Public Health Approach to the Study of Mental Retardation

Derek A. Chapman
Virginia Commonwealth University

Keith G. Scott
University of Miami

Tina L. Stanton-Chapman
University of Virginia

Abstract
We applied a public health approach to the study of mental retardation by providing a basic descriptive epidemiological analysis using a large statewide linked birth and public school record database (N = 327,831). Sociodemographic factors played a key role across all levels of mental retardation. Birthweight less than 1000 g was associated with the highest individual-level risk, but the impact varied considerably, depending on maternal educational level. Low maternal education was associated with the largest effects at the population level for mild and moderate/severe mental retardation. Focusing exclusively on specific biomedical causes is of little use in developing public health plans; a broader biosocial perspective reflecting the interactive complexity of the risk factors comprising the various etiological patterns is needed.

Historically, etiologic studies of mental retardation have been focused primarily on biomedical/genetic causes based on the long-standing premise that children with mental retardation can be classified into one of two groups based on the presence or absence of these conditions (Lewis, 1933; Penrose, 1938; Tredgold, 1908; H. Werner & Strauss, 1939; Zigler, 1967). In modern versions of the two-group approach, researchers have noted that individuals with organic mental retardation generally exhibit severe mental retardation (IQ < 50) and show at least one of several hundred prenatal (including genetic), perinatal, or postnatal etiologies. Familial mental retardation typically results in mild mental retardation (IQ between 50 and 70) and is thought to be a result of sociocultural and environmental factors (Burack, 1990). The two-group model is supported by the fact that an identifiable medical etiology is present in up to 70% of children with severe mental retardation but in only 24% of children with mild mental retardation (Accardo & Capute, 1998; Cans et al., 1999; C. Murphy, Boyle, Schendel, Decoufle, & Yeargin-Allsopp, 1998; Yeargin-Allsopp, Murphy, Cordero, Decoufle, & Hollowell, 1997).

Prevention of mental retardation is difficult because the hundreds of known biomedical causes are extremely heterogeneous in nature (D. Alexander, 1998), and a cause has been diagnosed in only 50% of all cases (Winnepenningkx, Rooms, & Kooy, 2003). Some causes, such as Down syndrome, may result in both mild and severe mental retardation (Yeargin-Allsopp et al., 1997). Even in cases where a single gene disorder is present, environmental factors before, during, and after a pregnancy can influence its expression (Horowitz & Haritos, 1998). Further, almost all of the biomedical causes of severe mental retardation are strongly correlated with sociodemographic factors.
typically linked to mild mental retardation (Accardo & Capute, 1998). A continued reliance solely on biomedical investigations using the two-group approach limits our ability to implement effective population-based preventive measures, particularly among cases of mental retardation of "unknown" origin. Instead, we propose that researchers apply the concepts and methods of population-based prevention from the field of public health to the study of developmental disabilities.

The field of public health focuses on preventing disease in populations by identifying risk factors and devising strategies to avoid these risks. From a public health perspective, trying to separate biomedical conditions from their environmental contexts makes little sense. Most diseases have both biological and environmental causes that cannot fully be understood without a balanced approach to both. For example, causal models of heart disease include environmental (e.g., socioeconomic status [SES], work environment), behavioral (e.g., diet, exercise), genetic (e.g., family history), and biological factors (e.g., hypertension, blood lipids). Developmental disabilities such as mental retardation are similar to chronic diseases in that they are typically multifactorial in causation, generally have a long latency or period of time between risk exposure and diagnosis, and are present throughout the lifetime. Based on these similarities, the field of developmental epidemiology emerged, in which the research methods of chronic disease epidemiology are applied to the study of developmental disabilities (Scott, Shaw, & Urbano, 1994).

The public health approach is quite distinct from a clinical or medical approach, which is focused on the identification and treatment of immediate causes of disease within individuals. The contrast with a medical approach is clear when one considers the three basic tenets upon which the public health model rests. First, only a small proportion of the population are at the extremes of high or low risk (Institute of Medicine, 2002). For most chronic diseases, the large majority of cases arise from the mass of the population with risk factor values around the average (Rockhill, 2001). Many risk factors for chronic disease are neither sufficiently (i.e., can cause disease alone) nor necessarily (i.e., must be present to cause disease) causal at the individual level and, thus, are quite inadequate at distinguishing between those individuals who will eventually develop a disease and those who will not (Rockhill, Kawachi, & Colditz, 2000). It follows that preventive strategies will have different consequences based on an individual versus population-level focus. Instead of intervening with just the highest risk individuals, public health officials seek to control the determinants of incidence by shifting the entire distribution of a given exposure in a favorable direction (Hunt & Emslie, 2001), even though the majority of individuals would not have developed the disease anyway. Rose (1981) termed this the prevention paradox because a population shift in the distribution of risk would result in the greatest population improvement, yet be of little benefit to most individuals. For example, most individuals who smoke do not develop lung cancer, but elimination of smoking in the population would result in approximately an 80% decrease in cases of lung cancer (American Lung Association, 2006).

Second, an individual’s risk of illness cannot be considered in isolation from the risk for the population to which they belong (Institute of Medicine, 2002). Causes of intra-individual variation, which tend to be dominated by genetic factors (Rose, 1985), may be very different from the causes of between-population variability, which are often social factors (Schwartz & Diez-Roux, 2001). Researchers who focus exclusively on intra-individual variation may miss important disease determinants and opportunities for prevention (Schwartz & Carpenter, 1999). For example, to prevent lung cancer, one could encourage individual patients to stop smoking. A more efficient approach, however, may be to identify and address factors at the population level that may influence the smoking behaviors of individuals (e.g., advertising, role models, stress). These social antecedents to individual behaviors cannot be detected in intra-individual studies because their exposure is virtually ubiquitous (Schwartz & Diez-Roux, 2001).

Third, understanding the underlying biological causal mechanism is not a prerequisite of prevention. There is a long history in public health of effective preventive measures being introduced long before the discovery of the causative or preventive agent (Wynder, 1994). Successful examples of this include the dramatic decreases in infectious disease and subsequent enhanced longevity over the past 3 centuries due to improvements in nutrition, sanitation, and hygiene—long before vaccines and antibiotics were discovered (Mckeown, 1979). Other well-known historical examples include Lind’s (1753, as cited in Wynder,
Prevention conducted by the Centers for Disease Control and Prevention (hereafter called the Atlanta Study) conducted a series of case-control studies based on the Metropolitan Atlanta Developmental Disabilities Research Center population. The first published reports came in the late 1980s (Strømme & Magnus, 2000). To date, only a few researchers in the United States have examined the role of sociodemographic risk factors in the prevention of mental retardation at the population level. A population-level risk factor identified by epidemiological studies (Malloy & Freeman, 2000).

Early studies on the epidemiology of mental retardation were focused on the identification of specific prenatal, perinatal, and postnatal factors known to be associated with mental retardation (see McLaren & Bryson, 1987, for a review). In more recent investigations from France (Cans et al., 1999) and Norway (Strømme & Hagberg, 2000), researchers have continued to emphasize biomedical factors, particularly among children with severe mental retardation. However, associations between low SES and mild mental retardation have been reported in samples from Bangladesh (Islam, Durkin, & Zaman, 1993) and Norway (Strømme & Magnus, 2000). To date, only a few researchers in the United States have examined the role of sociodemographic risk factors in the prevention of mental retardation at the population level. The first published reports came in a series of case-control studies based on the Metropolitan Atlanta Developmental Disabilities Study (hereafter called the Atlanta Study) conducted by the Centers for Disease Control and Prevention—CDC (Decoufle & Boyle, 1995; Drews, Yeargin-Allsopp, Decoufle, & Murphy, 1995; Yeargin-Allsopp, Drews, Decoufle, & Murphy, 1995). In the Atlanta Study, the investigators utilized a record review from multiple sources, including hospitals and schools, to identify children with mental retardation born in 1975 or 1976 to residents of the five-county metropolitan Atlanta area (Yeargin-Allsopp, Murphy, Oakley, & Sikes, 1992).

In one of the first Atlanta Study publications, Drews et al. (1995) analyzed 458 ten-year-old children with mental retardation and 563 comparison children from the Atlanta Study project data. Low maternal education, low SES, and Black race were associated with mild but not severe mental retardation. In cases where neurological conditions were not present, referred to as isolated mental retardation, these risk factors exhibited similar patterns regardless of severity.

In a second Atlanta Study, Yeargin-Allsopp et al. (1995) focused on differences in the prevalence of mild mental retardation in Black and White children using a sample of 330 case and 563 control children. The overall crude Black–White odds ratio (OR) was 2.6, but it was reduced to 1.8 after adjustment for maternal age, gender, birth order, maternal education, and economic status. When stratified by age at first diagnosis, the adjusted OR was only significant for children diagnosed between 8 to 10 years, indicating that much of the excess prevalence in mental retardation among Black children is preventable.

In the third of the initial Atlanta Study publications, Decoufle and Boyle (1995) examined a sample of 526 case and 650 control children from the Atlanta Study data to examine the relationship between maternal education and mental retardation with and without the presence of neurological conditions. Other factors considered were birth order, median household income, low birthweight, gender, maternal race, and maternal age. For children with isolated mental retardation of any severity, maternal education at the time of delivery was strongly and inversely related and was far the most important predictor of the seven variables in the study. Odds ratios ranged from 6.1 for women with less than 8 years of education to 0.2 for those with 16 years or more. Stratification by race revealed a steeper SES gradient among Black mothers; those with 16 or more years of education were the only group of Black mothers having a lower risk of isolated mental retardation than did White women with 12 years of education.

Camp, Broman, Nichols, and Leff (1998) reported 7-year outcomes of 35,704 children who were part of the Collaborative Perinatal Project, in which they were followed since the prenatal period. All 1,311 children in the sample with a score of less than 70 on the Wechsler Intelligence Scale for Children (WISC) were combined into a single mental retardation group for analyses. For Black children, significant maternal predictors of mental retardation included maternal IQ less than 70, pregnancy weight gain less than 10 pounds, anemia, seizures, and age less than 18 years. Maternal risks for mental retardation among White children included maternal IQ less than 70, pregnancy weight gain less than 10 pounds, and the presence of less than 70 on the Wechsler Intelligence Scale for Children (WISC).
of urinary tract infections. For both White and Black children, however, low SES of the family (44% and 50%, respectively) and maternal education less than 9 years (20%) were associated by far with the largest percentage of cases of mental retardation.

More recently, two groups of researchers utilized administrative datasets to generate large samples of children with mental retardation. Croen, Grether, and Selvin (2001) identified 16,735 children with mental retardation of no known cause from California Department of Developmental Services records. Male gender, low birthweight, Black race, older maternal age, and low maternal education were associated with increased risk for both mild and severe mental retardation. Chapman, Scott, and Mason (2002) investigated the effect of maternal education and maternal age on risk for mild and moderate/severe mental retardation identified through public school records. Older maternal age was associated with increased risk for both levels of mental retardation, but maternal education over 12 years reduced the risk for mild and moderate/severe mental retardation by a factor of 15 and 4, respectively, compared to women with less than 12 years of education.

Our purpose in the current study was to demonstrate how a public health approach can be applied to the study of mental retardation using large population-based administrative data. Our specific research aims were to (a) provide a descriptive epidemiological analysis of mild, moderate, and severe mental retardation and (b) highlight the importance of population-level sociodemographic factors for the prevention of mental retardation.

Method

University of Miami Data Linkage Project

Since 1992, the University of Miami Data Linkage Project has included a large data warehouse with Florida birth certificate records linked to Florida public school records as the core dataset. Birth records were obtained from the Florida Department of Health via an exemption in confidentiality statutes for research entities conducting research approved by the Department. The Florida Department of Education public school records were received for the purpose of conducting this and other research for the Florida Department of Education to improve instruction and planning for instructional services. In addition to numerous reports completed for this department, project staff members have published a number of epidemiological studies based on these linked datasets, covering a variety of outcomes ranging from juvenile arrests to learning disabilities (Avchen, Scott, & Mason, 2001; Hollomon, Dobbs, & Scott, 1998; Hurtado, Claussen, & Scott, 1999; Mason, Chapman, Chang, & Simons, 2003; Mason, Chapman, & Scott, 1999; Stanton-Chapman, Chapman, Bainbridge, & Scott, 2002; Stanton-Chapman, Chapman, & Scott, 2001; Yale, Scott, Gross, & Gonzalez, 2003).

Participants

Participants were a sample of 12- to 14-year-old children who were born in Florida between 1986 and 1988 and attended a Florida public school during the 1999–2000 school year. Florida Department of Health birth certificate records were electronically linked with Florida Department of Education school records to create a final sample of 327,831 children (59.7% White, 28.8% Black, 11.0% Hispanic, and 1.5% other). Of these, 5,677 (17.3 per 1000) were classified as having mild mental retardation, 1,128 (3.4 per 1000) with moderate/severe mental retardation, and 344 (1.0 per 1000) with profound mental retardation. (The categories used by the Florida Department of Education to provide services for children with mental retardation are educable mentally handicapped—EMH, trainable mentally handicapped—TMH, and profoundly mentally handicapped—PMH [Florida State Board of Education, 2006]. Although these labels are not commonly used, the IQ criteria associated with them is consistent with the levels of severity referenced in previous studies, so EMH, TMH, and PMH will be referred to as mild, moderate/severe, and profound mental retardation, respectively, in this paper.)

For most analyses, children with a primary mental retardation placement were compared to the 267,750 children with no exceptionality or those who were classified as gifted. Children with other primary exceptionalities, such as autism or learning disabilities, were excluded from risk analyses (n = 60,081). Information regarding secondary diagnoses were not available for analysis in this study.

Procedure

Extant databases of birth certificate records and public school records were linked based on
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an exact match of the child’s first name, middle initial, last name, and date of birth. This electronic linkage methodology has sensitivity and specificity rates of 97% when compared to manual linkage (Boussy, 1992). Following linkage, personally identifying variables were removed from the secure server and replaced with a system-generated identification number (ID). All analyses were conducted on site using anonymous datasets with a system-generated ID inserted and all personal identifiers removed. A more comprehensive review of the linkage procedures and the validation of this linkage methodology has been described elsewhere (Boussy & Scott, 1993).

Variables

The child’s primary special education placement, school free-lunch status, and race/ethnicity were abstracted from school records and reflected the child’s status at the end of the 1999–2000 school year. Child’s race/ethnicity in school records was coded in a single variable as White, Black, Hispanic, or other. All other variables were obtained from birth certificate records and, thus, reflect the child or family status information that was collected at birth. Childbirth variables abstracted from the birth certificate records were: birthweight, gestational age, 5 minute APGAR score, and a congenital anomalies indicator. Maternal variables included the number of prenatal visits and pregnancy complication, marital illness during pregnancy, and labor/delivery complication indicators. Sociodemographic variables were maternal age, maternal and paternal education, and mother’s marital status.

Analytical Methods

We performed analyses using epidemiological methodology. Although traditional regression/ANOVA models focus on means, slopes, and variances, epidemiological measures of effect focus on proportions and provide an inherently different measure of effect, which can provide unique insight into the study of risk (Mason, Scott, Chapman, & Tu, 2000). An epidemiological perspective is important for three reasons. First, with many uncommon outcomes, it may be mathematically impossible to obtain a large correlation or account for a large proportion of variance. This is particularly true when dealing with cases who have common risk factors, such as low maternal education. Second, because epidemiological measures of effect focus on rates, ratios, and proportions, they map well onto typical concerns of policymakers, such as the reduction in number of cases of a disorder or the specific cost savings associated with successful prevention of a given risk factor (Scott, Mason, & Chapman, 1999). Third, epidemiological measures of effect enable researchers to address the effect of a risk factor on overall rates of a disorder in the population. The distinction between population and individual level risk is important because relatively rare risk factors, such as low birthweight, may have a large effect on individuals who experience it but have small impact on the overall number of cases in the population because so few are exposed to that risk factor. On the other hand, a more prevalent risk factor, such as poverty, may have a modest effect on individuals but may have a large impact in the population because it is so common (Mason et al., 2000).

Risk ratio. The risk ratio, a measure of individual-level risk, is the relative increase or decrease in the probability of a given outcome when one, rather than another condition is true (Greenland & Rothman, 1998). The risk ratio is typically defined as the increased probability of an adverse outcome after exposure to a given risk factor, relative to a comparison or referent group that was not exposed to that risk factor. For ease of interpretation, all risk ratios were computed using the lowest risk group as the referent group, which was assigned a risk ratio of 1.0. For each risk ratio, 95% confidence intervals were also calculated, and because the referent group was the lowest risk category, only risk ratios with a lower limit exceeding 1.0 were statistically significant.

Population attributable fraction percentage. The population attributable fraction percentage, an estimate of risk to the population, weights the risk ratio based on the prevalence of a given risk factor. Assuming a causal relationship, the population attributable fraction percentage estimates the effect of a risk factor on the population as a whole and is the proportion by which the rate of a given outcome would be reduced in the population if the rate associated with a given risk factor was reduced to that of the referent group. Even if a causal relationship cannot be established, the population attributable fraction percentage will still serve to identify the group that is having the largest impact on the overall rate or number of cases in the population (Walter, 1998). This high risk group can be targeted for services or programs aimed at reducing their rates of disorder. A more
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Results

The overall administrative prevalence of mental retardation in the current study was 21.8 per 1000. Mild mental retardation was the most prevalent (17.3 per 1000), followed by moderate/severe mental retardation (3.4 per 1000) and profound mental retardation (1.0 per 1000). Although racial/ethnic differences were found across all levels of mental retardation, they were much more pronounced among children with mild mental retardation (see Table 1). Males had higher rates than did females for mild (19.0 vs. 15.5 per 1000), moderate/severe (4.0 vs. 2.9 per 1000), and profound mental retardation (1.1 vs. 1.0 per 1000).

The crude (or unadjusted) risk ratio and their associated 95% confidence intervals and population attributable fraction percentage were computed separately for each risk factor within each category of mental retardation (see Table 2). The prevalence of each risk factor in this sample is also included in Table 2 to facilitate interpretation of the population attributable fraction percentage. No population attributable fraction percentage was computed when the lower limit of the 95% confidence interval of the risk ratio was less than or equal to 1.0, because the rate of mental retardation in these risk groups was not significantly greater than that of the referent group.

Birthweight less 1000 g was associated with the highest individual-level risk for all levels of mental retardation, and the risk increased with the severity of impairment, as was the case for all of the child birth risk factors in this study. With the exception of older maternal age, all of the sociodemographic variables showed the opposite trend, where the highest risk was found for mild mental retardation. Having less than three prenatal visits (all levels) and labor/delivery complications (profound only) were the only maternal birth risk factors associated with increased risk for mental retardation.

For mild and moderate/severe mental retardation, the highest population attributable fraction percentage was found for participation in the school free-lunch program and maternal education of less than 12 years. Preterm births and maternal education less than 12 years had the highest population attributable fraction percentage for profound mental retardation. We note that the population attributable fraction percentages in Table 2 were computed using unadjusted risk ratios, thus these values should be interpreted separately for each risk factor.

To demonstrate the importance of considering the sociodemographic context of biologic risk, we examined the combined effects of birthweight and maternal education on mild and moderate/severe mental retardation. All risk groups were compared to a single referent, normal birthweight children born to mothers with over 12 years of education (see Table 3). Within each birthweight group, women with more than 12 years of education had the lowest risk for having a child with both mild and moderate/severe mental retardation. However, within each level of maternal education, risk for mild and moderate/severe mental retardation increased with decreasing birthweight. A simple plot of the raw administrative prevalence data for mild and moderate/severe mental retardation demonstrates graphically the importance of both variables (see Figures 1 and 2). Because the prevalence of low birthweight was only 7% in this sample, the largest population attributable fraction percentages were found among normal birthweight infants born to mothers with 12 years of education or less.

Discussion

In this paper we have demonstrated how a public health approach can be applied to the study of mental retardation by providing a basic descriptive epidemiological analysis. The importance of taking exposure prevalence into account is clear when comparing the population attribut-
Table 2. Crude (Unadjusted) Risk Ratios (RR) With 95% Confidence Intervals (CI) and Population Attributable Fraction Percentages (PAF%) for Risk Factors by Level of Mental Retardation

| Risk factor                                | Prevalence (%)
<table>
<thead>
<tr>
<th></th>
<th>Mild RR (95% CI)  PAF%</th>
<th>Moderate/Severe RR (95% CI)  PAF%</th>
<th>Profound RR (95% CI)  PAF%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Child birth</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Birthweight (in g)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;1000</td>
<td>0.3</td>
<td>9.1 (7.5–11.1) 2.1</td>
<td>23.4 (17.1–32.0) 5.3</td>
</tr>
<tr>
<td>1000–1499</td>
<td>0.6</td>
<td>5.8 (5.0–6.7) 2.5</td>
<td>8.9 (6.5–12.1) 3.7</td>
</tr>
<tr>
<td>1500–2499</td>
<td>6.3</td>
<td>2.6 (2.4–2.8) 8.7</td>
<td>3.4 (2.9–4.0) 11.9</td>
</tr>
<tr>
<td>2500+</td>
<td>92.8</td>
<td>Referentb</td>
<td>Referent</td>
</tr>
<tr>
<td>Gestational age (wks)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;37</td>
<td>21.8</td>
<td>2.0 (1.9–2.1) 17.8</td>
<td>2.2 (1.9–2.5) 20.7</td>
</tr>
<tr>
<td>37–42</td>
<td>76.2</td>
<td>Referent</td>
<td>Referent</td>
</tr>
<tr>
<td>&gt;42</td>
<td>2.0</td>
<td>1.4 (1.1–1.7) 0.7</td>
<td>0.9 (0.5–1.6)</td>
</tr>
<tr>
<td>APGAR, 5 min</td>
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<td></td>
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<tr>
<td>0 to 3</td>
<td>0.2</td>
<td>5.0 (3.8–6.7) 0.8</td>
<td>5.9 (3.1–11.2) 0.9</td>
</tr>
<tr>
<td>4 to 6</td>
<td>0.8</td>
<td>3.4 (2.9–4.0) 1.9</td>
<td>6.8 (5.2–9.0) 4.4</td>
</tr>
<tr>
<td>7 to 10</td>
<td>99.0</td>
<td>Referent</td>
<td>Referent</td>
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<td></td>
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<tr>
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<td>1.0 (1.0–1.1) —</td>
<td>1.2 (1.0–1.3) 6.0</td>
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<td>None</td>
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</tr>
<tr>
<td>Maternal birth</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pregnancy complications</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>36.4</td>
<td>1.1 (1.0–1.1) —</td>
<td>1.0 (0.9–1.2) —</td>
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<tr>
<td>None</td>
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<td>33.3</td>
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<td>1.0 (0.9–1.1) —</td>
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<tr>
<td>None</td>
<td>66.7</td>
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<tr>
<td>Labor/delivery complications</td>
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<td></td>
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<tr>
<td>Yes</td>
<td>53.9</td>
<td>1.0 (0.9–1.0) —</td>
<td>1.1 (1.0–1.2) —</td>
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<tr>
<td>None</td>
<td>46.1</td>
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<td>Prenatal visits</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;3</td>
<td>5.0</td>
<td>3.4 (3.2–3.7) 10.7</td>
<td>2.3 (1.9–2.8) 6.1</td>
</tr>
<tr>
<td>3 or more</td>
<td>95.0</td>
<td>Referent</td>
<td>Referent</td>
</tr>
<tr>
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<td></td>
<td></td>
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<tr>
<td>Maternal age</td>
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<td></td>
<td></td>
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<tr>
<td>15 to 17</td>
<td>5.7</td>
<td>2.8 (2.4–3.2) 7.9</td>
<td>1.6 (1.3–2.0) 3.1</td>
</tr>
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<td>18 to 19</td>
<td>8.5</td>
<td>2.3 (2.0–2.6) 8.6</td>
<td>1.2 (1.0–1.5) 1.5</td>
</tr>
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<td>20 to 34</td>
<td>78.9</td>
<td>1.1 (1.0–1.3) 6.1</td>
<td>Referent —</td>
</tr>
<tr>
<td>35 to 39</td>
<td>6.0</td>
<td>Referent</td>
<td>1.8 (1.5–2.2) 4.3</td>
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<td>40 to 44</td>
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<td>1.2 (0.9–1.6) —</td>
<td>3.1 (2.1–4.5) 1.7</td>
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<tr>
<td>Maternal education</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>&lt;12</td>
<td>26.5</td>
<td>8.9 (8.1–9.9) 50.9</td>
<td>2.5 (2.1–2.9) 25.4</td>
</tr>
<tr>
<td>High school</td>
<td>42.5</td>
<td>3.4 (3.1–3.8) 24.8</td>
<td>1.4 (1.2–1.7) 10.8</td>
</tr>
<tr>
<td>&gt;12</td>
<td>31.0</td>
<td>Referent</td>
<td>Referent</td>
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Table 2. Continued

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Prevalence (%)&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Mild</th>
<th>Moderate/Severe</th>
<th>Profound</th>
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<tbody>
<tr>
<td></td>
<td>RR (95% CI)</td>
<td>PAF%</td>
<td>RR (95% CI)</td>
<td>PAF%</td>
</tr>
<tr>
<td>Paternal education</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;12</td>
<td>16.3</td>
<td>7.2</td>
<td>(6.5–8.1)</td>
<td>22.1</td>
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<tr>
<td>High school</td>
<td>36.8</td>
<td>3.2</td>
<td>(2.9–3.6)</td>
<td>17.7</td>
</tr>
<tr>
<td>&gt;12</td>
<td>29.4</td>
<td>Referent</td>
<td>—</td>
<td>Referent</td>
</tr>
<tr>
<td>Not listed</td>
<td>17.5</td>
<td>11.0</td>
<td>(9.9–12.2)</td>
<td>38.3</td>
</tr>
<tr>
<td>Lunch program</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Free</td>
<td>34.2</td>
<td>6.0</td>
<td>(5.6–6.4)</td>
<td>60.5</td>
</tr>
<tr>
<td>Reduced</td>
<td>7.7</td>
<td>2.5</td>
<td>(2.2–2.8)</td>
<td>4.1</td>
</tr>
<tr>
<td>Not eligible</td>
<td>3.1</td>
<td>1.1</td>
<td>(0.9–1.4)</td>
<td>—</td>
</tr>
<tr>
<td>Not apply</td>
<td>55.0</td>
<td>Referent</td>
<td>—</td>
<td>Referent</td>
</tr>
<tr>
<td>Mother married</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>29.1</td>
<td>3.9</td>
<td>(3.7–4.2)</td>
<td>45.8</td>
</tr>
<tr>
<td>Yes</td>
<td>70.9</td>
<td>Referent</td>
<td>—</td>
<td>Referent</td>
</tr>
</tbody>
</table>

<sup>a</sup>Exposure prevalence (or the percentage of the population exposed to each factor).<sup>b</sup>The lowest risk group within each risk factor was chosen as the referent or comparison group. In years.

able fraction percentages to the risk ratios for various risk factors. High risk variables will have very little effect on overall rates of mental retardation in the population if few persons are exposed to them. Alternately, high prevalence risk factors can have a substantial impact at the population level, even if the risk to the individual is low. Analysis of maternal educational level in combination with birthweight demonstrated why distributions of sociodemographic factors in the population cannot be ignored, even for what may appear to be purely biologic factors.

One of our most striking findings in the current study was the major role of maternal and paternal characteristics for both mild and moderate/severe mental retardation. Low maternal education resulted in the highest risk by far at the population level. For example, the population attributable fraction percentage associated with maternal education of less than 12 years was 24.2, 4.8, and 1.8 times that of birthweight less than 1000 g for mild, moderate/severe, and profound mental retardation, respectively. Even among children with profound mental retardation, risk associated with sociodemographic factors were as high as those associated with most child and maternal birth risk factors.

The predictive value of maternal education across all levels of mental retardation is not surprising to anyone familiar with the SES and health literature. Socioeconomic status differences on morbidity and mortality have been found for nearly every disease and condition, with persons of low SES having worse health than those at higher levels (Illsley & Baker, 1991). There is compelling evidence that the relationship between SES and health is graded, such that better health is enjoyed among those at higher levels throughout the SES hierarchy (Adler et al., 1994). Most chronic diseases show a strong SES gradient, including cardiovascular disease, diabetes, arthritis, and cancer (Adler & Ostrove, 1999; Cunningham & Kelsey, 1984; Kaplan & Keil, 1993; National Center for Health Statistics, 1998; Pickering, 1999). The SES gradient also extends across numerous risk factors for disease (Winkleby, Fortmann, & Barrett, 1990) and has also been demonstrated in a number of developmental outcomes, including low birthweight, emotional and behavioral problems, academic achievement, and cognitive functioning (Duncan, Brooks-Gunn, & Klebanov, 1994; Entwisle & Alexander, 1990; Hughes & Simpson, 1995; Korenman, Miller, & Sjaastad, 1995; Kramer, Allen, & Gergen, 1995; Mason et al., 1999; McLoyd, 1998; McLoyd, Ceballo, & Mangelsdorf, 1996; Patterson, Kupersmidt, & Vaden, 1990; Stanton-Chapman et al., 2001; Stanton-Chapman et al., 2002; White, 1982).

The key question is what mechanism(s) lead
Table 3. Single-Referent Risk Ratios (RR), 95% Confidence Intervals (CI), and Population Attributable Fraction Percentages (PAF%) by Birthweight Group, Maternal Educational Level, and Level of Mental Retardation

<table>
<thead>
<tr>
<th>Birthweight/Maternal education level</th>
<th>Prevalence&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Mild</th>
<th>Moderate/Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RR (95% CI)</td>
<td>PAF%</td>
<td>RR (95% CI)</td>
</tr>
<tr>
<td>&lt;1000 g</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;12 yrs</td>
<td>0.1</td>
<td>57.4 (43.1–76.4)</td>
<td>1.1</td>
</tr>
<tr>
<td>High school</td>
<td>0.1</td>
<td>37.7 (27.5–51.6)</td>
<td>0.7</td>
</tr>
<tr>
<td>&gt;12 yrs</td>
<td>0.1</td>
<td>16.3 (8.9–29.8)</td>
<td>0.3</td>
</tr>
<tr>
<td>1000–1499 g</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;12 yrs</td>
<td>0.2</td>
<td>38.3 (30.6–48.0)</td>
<td>1.5</td>
</tr>
<tr>
<td>High school</td>
<td>0.3</td>
<td>20.3 (15.4–26.7)</td>
<td>1.2</td>
</tr>
<tr>
<td>&gt;12 yrs</td>
<td>0.1</td>
<td>11.2 (7.1–17.8)</td>
<td>0.2</td>
</tr>
<tr>
<td>1500–2499 g</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;12 yrs</td>
<td>2.2</td>
<td>19.1 (16.7–21.8)</td>
<td>8.0</td>
</tr>
<tr>
<td>High school</td>
<td>2.6</td>
<td>8.8 (7.5–10.2)</td>
<td>4.1</td>
</tr>
<tr>
<td>&gt;12 yrs</td>
<td>1.5</td>
<td>3.9 (3.0–5.1)</td>
<td>0.9</td>
</tr>
<tr>
<td>2500+ g</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;12 yrs</td>
<td>23.9</td>
<td>9.6 (8.6–10.7)</td>
<td>41.3</td>
</tr>
<tr>
<td>High school</td>
<td>39.5</td>
<td>3.6 (3.3–4.1)</td>
<td>20.3</td>
</tr>
<tr>
<td>&gt;12 yrs</td>
<td>29.3</td>
<td>Referent</td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup>Exposure prevalence, or the percentage of the population exposed to each factor.

Figure 1. Administrative prevalence of mild mental retardation by birthweight group and maternal educational level at delivery.

Figure 2. Administrative prevalence of moderate/severe mental retardation by birthweight group and maternal educational level at delivery.
wastes, ambient and indoor pollutants, and water quality (Evans & Kantowitz, 2002). Postnatally, pathways though which low maternal education influences cognitive development include higher levels of lead in the blood (Canfield et al., 2003; Lanphear et al., 2005), poor nutrition and less cognitive stimulation in the home environment (Bee et al., 1982; Satcher, 1995; Siegel, 1982; Smith, Brooks-Gunn, & Klebanov, 1997), knowledge of and access to early intervention services, and childhood injuries (Chen, Martin, & Matthews, 2006).

Addressing all of these potential pathways related to maternal education may still not be enough to eliminate the large racial disparities found in mental retardation and among mild mental retardation placements in particular. Compared to White children, the prevalence of mild and moderate/severe mental retardation among Black children was 4.5 and 2.1 times higher. These racial disparities have persisted, even after controlling for sociodemographic factors (Yeargin-Allsopp et al., 1995). To fully address this problem, we may need to consider intergenerational risk factors, which involve the mother’s own developmental history. Maternal intergenerational factors clearly play a role in low birthweight (Emanuel, 1986; Emanuel, Filakti, Alberman, & Evans, 1992), and it is likely that other aspects of development, including cognitive development, also have an intergenerational component (Chapman & Scott, 2001). Intergenerational factors may explain, in part, why race differences in mental retardation placements and risk factors associated with mental retardation, such as low birthweight, have persisted, even after controlling for maternal factors, such as age, education, SES, and prenatal care (G. Alexander, Kogan, Himes, Mor, & Goldenberg, 1999; Din-Dzietham & Hertz-Picciotto, 1998; Foster, Wu, Bracken, Semenya, & Thomas, 2000; Migone, Emanuel, Mueller, Daling, & Little, 1991; Starfield et al., 1991).

There are limitations associated with the use of administrative datasets. First, the research is limited to those variables that have already been collected for another purpose. In the current study, for example, IQ and adaptive behavior measures were not available for use in case ascertainment because they were not part of the state student database. Second, the placement of children into special education is based in part on professional judgment, which can result in classification errors. Teachers’ decision making during the initial referral process (e.g., criteria and resources used, interventions initiated prior to referral) has been identified as a potential source of classification inconsistencies (Gerber & Semmel, 1984; Waldron, McLeskey, & Skiba, 1998). Some investigators have argued that Black students are overrepresented in programs for students with mild mental retardation due, in part, to biased referral, evaluation, and placement practices (Artiles & Trent, 1994; Coutinho & Oswald, 1998; Gottlieb, Alter, & Gottlieb, 1994; MacMillan, Gresham, & Bocian, 1998). Others have not found this to be the case (Reschly & Ward, 1991; Tobias, Zibrin, & Menell 1983). There is also a concern that in order to address racial disparities, some children formerly categorized as having mild mental retardation are now being labeled as students with learning disabilities (Coutinho & Oswald, 2000). Although these controversial questions remain unresolved, the use of standardized intelligence tests and criteria that are defined by the state legislature, standard across the state, and subject to audit in Florida, provides consistency that reduces the likelihood of misclassification in the current study.

The potential drawbacks of using administrative datasets are far outweighed by their advantages. Large longitudinal samples can be drawn without the time, expense, and attrition typically associated with prospective studies. Although not equivalent to conducting cognitive and adaptive behavior testing, use of the primary special education placement category alone has been shown to yield prevalence rates that are similar to that found in studies where investigators tested all subjects (Camp et al., 1998). Regardless of whether children would meet the eligibility criteria based on specific clinical criteria, they are still of interest from a public health standpoint, as this is the population of children receiving publicly funded special education services. Finally, large administrative datasets can provide critical public health surveillance data that can be used to (a) serve as a basis for planning more effective prevention activities; (b) identify high risk population groups or geographic areas to target interventions, anticipate needs, and guide analytic studies; (c) provide baseline data that can be used to assess the effectiveness of prevention and control measures (Merwether, 1996).

In summary, a public health approach to the study of mental retardation that includes regular and ongoing surveillance and employs epidemi-
logical measures of effect is needed to complement existing efforts. Collection and use of good surveillance data for mental retardation will lead to more effective prevention and improved targeting for screening and intervention; further, such data can be used to assess the effectiveness of prevention and control measures. Focusing exclusively on specific genetic causes and viewing mental retardation as caused by either biological or environmental factors alone is of little use in developing public health plans. Instead, a broader biosocial perspective that reflects the interactive complexity of the risk factors comprising the various etiological patterns is needed.

References


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Public health approach

D. A. Chapman, K. G. Scott, and T. L. Stanton-Chapman


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