 | (.85, 1.25) | **.84** | **(.73, .98)** | **.73** | **(.64, .83)** | **.74** | **(.66, .83)** | **.77** | **(.69, .86)** | **.80** | **(.70, .90)** | **.84** | **(.75, .95)** |
| Male | **4.13** | **(3.80, 4.50)** | **4.04** | **(3.73, 4.39)** | **4.61** | **(4.15, 5.12** | **4.14** | **(3.82, 4.49)** | **4.82** | **(4.48, 5.17)** | **4.57** | **(4.27, 4.90)** | **4.07** | **(9.80, 4.37)** | **4.23** | **(3.95, 4.52)** | **4.23** | **(3.97, 4.51)** |
| Low Apgar | 1.13 | (.80, 1.59) | 1.23 | (.89, 1.70) | **1.30** | **(.92, 1.85)** | 1.28 | (.96, 1.72) | .92 | (.77, 1.20) | 1.16 | (.92, 1.49) | 1.21 | (.96, 1.52) | .96 | (.75, 1.24) | 1.35 | (1.09, 1.67) |
| Max Parental Age | **1.03** | **(1.03, 1.04)** | **1.04** | **(1.04, 1.05)** | **1.03** | **(1.02, 1.04)** | **1.03** | **(1.03, 1.04)** | **1.03** | **(1.03, 1.04)** | **1.04** | **(1.03, 1.04)** | **1.04** | **(1.03, 1.04)** | **1.03** | **(1.03, 1.04)** | **1.04** | **(1.03, 1.04)** |
| Education | **1.14** | **(1.13, 1.16)** | **1.13** | **(1.11, 1.15)** | **1.11** | **(1.09, 1.13)** | **1.11** | **(1.09, 1.13)** | **1.13** | **(1.12, 1.15)** | **1.09** | **(1.08, 1.11)** | **1.09** | **(1.08, 1.11)** | **1.10** | **(1.09, 1.12)** | **1.08** | **(1.07, 1.09)** |
| Missing Father | .99 | (.84, 1.16) | **1.43** | **(1.24, 1.65)** | 1.15 | (.91, 1.46) | 1.12 | (.96, 1.31) | 1.19 | (1.06, 1.35) | 1.24 | (1.12, 1.39) | 1.04 | (.93, 1.16) | .98 | (.87, 1.12) | 1.11 | (1.01, 1.23) |
| *Neighborhood Level* | | | | | | | | | | | | | | | | | | |
| Logged Property | **1.64** | **(1.37, 1.95)** | **1.59** | **(1.41, 1.80)** | **2.05** | **(1.73, 2.43)** | **1.84** | **(1.62, 2.09)** | **1.52** | **(1.34, 1.72)** | **1.68** | **(1.48, 1.90)** | **1.46** | **(1.32, 1.63)** | **1.47** | **(1.32, 1.64)** | **1.33** | **(1.20, 1.47)** |
| Percent College Graduate | .84 | (.46, 1.53) | .90 | (.57, 1.41) | .61 | (.34, 1.11) | .48 | (.29, .76) | .75 | (.49, 1.18) | .57 | (.37, .87) | .85 | (.58, 1.25) | .74 | (.50, 1.08) | .85 | (.59, 1.23) |
| Number of Child Psychiatrists | .97 | (.94, 1.01) | 1.00 | (.97, 1.02) | .99 | (.96, 1.03) | **1.02** | **(1.004, 1.04)** | **1.03** | **(1.01, 1.05)** | 1.05 | (1.02, 1.07) | 1.02 | (.98, 1.05) | 1.02 | (.99, 1.04) | 1.01 | (.98, 1.04) |
| Number of Pediatricians | 1.005 | (1.003, 1.009) | 1.001 | (.86, 1.10) | **1.01** | **(1.00, 1.01)** | **1.004** | **(1.001, 1.009)** | **1.003** | **(1.00, 1.01)** | 1.003 | (1.00, 1.01) | 1.001 | (.998, 1.004) | **1.002** | **(1.00, 1.01)** | **1.004** | **(1.002, 1.008)** |
| Autism Orgs | .85 | (.64, 1.12) | **.78** | **(.64, .96)** | .82 | (.64, 1.05) | **.73** | **(.57, .94)** | .74 | (.49, 1.18) | .91 | (.78, 1.09) | 1.00 | (.87, 1.15) | .96 | (.86, 1.08) | 1.04 | (.92, 1.17) |
| *Cross Level* | | | | | | | | | | | | | | | | | | |
| Medi-Cal × Property | .89 | (.73, 1.09) | **1.34** | **(1.12, 1.62)** | **1.51** | **(1.21, 1.90)** | 1.07 | (.90, 1.28) | **1.30** | **(1.13, 1.51)** | **1.21** | **(1.04, 1.41)** | **.82** | **(.70, .97)** | 1.15 | (.99, 1.04) | **1.49** | **(1.32, 1.68)** |
| Education × Percent College | .83 | (.74, .93) | .91 | (.82, 1.02) | **.85** | **(.75, .97)** | .92 | (.83, 1.03) | .99 | (.90, 1.09) | **.87** | **(.80, .93)** | **.87** | **(.81, .96)** | **.89** | **(.83, .96)** | **.90** | **(.85, .96)** |
| *Random Effects* | Variance | *SD* | Variance | *SD* | Variance | *SD* | Variance | *SD* | Variance | *SD* | Variance | *SD* | Variance | *SD* | Variance | *SD* | Variance | *SD* |
| Intercept Full Model | **.10** | **.32** | .10 | .32 | .13 | .36 | .09 | .30 | .14 | .37 | .18 | .42 | **.16** | **.41** | .12 | .35 | .18 | .42 |
| Medi-Cal Slope | .02 | .16 | .06 | .24 | .03 | .18 | .04 | .19 | .06 | .24 | .04 | .21 | .05 | .24 | .08 | .28 | .08 | .28 |
| Education Slope | **.003** | **.05** | .001 | .03 | .001 | .03 | .001 | .04 | .001 | .04 | .001 | .03 | .002 | .05 | .002 | .04 | .001 | .03 |

*Note:* Odds ratios for factors examined for a potential association with autism risk appear in the column labeled OR. An odds ratio over one indicates an increased risk for autism. An odds ratio of less than one suggests that the variable is associated with a decreased risk for autism. The 95 percent confidence intervals appear in parentheses. Factors that are statistically significant at the $p < .05$ level appear in bold. The year heading for each column indicates the birth cohort that the results pertain to. The estimated variance of $\beta_{0k}$, $\tau_{00}$ is statistically significant in the unconditional model for all years at $p < .001$ except 1995.

## APPENDIX Table A1

Descriptive Statistics

| | 1992 | | 1993 | | 1994 | | 1995 | | 1996 | | 1997 | | 1998 | | 1999 | | 2000 | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **Mean** | ***SD*** | **Mean** | ***SD*** | **Mean** | ***SD*** | **Mean** | ***SD*** | **Mean** | ***SD*** | **Mean** | ***SD*** | **Mean** | ***SD*** | **Mean** | ***SD*** | **Mean** | ***SD*** |
| *Individual Level* | | | | | | | | | | | | | | | | | | |
| Medi-Cal | .42 | .49 | .43 | .50 | .42 | .49 | .44 | .50 | .43 | .50 | .40 | .49 | .38 | .49 | .38 | .48 | .38 | .49 |
| First Born | .39 | .49 | .39 | .49 | .38 | .49 | .39 | .49 | .38 | .49 | .38 | .49 | .38 | .49 | .38 | .49 | .38 | .49 |
| Premature | .12 | .33 | .13 | .33 | .12 | .33 | .13 | .34 | .14 | .34 | .14 | .35 | .15 | .35 | .15 | .35 | .15 | .35 |
| Normal Weight | .94 | .23 | .94 | .24 | .94 | .24 | .94 | .24 | .94 | .24 | .94 | .24 | .94 | .24 | .94 | .24 | .94 | .24 |
| Male | .51 | .50 | .51 | .50 | .51 | .50 | .51 | .50 | .51 | .50 | .51 | .50 | .51 | .50 | .51 | .50 | .51 | .50 |
| Autism | .002 | .05 | .003 | .05 | .003 | .05 | .003 | .06 | .003 | .06 | .004 | .06 | .004 | .06 | .004 | .06 | .004 | .07 |
| Low Apgar | .01 | .09 | .01 | .10 | .01 | .10 | .01 | .11 | .01 | .11 | .01 | .11 | .01 | .11 | .01 | .11 | .01 | .11 |
| Max Parental Age | 29.90 | 6.88 | 29.97 | 6.93 | 30.45 | 6.90 | 30.20 | 7.07 | 30.38 | 7.09 | 30.46 | 7.17 | 30.54 | 7.20 | 30.66 | 7.21 | 30.77 | 7.20 |
| Education | 12.31 | 3.51 | 12.38 | 3.42 | 12.56 | 3.38 | 12.51 | 3.32 | 12.58 | 3.24 | 12.70 | 3.20 | 12.78 | 3.14 | 12.84 | 3.13 | 12.90 | 3.14 |
| Missing Father | .08 | .27 | .08 | .27 | .04 | .18 | .08 | .26 | .07 | .26 | .09 | .28 | .09 | .29 | .09 | .28 | .08 | .28 |
| Total *N* | 586,193 | | 520,433 | | 523, 594 | | 539, 128 | | 526, 899 | | 514, 988 | | 515, 301 | | 516,173 | | 520433 | |
| *Neighborhood Level* | | | | | | | | | | | | | | | | | | |
| Logged Property | 11.93 | 1.05 | 11.98 | .97 | 12.02 | 0.85 | 12.03 | .80 | 12.07 | .74 | 12.09 | .72 | 12.10 | .70 | 12.12 | .70 | 12.16 | .63 |
| Percent College Graduate | .22 | .15 | .23 | .15 | .24 | .16 | .24 | .16 | .24 | .16 | .25 | .17 | .25 | .17 | .25 | .17 | .26 | .18 |
| Number of Child Psychiatrists | .40 | 1.23 | .44 | 1.38 | .45 | 1.41 | .45 | 1.40 | .45 | 1.37 | .44 | 1.27 | .45 | 1.17 | .50 | 1.28 | .54 | 1.35 |
| Number of Pediatricians | 3.81 | 7.63 | 3.84 | 7.71 | 3.98 | 7.55 | 4.01 | 7.47 | 4.08 | 7.60 | 4.20 | 7.86 | 4.33 | 8.10 | 5.29 | 10.29 | 5.34 | 10.35 |
| Autism Orgs | .02 | .16 | .03 | .15 | .03 | .18 | .03 | .18 | .03 | .19 | .14 | .22 | .04 | .21 | .04 | .22 | .04 | .22 |
| Total *N* | 1,366 | | 1,374 | | 1,353 | | 1,368 | | 1,378 | | 1,388 | | 1,331 | | 1,364 | | 1,345 | |

*Am Sociol Rev*. Author manuscript; available in PMC 2011 May 3.