OBJECTIVE: Exposure to thimerosal, a mercury-containing preservative that is used in vaccines and immunoglobulin preparations, has been hypothesized to be associated with increased risk of autism spectrum disorder (ASD). This study was designed to examine relationships between prenatal and infant ethylmercury exposure from thimerosal-containing vaccines and/or immunoglobulin preparations and ASD and 2 ASD subcategories: autistic disorder (AD) and ASD with regression.

METHODS: A case-control study was conducted in 3 managed care organizations (MCOs) of 256 children with ASD and 752 controls matched by birth year, gender, and MCO. ASD diagnoses were validated through standardized in-person evaluations. Exposure to thimerosal in vaccines and immunoglobulin preparations was determined from electronic immunization registries, medical charts, and parent interviews. Information on potential confounding factors was obtained from the interviews and medical charts. We used conditional logistic regression to assess associations between ASD, AD, and ASD with regression and exposure to ethylmercury during prenatal, birth-to-1 month, birth-to-7-month, and birth-to-20-month periods.

RESULTS: There were no findings of increased risk for any of the 3 ASD outcomes. The adjusted odds ratios (95% confidence intervals) for ASD associated with a 2-SD increase in ethylmercury exposure were 1.12 (0.83–1.51) for prenatal exposure, 0.88 (0.62–1.26) for exposure from birth to 1 month, 0.60 (0.36–0.99) for exposure from birth to 7 months, and 0.60 (0.32–0.97) for exposure from birth to 20 months.

CONCLUSIONS: In our study of MCO members, prenatal and early-life exposure to ethylmercury from thimerosal-containing vaccines and immunoglobulin preparations was not related to increased risk of ASDs. Pediatrics 2010;126:656–664
Thimerosal has been used as a preservative in vaccines since the 1930s. It is 49.6% mercury by weight and is metabolized into ethylmercury and thiosalicylate. In 1999, the US Food and Drug Administration estimated that infants who were immunized according to the recommended schedule might have received amounts of ethylmercury that exceed Environmental Protection Agency limits for exposure to methylmercury. As a precautionary measure, the US Public Health Service and the American Academy of Pediatrics urged vaccine manufacturers to remove thimerosal from all infant vaccines as soon as practical and recommended that studies be conducted to investigate the risks associated with ethylmercury exposure from thimerosal-containing vaccines. In response, the Centers for Disease Control and Prevention (CDC) planned studies to examine potential links between ethylmercury exposure and developmental outcomes. The first, a screening analysis that used computerized databases from 3 large managed care organizations (MCOs), examined relationships between ethylmercury exposure from childhood vaccines and several neurodevelopmental conditions. No significant associations with autism spectrum disorder (ASD) were found. Two subsequent CDC-sponsored studies examined neuropsychological outcomes, but ASD was not assessed in either of them.

Our current study was designed to examine the relationships between ethylmercury exposure from thimerosal-containing injections (TCIs), which include thimerosal-containing vaccines and immunoglobulin preparations, and any of 3 ASD outcomes: ASD; autistic disorder (AD); and ASD with regression. We used state-of-the-art assessment tools to confirm ASD outcomes and evaluated both prenatal and postnatal exposure.

**METHODS**
We performed a case-control study in 3 MCOs that participate in the CDC’s Vaccine Safety Datalink. The institutional review boards of the 3 MCOs, CDC, and Abt Associates Inc approved the study. The study protocol was developed before data collection in consultation with a panel of external consultants that included autism advocates and experts in autism, child development, toxicology, epidemiology, biostatistics, and vaccine safety. All subgroup analyses and interaction tests were specified in the study protocol before data collection.

Data sources included MCO computerized data files, abstraction of maternal and child medical charts, and standardized telephone interviews with the children’s biological mothers. Case-children underwent standardized in-person assessments to verify case status. Additional details regarding study design, analyses, and results can be found in technical reports available online.

**Study Population**
Children from each MCO were eligible to participate if they were born between January 1, 1994, and December 31, 1999; had been continuously enrolled in the MCO from birth until their second birthday and were currently enrolled at the time of sample selection; and lived within 60 miles of a study assessment clinic. Children were 6 to 13 years old at the time of data collection. Children had to have lived with their biological mother since birth, and their family had to be fluent in English. Parents provided written consent to participate in the study. Children were excluded if they had the following medical conditions with known links to ASD traits: fragile X syndrome; tuberous sclerosis; Rett syndrome; congenital rubella syndrome; or Angelman syndrome. Recruitment was attempted for all eligible case-children within the MCO populations. Control children were randomly selected from the MCO populations to match case-children within matching strata defined by birth year, gender, and MCO.

**Case Enrollment and Verification**
Potential case-children were identified by searching the MCO computerized records for relevant ASD International Classification of Diseases, Ninth Revision, codes (299.0-ASD or 299.8-PDD NOS), supplemented by text-string searches at 1 MCO, and text strings and autism registries at another. Mothers of case-children were administered the Autism Diagnostic Interview-Revised (ADI-R), and case-children were directly assessed by using the Autism Diagnostic Observation Schedule (ADOS). ASD consists of qualitative abnormalities in reciprocal social interactions and communication and restrictive, repetitive, and stereotyped patterns of behavior. Children who meet study criteria for ASD had ADOS scores that indicated abnormalities in all 3 areas and had ADI-R scores that indicated abnormalities in reciprocal social interactions and either communication or patterns of behavior. Children who met study criteria for AD were a subset of ASD children who had higher scores on all 3 areas of the ADOS, had ADI-R scores that indicated abnormalities in all 3 areas, and had onset at younger than 36 months. Using items from the ADI-R, ASD with regression was defined as the subset of case-children with ASD who reported loss of previously acquired language skills after acquisition. For additional details on case-ascertainment criteria, see the technical report.
Assessors were trained and assessed for reliability using procedures developed by Dr Catherine Lord, 1 of the developers of the ADOS and ADI-R instruments. Assessors were blinded with respect to the thimerosal exposure status of the child and mother.

Controls
To reduce the likelihood that the control group included children with undiagnosed ASD, the lifetime form of the Social Communication Questionnaire (SCQ) was administered as part of the maternal interview for children who had indicated interview was administered as part of the maternal interview for children who had indications of neurodevelopmental difficulties. Seven control-group children with SCQ scores higher than 15 were excluded from the analyses (C. Lord, PhD, personal verbal communication, 2004).

Sample
Physician consent was required before families could be recruited. Consent was requested for all case-children who met the eligibility requirements that could be ascertained from MCO records, before recruitment and eligibility calls, and for a randomly selected sample of controls that were matched to case-children within birth year, gender, and MCO matching strata. This sampling stage resulted in a pool of controls with physician consent (Fig 1). As case-children were confirmed as eligible and enrolled as study participants, random samples of matched controls were selected for recruitment from the pool of controls. The targeted control to case ratio was 3 to 1 within each matching stratum. Controls who were matched to case-children who later did not meet the study’s clinical assessment criteria for ASD were excluded from the analyses.

Ethylmercury Exposure From TCIs
Children’s histories of TCI receipts were obtained from computerized immunization records and abstracted medical charts. Mercury content of the TCIs was determined by linking the manufacturer, lot number, and year of receipt information to published data and manufacturer records. Maternal receipt of immunoglobulins, tetanus toxoids, and diphtheria-tetanus during pregnancy was primarily ascertained from medical charts (81 receipts) and less often from maternal interviews (6 receipts). Maternal receipt of flu vaccine during pregnancy was infrequently recorded in medical charts (2 receipts) and primarily came from maternal report (36 receipts). We defined postnatal exposure as micrograms of ethylmercury divided by the weight of the child (in kilograms) at the time of administration of each TCI. Exposures were summed over the time periods of interest. Prenatal exposure was defined as the cumulative ethylmercury amount (in micrograms) of all TCIs received by the mother during her pregnancy with the child.

Covariates
Covariates tested for inclusion in the statistical models were child and family characteristics (maternal and paternal age at birth of child, maternal education level, family income, single-parent status, birth order, twin/triplet, breastfeeding duration); maternal exposures during pregnancy (exposure to mercury from fish, from cosmetics or medicines, or from dental fillings; use of tobacco, alcohol, or illegal drugs; use of folic acid or valproic acid; viral infections; lead exposure); child birth conditions (birth weight, Apgar score, birth asphyxia, respiratory distress syndrome, hyperbilirubinemia); early-childhood health conditions (anemia, lead exposure, pica, encephalitis); and maternal health care-seeking behavior (Kotelchuck prenatal care index, cholesterol and Papanicolaou test screenings).

Statistical Analysis
We used the SAS 9.1 (SAS Institute Inc, Cary, NC) PHReg procedure to fit conditional logistic regression models that accounted for matching within strata defined by birth year, gender, and MCO to estimate the odds ratios (ORs) for ASD outcomes associated with increases in ethylmercury exposure for 4 different periods: prenatal; birth to 1 month; birth to 7 months; and birth to 20 months. Models were fit with and without covariates. Covariates were retained in the final models if they satisfied a change-in-estimate criterion evaluated by dropping terms that resulted in a <10% change in exposure coefficients relative to a full model with all potential covariates.

All tests were 2-tailed, and statistical significance was set at P < .05. To facilitate interpretation of results, we present ORs in 2 forms. The first is the OR associated with an increase of 1 unit of exposure, in which 1 unit equals 1 μg of ethylmercury for prenatal exposure or 1 μg of ethylmercury per kilogram of body weight for postnatal exposure. The second, which is used as an indication of the difference between low and high exposure, is the OR for a difference in exposure equal to 2 SDs for each particular exposure measure of interest. A 2 SD increase in exposure can be thought of as roughly the difference between the 10th and 90th percentiles on these measures. For the measure of prenatal ethylmercury exposure, 2 SDs is equal to 16.34 μg or a little more than the amount in typical Rhoγam injections in use during the years included in our study. Two SDs of the birth-to-1-month measure is 4.08 μg/kg, and 2 SDs for the birth-to-7-month and the birth-to-20-month measures are 15.56 and 17.82 μg/kg, respectively.

For the ASD outcome, for each 2 SD increase in mercury received in the prenatal, birth-to-1-month, birth-to-7-month, and birth-to-20 month periods,
posthoc calculations indicate that the study had ~80% power to detect ORs of 1.5, 1.7, 2.1, and 2.2, respectively.

In addition, by adding model terms to test for interactions, we examined whether the effect of postnatal thimerosal exposure on the risk of the 3 ASD outcomes was modified by the gender of the child, concurrent

FIGURE 1
Sample flow diagram. a Potential case-children had a diagnosis of ASD in their medical charts (see text for eligibility criteria). b Before recruitment, physician consent was required. c Physician consent was obtained for 4854 potential controls. From this group, random samples of controls (totaling 2760) were drawn, as needed, to match participating case-children within birth year, gender, and MCO matching strata. d Ineligibility was determined during recruitment or eligibility calls. e Ineligibility was determined from information obtained from parent interview, SCQ, or medical chart abstraction. f Controls were matched to case-children by birth year, gender, and MCO. If there were no potential case-children who met study criteria for ASD within a birth year, gender, and MCO matching stratum, the controls in that stratum could not be used in the analysis.
RESULTS

Characteristics of the Children

Of 771 potential case-children and 2760 controls selected for recruitment, 103 case-children (13.4%) and 316 controls (11.4%) were found to be ineligible (Fig 1). Among the 668 case-children and 2444 controls remaining, 321 case-children (48.1%) and 774 controls (31.7%) participated in all phases of the study. Reasons for nonparticipation included inability to locate (cases: n = 27 [4.0%]; controls: n = 467 [19.1%]), refusal to participate (cases: n = 255 [38.2%]; controls: n = 1203 [49.2%]), and difficulty scheduling or completing the clinical assessment (cases: n = 65 [9.7%]). Ninety-four control mothers and 14 case-mothers participated in a refusal survey. Among control mothers, lack of time (62%) and distrust or ambivalence toward research (23%) were stated as primary reasons for nonparticipation. For case-mothers, the primary reasons were lack of time (50%), belief that child was ineligible (14%), and maternal health (14%). Among the 774 control participants, 12 (1.6%) were excluded because the analysis of their medical charts and parent interview data revealed they had exclusionary conditions. In addition, 10 controls were not included in the analysis because there were no case-children who met study criteria for ASD within the relevant birth year, gender, and MCO matching strata (Fig 1).

Of the 321 potential case-children who participated in standardized assessments, 256 (79.8%) met study criteria for ASD (Fig 1). Among those who met criteria for ASD, 187 (73%) met the stricter criteria for AD, and 49 (19%) met criteria for ASD with regression.

Children were 6 to 13 years old at the time of data collection, 85% were male, and 7% had low birth weight (Table 1). Maternal age, maternal education, maternal marital status, and paternal age were similar for case-children and controls.

### Relationships of ASD Outcomes to Ethylmercury Exposure

On average, case-children and control children had similar cumulative ethyl-
Exposures in the age ranges from birth to 7 months and birth to 20 months were both associated with decreased risk of all 3 ASD outcomes. We found no significant differences in exposure effects between boys and girls for any of the ASD outcomes, no evidence that higher prenatal exposure exacerbated the effects of postnatal exposure, and no evidence that concurrent ethylmercury exposure and antimicrobial use was associated with risk of ASDs (for full model results, see the technical report). 10

TABLE 2 Cumulative Exposure to Ethylmercury According to Exposure Period

<table>
<thead>
<tr>
<th>Case-Control Comparison/Exposure Period</th>
<th>Case-Children (n = 256)</th>
<th>Cumulative Exposure Amount, μg</th>
<th>Controls (n = 752)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Minimum</td>
<td>Maximum</td>
</tr>
<tr>
<td>Case-children with ASD (n = 187) vs controls (n = 724)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prenatal</td>
<td>2.70</td>
<td>0</td>
<td>74.00</td>
</tr>
<tr>
<td>Birth to 1 mo (28 d)</td>
<td>9.01</td>
<td>0</td>
<td>45.00</td>
</tr>
<tr>
<td>Birth to 7 mo (214 d)</td>
<td>101.13</td>
<td>0</td>
<td>190.83a</td>
</tr>
<tr>
<td>Birth to 20 mo (609 d)</td>
<td>133.58</td>
<td>0</td>
<td>300.00</td>
</tr>
<tr>
<td>Case-children with AD (n = 187) vs controls (n = 724)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prenatal</td>
<td>2.96</td>
<td>0</td>
<td>62.75</td>
</tr>
<tr>
<td>Birth to 1 mo (28 d)</td>
<td>9.40</td>
<td>0</td>
<td>45.00</td>
</tr>
<tr>
<td>Birth to 7 mo (214 d)</td>
<td>101.42</td>
<td>0</td>
<td>190.83</td>
</tr>
<tr>
<td>Birth to 20 mo (609 d)</td>
<td>134.64</td>
<td>0</td>
<td>253.33</td>
</tr>
<tr>
<td>Case-children with ASD with regression (n = 49) vs controls (n = 652)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prenatal</td>
<td>3.34</td>
<td>0</td>
<td>25.00</td>
</tr>
<tr>
<td>Birth to 1 mo (28 d)</td>
<td>9.08</td>
<td>0</td>
<td>45.00</td>
</tr>
<tr>
<td>Birth to 7 mo (214 d)</td>
<td>101.09</td>
<td>0</td>
<td>190.83</td>
</tr>
<tr>
<td>Birth to 20 mo (609 d)</td>
<td>140.12</td>
<td>0</td>
<td>253.33</td>
</tr>
</tbody>
</table>

Ethylmercury from thimerosal-containing vaccines and immunoglobulins. For descriptive purposes, the postnatal exposure amounts shown here were not divided by weight at time of vaccine receipt. Most vaccines in use at the time that case-children were infants contained 0, 12.5, or 25 μg of ethylmercury per dose. Among case-children with ASD, mean prenatal ethylmercury exposure was 2.70 and ranged from 0 to 74 μg of ethylmercury from thimerosal-containing vaccines and immunoglobulins received by the mother during her pregnancy with the study child.

- Maximum from maternal receipt of 2 immunoglobulins during pregnancy, each containing 50 μg of ethylmercury.
- Maximum from child receipt of hepatitis B immunoglobulin (25 μg) at birth and hepatitis B vaccine (12.5 μg) at 28 days of age.
- Maximum from child receipt of 3 hepatitis B (12.5 μg), 3 diphtheria-tetanus-acellular pertussis (25 μg), and 3 Hib (25 μg) vaccines in 1st 7 months.
- Maximum from child receipt of 2 hepatitis B (12.5 μg), 1 rabies (20 μg), 3 diphtheria-tetanus toxoids-pertussis (24.27, 23.28, and 23.28 μg), and 3 Hib (25 μg) vaccines in 1st 7 months.

DISCUSSION

We found no evidence that increasing ethylmercury exposure from TCIs was associated with increased risk of ASD, AD, or ASD with regression. The unadjusted model results showed no significant associations between exposure and risk of ASD or AD. In the covariate adjusted models, we found that an increase in ethylmercury exposure in 2 of the 4 exposure time periods evaluated was associated with decreased risk of each of the 3 ASD outcomes. We are not aware of a biological mechanism that would lead to this result. Analyses to explore potential explanations are presented in the technical report. 10,11 For example, there were no significant differences between case-children and controls in the numbers of vaccines received up to ages 7 or 20 months. Case-children were more likely to have received thimerosal-free or combined Hib vaccines than controls and more likely to have received thimerosal-free hepatitis B vaccines, resulting in the slightly lower cumulative exposure amounts. Knowledge that a child had ASD was not likely to have influenced choice of vaccines because none of the case-children had ASD diagnoses by 7 months old, and few had diagnoses by 20 months. There was no significant association between having an older autistic sibling and exposure levels. In addition,
there was no substantive difference in the association between thimerosal exposure and autism outcomes.

### TABLE 3  Association Between Thimerosal Exposure and Autism Outcomes

<table>
<thead>
<tr>
<th>Exposure Measure</th>
<th>Unadjusted Model Results (No Covariates)</th>
<th>Covariate Adjusted Model Results</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1-U Difference in Exposure, OR (95% CLs)a</td>
<td>2-SD Difference in Exposure, OR (95% CLs)b</td>
</tr>
<tr>
<td>Case-children with ASD (n = 256) vs controls (n = 752)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prenatal</td>
<td>1.007 (0.990, 1.025)</td>
<td>1.125 (0.846, 1.465)</td>
</tr>
<tr>
<td>Birth to 1 mo (28 d)</td>
<td>0.973 (0.858, 1.054)</td>
<td>0.894 (0.644, 1.420)</td>
</tr>
<tr>
<td>Birth to 7 mo (214 d)</td>
<td>0.992 (0.966, 1.020)</td>
<td>0.885 (0.592, 1.351)</td>
</tr>
<tr>
<td>Birth to 20 mo (609 d)</td>
<td>0.991 (0.965, 1.016)</td>
<td>0.862 (0.533, 1.336)</td>
</tr>
<tr>
<td>Case-children with AD (n = 187) vs controls (n = 724)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prenatal</td>
<td>1.010 (0.991, 1.030)</td>
<td>1.179 (0.862, 1.614)</td>
</tr>
<tr>
<td>Birth to 1 mo (28 d)</td>
<td>1.010 (0.927, 1.100)</td>
<td>1.040 (0.732, 1.478)</td>
</tr>
<tr>
<td>Birth to 7 mo (214 d)</td>
<td>0.981 (0.962, 1.022)</td>
<td>0.875 (0.545, 1.404)</td>
</tr>
<tr>
<td>Birth to 20 mo (609 d)</td>
<td>0.992 (0.964, 1.021)</td>
<td>0.884 (0.520, 1.449)</td>
</tr>
<tr>
<td>Case-children with ASD with regression (n = 49) vs controls (n = 652)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prenatal</td>
<td>1.031 (0.993, 1.072)</td>
<td>1.656 (0.885, 3.095)</td>
</tr>
<tr>
<td>Birth to 1 mo (28 d)</td>
<td>0.958 (0.794, 1.108)</td>
<td>0.769 (0.390, 1.519)</td>
</tr>
<tr>
<td>Birth to 7 mo (214 d)</td>
<td>0.936 (0.880, 0.994)c</td>
<td>0.355 (0.138, 0.915)c</td>
</tr>
<tr>
<td>Birth to 20 mo (609 d)</td>
<td>0.953 (0.900, 1.008)</td>
<td>0.473 (0.154, 1.470)</td>
</tr>
</tbody>
</table>

 Covariates for ASD models: birth weight, maternal age, birth order, breastfeeding duration, family income, maternal health care–seeking behavior (Kotelchuck inadequacy of prenatal care, use of cholesterol screening, use of Papnicolaou test screening), maternal exposures during pregnancy with study child (alcohol use, folic acid use, viral infection, lead exposure), and early childhood health conditions (anemia between 6 and 30 months of age; pica before 5 years of age). Covariates for AD models: birth weight, maternal age, birth order, breastfeeding duration, family income, maternal health care–seeking behavior (Kotelchuck inadequacy of prenatal care, use of cholesterol screening, use of Papnicolaou test screening), maternal exposures during pregnancy with study child (folic acid use), and early childhood health conditions (anemia between 6 and 30 months of age; pica before 5 years of age). Covariates for ASD with regression models: birth weight, maternal age, family income, maternal education level, maternal exposures during pregnancy with study child (alcohol use).

 a DR for autism associated with a 1-U increase in exposure. For prenatal exposure, 1 U = 1 μg of ethylmercury. For postnatal exposure, 1 U = 1 μg of ethylmercury per 1 kg of body weight at time of vaccine or immunoglobulin receipt.

 b OR for autism associated with a 1-U increase in exposure. For prenatal exposure, 1 U = 1 μg of ethylmercury. For postnatal exposure, 2 SDs of birth-to-1-month exposure measure is 4.06 μg/kg. Similarly, 2 SDs of birth-to-7-month and birth-to-20-month exposures are 15.56 μg/kg and 17.82 μg/kg.

 Covariates for the MCO and exposure level that differed according to case/control status, then the results could be biased. Reporting bias can also be a concern with case-control studies, particularly because of differential recall of exposures by case-children compared with

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controls. For measures of prenatal exposure, we used information obtained from the maternal interview on vaccination and immunoglobulin exposures during pregnancy. However, we attempted to minimize the effects of recall bias by also using information recorded in maternal medical charts.

ASDs are behaviorally defined and therefore difficult to diagnose definitively. Among the strengths of our study was the use of state-of-the-art assessment tools to validate the ASD diagnoses in children’s medical charts and the use of the SCQ assessment tool to exclude children with potentially undiagnosed ASDs from the control group. Additional strengths were that measures of childhood exposure to ethylmercury from TCIs were derived from computerized and medical chart data sources and were therefore not susceptible to recall bias, and the collection of extensive information regarding potential confounding factors.

Given that a large-scale prospective randomized trial is not ethically feasible, no single study can definitively establish or disprove the hypothesis that thimerosal exposure increases the risk of ASDs. Our study adds to the growing base of epidemiologic studies that have been conducted to investigate the hypothesis. In 2004 the immunization safety review committee of the Institute of Medicine published a review of the research evidence concerning relationships between thimerosal-containing vaccines and ASDs. The committee discussed the strengths and limitations of each study reviewed and concluded that the evidence available at that time did not demonstrate a link between thimerosal-containing vaccines and ASDs. Subsequently, 2 ecological studies have found that the prevalence of ASDs continued to increase after the removal of thimerosal from childhood vaccines that began in 1999 and 2001.

The results of our study of MCO members do not support the hypothesis that ethylmercury exposure from TCIs administered prenatally or during infancy is related to increased risk of ASDs.

CONCLUSION

The results of our study of MCO members do not support the hypothesis that ethylmercury exposure from TCIs administered prenatally or during infancy is related to increased risk of ASDs.

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REFERENCES
