Maternal Metabolic Conditions and Risk for Autism and Other Neurodevelopmental Disorders

OBJECTIVE: We examined whether metabolic conditions (MCs) during pregnancy (diabetes, hypertension, and obesity) are associated with autism spectrum disorder (ASD), developmental delays (DD), or impairments in specific domains of development in the offspring.

METHODS: Children aged 2 to 5 years (517 ASD, 172 DD, and 315 controls) were enrolled in the CHARGE (Childhood Autism Risks from Genetics and the Environment) study, a population-based, case-control investigation between January 2003 and June 2010. Eligible children were born in California, had parents who spoke English or Spanish, and were living with a biological parent in selected regions of California. Children’s diagnoses were confirmed by using standardized assessments. Information regarding maternal conditions was ascertained from medical records or structured interview with the mother.

RESULTS: All MCs were more prevalent among case mothers compared with controls. Collectively, these conditions were associated with a higher likelihood of ASD and DD relative to controls (odds ratio: 1.61 [95% confidence interval: 1.10–2.37; odds ratio: 2.35 [95% confidence interval: 1.43–3.88], respectively). Among ASD cases, children of women with diabetes had Mullen Scales of Early Learning (MSEL) expressive language scores 0.4 SD lower than children of mothers without MCs (P < .01). Among children without ASD, those exposed to any MC scored lower on all MSEL and Vineland Adaptive Behavior Scales (VABS) subscales and composites by at least 0.4 SD (P < .01 for each subscale/composite).

CONCLUSIONS: Maternal MCs may be broadly associated with neurodevelopmental problems in children. With obesity rising steadily, these results appear to raise serious public health concerns. Pediatrics 2012;129:e1121–e1128

WHAT’S KNOWN ON THIS SUBJECT: Diabetes during pregnancy has been associated with general development impairments in offspring; however, associations between autism and maternal diabetes have been inconsistent. Few studies have examined related conditions accompanied by underlying increased insulin resistance and their association with developmental outcomes.

WHAT THIS STUDY ADDS: This population-based study in young children provides evidence that maternal metabolic conditions are a risk factor for autism, developmental delay without autistic symptoms, and impairments in several domains of development, particularly expressive language, after adjusting for sociodemographic and other characteristics.
Autism spectrum disorders (ASDs) are neurodevelopmental disorders characterized by impairments in social interaction, communication deficits, and stereotyped behaviors. Approximately 1 in 110 children has ASD, and the cumulative incidence of this disorder seems to be increasing. Moreover, 1 in 85 children has other developmental delays (DDs). Language and cognitive delays are also seen in a majority of children with ASD, suggesting that common exposures may be contributing to the pathology of both of these developmental disorders. To date, the etiology of ASD is unknown; however, several studies suggest that its pathogenesis most likely begins in utero. Similarly, the causes of cognitive impairment remain unknown for most children. An association between general developmental impairments and maternal diabetes has been previously observed. Studies involving women with diabetes found correlations between gestational measures of maternal lipid and glucose metabolism and poorer performance of the offspring on standardized IQ tests and motor development assessments. Dionne et al reported significant expressive language impairments in young children born to mothers with gestational diabetes (GDM) compared with children of women without diabetes. Moreover, 2 small studies demonstrated mild deficits in recognition memory performance in infants of mothers with diabetes, suggesting aberrations in hippocampal function. Abnormalities within the limbic system have also been documented in children with ASD, and language deficits are among the core features of this disorder. However, the association between ASD and maternal diabetes has not been consistently reported in population-based studies, highlighting the need for further investigation.

Insulin resistance and chronic inflammation in type 2 diabetes (T2D) and related conditions, including obesity and hypertension, have been well established. In addition, because sensitivity to insulin naturally decreases during gestation, women with impaired glucose tolerance before pregnancy may develop GDM when their insulin production becomes insufficient to maintain euglycemia. In the United States, nearly 60% of women of childbearing age (20–39 years) are overweight, one-third are obese, and 16% have metabolic syndrome. Moreover, recent studies found that 1.1% of US pregnancies were complicated by chronic hypertension, and in California, 1.3% of pregnant women had T2D and another 7.4% had GDM. To date, no studies involving humans have examined the relationship between these metabolic conditions (MCs) collectively and developmental outcomes in children. The aims of this study were to describe the prevalence of diabetes (T2D/GDM), hypertension, and obesity during the index pregnancy in mothers of children with ASD, DD, and typical development (TD) and to investigate whether these conditions were associated with impairments in specific developmental domains in the offspring.

**METHODS**

**Study Population**

This study was conducted by using data from the CHARGE (Childhood Autism Risks from Genetics and the Environment) study, an ongoing, population-based, case-control study. Participants were selected from 3 strata: ASD, DD without ASD, and general population (GP). Eligible children were between the ages of 24 and 80 months, born in California, living with at least 1 biological parent who spoke English or Spanish, and residing in the catchment areas of a specified list of regional centers in California. Children with major motor and sensory impairments (eg, blindness, deafness) that would preclude a valid developmental assessment were excluded. No other exclusions were made on the basis of genetics or family phenotype. Children with ASD or DD were identified through regional centers, providers/clinics, self-referrals, and general public outreach. GP children were identified from state birth files, and a stratified random sample was generated by frequency-matching to a projected distribution of ASD cases on age, gender, and regional center catchment area. Children with DD were not frequency-matched to either group. The CHARGE study protocol was approved by institutional review boards of the University of California in Davis and Los Angeles and the State of California Committee for the Protection of Human Subjects. Written informed consent was obtained before participation.

**Diagnostic Validation**

All children referred for the study with a diagnosis of autism/ASD were reevaluated with the Autism Diagnostic Interview, Revised (ADI-R), and the Autism Diagnostic Observation Schedule (ADOS) by trained clinicians at the UC Davis MIND (Medical Investigation of Neurodevelopmental Disorders) Institute to confirm the diagnosis by using criteria described by Risi et al. The Social Communication Questionnaire, designed to screen for ASD, was administered to parents of DD and GP children; children with scores above the ASD cutoff (≥15) were assessed with the ADOS and ADI-R, and reclassified to ASD if criteria were satisfied.

Mullen Scales of Early Learning (MSEL) and Vineland Adaptive Behavior Scales (VABS) were administered to all children to determine cognitive and adaptive development, respectively, and diagnostic groups for children without ASD were defined on the basis of these assessments. The DD group consisted
of children with composite scores <70 on the MSEL and/or VABS. The TD group only included GP children with no previous diagnosis of ASD or DD, Social Communication Questionnaire score <15, and composite scores ≥70 on both the MSEL and VABS. All CHARGE study clinical assessment personnel had attained research reliability on the developmental assessments they administered (ADI-R, ADOS, MSEL, and VABS). Bilingual study staff were available to administer informed consent and all instruments/questionnaires in Spanish.

**Maternal Conditions and Potential Confounders**

Demographic and medical information was obtained from the CHARGE Environmental Exposure Questionnaire (EEQ; available for 97.6% of participants), birth files, and medical records (available for 57.7% of participants). The EEQ is a structured telephone-administered interview with the biological mother and includes questions about demographic characteristics, maternal medical history, and various environmental exposures. Trained study staff extracted data from medical records.

The primary MC of interest was maternal T2D or GDM in the index pregnancy. Other conditions of interest were hypertension and obesity, defined as BMI ≥30, with onset before the index pregnancy. BMI (kilograms per meter squared) was calculated by using the height and pre-pregnancy weight recorded in the medical records (when available) or from the EEQ. BMI obtained from self-reported measurements was validated against BMI calculated from medical record measurements in a subset of 346 (34.5%) women for whom both data sources were available. The intraclass correlation coefficient between these BMI calculations was 0.912, indicating strong agreement.

Diabetes and hypertension (with or without preeclampsia) were considered present if they were noted on the medical history form in the prenatal medical record (when available) or if mothers answered “yes” to “During this [index] pregnancy were you ever told by a physician or nurse that you had gestational diabetes?” or “At any time before you became pregnant with [index child], were you ever told by a doctor that you had [diabetes, high blood pressure]?” in the EEQ. Discrepancies between medical records and self-report were verified. Self-reported diabetes and hypertension were validated in a subset of 560 (55.8%) women. Agreement between self-report and medical records was excellent for diabetes (κ = 0.79) and fair for hypertension (κ = 0.38). Women who misrepresented having hypertension were more likely to be multiparous and to have a history of gestationally induced hypertension. Because obesity, hypertension, and diabetes (T2D/GDM) are closely linked clinical manifestations of insulin resistance, an “any metabolic condition” variable was created as another proxy measure of insulin resistance. For all analyses, these predictor variables were categorized as follows: (1) had condition of interest; (2) did not have condition of interest but had another MC or was overweight (BMI ≥25); and (3) did not have any MCs and had a BMI <25. For each MC variable, we present results for level 1 versus level 3 to maximize the contrast and simplify the interpretation of findings.

Covariates selected a priori included mother’s age at delivery, race/ethnicity (non-Hispanic white, other non-Hispanic, or Hispanic), education level (high school or less, some college, or bachelor degree or higher), delivery payer (government program or private insurance), calendar time defined as number of years from the first participant’s enrollment date, the child’s gender and age in years at study enrollment, and catchment area; the latter 3 factors were frequency-matching variables. An indicator for known chromosomal/genetic (eg, trisomy 21, Angelman syndrome), metabolic/mitochondrial (eg, Leigh syndrome, carnitine deficiency), or neurolologic (eg, cerebral palsy, epilepsy, hydrocephalus) disorders was created by using parent-report data from the child’s medical history completed by a study physician.

For this study, 1004 children (517 ASD, 172 DD, and 315 TD), consisting of 964 singletons and 20 sibling pairs, were included from a pool of 1317 participants who completed a clinic visit between January 2003 and June 2010. Excluded were 245 children who either did not complete the necessary assessments to confirm diagnosis or did not satisfy the diagnostic criteria for the groups considered in this study, and 68 children for whom data regarding maternal diabetes, hypertension, and obesity were unavailable.

**Statistical Analyses**

All analyses were performed by using SAS version 9.2 (SAS Institute Inc, Cary, NC). A directed acyclic graph35 (Fig 1) was constructed to represent the underlying causal relationships and used as guidance in evaluating associations among predictors, covariates, and outcomes. All variables with arrows pointing to the predictor (obesity, hypertension, or T2D/GDM) and the outcome (neurodevelopmental disorder) were defined as potential confounders and evaluated further. To determine whether MCs during pregnancy were associated with an increased risk of having a child with ASD or DD relative to TD, 4 multinomial logistic regression models were fitted and corrected for family clusters, 1 for each condition and 1 for the “any metabolic condition” predictor. Odds ratios (ORs) and 95% confidence intervals (CIs) were used as
estimates of relative risk. Final models were adjusted for mother’s age at delivery, race/ethnicity, education, payer, calendar time, child’s age and gender, and catchment area.

To evaluate children’s developmental scores in association with maternal MCs, linear regression models corrected for family clusters were fitted. Age-standardized scores from MSEL visual reception, fine motor, receptive language, and expressive language subscales (mean ± SD: 50 ± 10) and from VABS communication, socialization, and motor skills domains (mean ± SD: 100 ± 15) were examined; MSEL and VABS composite scores (mean ± SD: 100 ± 15) were also considered. Covariates in these models included mother’s age, race/ethnicity, education, payer, calendar time, child’s age and gender, and catchment area. These models compared mean assessment scores of children whose mothers had an MC of interest (diabetes, hypertension, obesity, or any MC) versus those whose mothers did not have any of these MCs and had a BMI < 25; comparisons were done separately among children with and those without ASD. Least squares (LS) means and SEs were used to measure association between MCs and developmental scores. The developmental domains examined were selected a priori because of their biological relevance to MCs, and adjustment for multiple comparisons was not performed. In the non-ASD subset, DD and TD groups were combined because the mean differences in developmental scores of children born to mothers with MCs compared with no MCs were similar in magnitude between these diagnostic groups. In addition, the combined sample provided more power to detect an association, through not only a larger sample but also the full range of variation in cognitive and adaptive scores.

RESULTS

Mothers of children with ASD were similar to controls in terms of race/ethnicity, education, and delivery payer (Table 1). Mothers of DD children were more likely to be Hispanic and to have lower education and less likely to have had private insurance compared with controls. Multiparas in both case groups were more likely to have a history of GDM compared with controls. As expected, because of frequency-matching, the ASD and TD groups were similar with respect to child’s age and gender; the DD group, which was not matched, had a higher proportion of girls compared with the other 2 groups. Discrepancies between the ASD and TD groups regarding the Los Angeles catchment area arose because of delayed implementation of the recruitment protocol for controls in the initial year of the study at the Los Angeles site, leading to some imbalance in the case-control ratio.

The proportions of T2D/GDM in the ASD (9.3%) and DD (11.6%) groups were higher compared with controls (6.4%); after adjustment for covariates, mothers with diabetes were 2.3 times more likely to have a child with DD (OR: 2.33 [95% CI: 1.08–5.05]), but the association between diabetes and ASD did not reach statistical significance (Table 2).

FIGURE 1

Directed acyclic graph. All relationships represented in this graph were based on review of the literature, with the exception of the frequency-matching variables (child’s age and gender, and catchment area). Solid arrows indicate stronger associations, and dashed arrows denote weaker associations.
to TD, was significantly increased among obese women (ASD, OR: 1.67 [95% CI: 1.10–2.56]; DD, OR: 2.08 [95% CI: 1.20–3.61]); >20% of case mothers were obese compared with 14.3% of controls. The prevalence of any MC was higher in the ASD (28.6%) and DD (34.9%) groups compared with controls (19.4%), with respective adjusted ORs of 1.61 (95% CI: 1.10–2.37) and 2.35 (95% CI: 1.43–3.88). Analyses restricted to children without known genetic, metabolic, or neurologic disorders conferred similar or slightly stronger associations (data not shown).

Within the ASD group, children of mothers with diabetes performed 0.37 SD lower on the MSEL expressive language scale compared with children of nondiabetic mothers (P = .01; Table 3); MSEL receptive language and VABS communication scores were also lower among children of diabetic mothers, with differences approaching statistical significance. No significant differences in MSEL or VABS scores were observed regarding MCs collectively among children with ASD.

Among children without ASD, MSEL receptive and expressive language scores were ~0.5 SD lower among children of mothers with diabetes compared with nondiabetic mothers (P = .03 for both subscales; Table 4); MSEL composite scores were borderline lower among children of mothers with diabetes. VABS socialization scores were 0.49 SD lower among children from diabetic pregnancies (P = .01). The presence of any MC was associated with lower scores on all MSEL subscales and composite (visual: −0.51 SD [P = .01]; motor: −0.53 SD [P = .01]; receptive: −0.50 SD [P = .004]; expressive: −0.58 SD [P = .0004]; composite: −0.65 SD [P = .001]) and all VABS domains and composite (communication: −0.43 SD [P = .01]; socialization: −0.50 SD [P = .0004]; motor: −0.39 SD [P = .004]; composite: −0.51 SD [P = .005]). Findings from restricted analyses were nearly identical (data not shown).

**DISCUSSION**

In this study, we observed that diabetes, hypertension, and obesity were more common among mothers of children with ASD and DD compared with controls. Furthermore, diabetes, in particular, was associated with statistically significantly greater deficits in expressive language among children with ASD, although the magnitude of the deficits was relatively small. Among children without ASD, MCs collectively were
this group included 267 in ASD, 64 in DD, and 172 in TD groups. VABS ASD and 236 in non-ASD strata.

and gender, and catchment area. Comparison group had no hypertension or diabetes (T2D or GDM) and also had BMI

Diabetes 48 9.3 20 11.6 20 6.4 1.52 0.82–2.83 2.33 1.08–5.05
Hypertension 19 3.7 6 3.5 4 1.3 2.64 0.84–8.56 3.58 0.93–13.78
Obesity 111 21.5 41 23.8 45 14.3 1.67 1.10–2.56 2.08 1.20–3.61
Any MC(a) 148 28.8 60 34.9 61 19.4 1.61 1.10–2.37 2.35 1.43–3.88

* Adjusted for mother’s age at delivery, race/ethnicity, education level, delivery payer, calendar time, child’s age at enrollment and gender, and catchment area. Comparison group had no hypertension or diabetes (T2D or GDM) and also had BMI <25; this group included 267 in ASD, 64 in DD, and 172 in TD groups.

b T2D or GDM only.

TABLE 2 OR for Autism/ASD or Other Delays in Relation to Diabetes and Related Conditions: CHARGE Study, January 2003–June 2010 (N = 1004)

<table>
<thead>
<tr>
<th>Conditions in Index</th>
<th>ASD</th>
<th>DD</th>
<th>TD</th>
<th>ASD Versus TD</th>
<th>DD Versus TD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pregnancy</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Diabetes(b)</td>
<td>48</td>
<td>9.3</td>
<td>20</td>
<td>11.6</td>
<td>20 6.4</td>
</tr>
<tr>
<td>Any MC(a)</td>
<td>148</td>
<td>28.8</td>
<td>60</td>
<td>34.9</td>
<td>61 19.4</td>
</tr>
</tbody>
</table>

TABLE 3 Assessment Scores of Children of Mothers With and Without Diabetes (T2D or GDM), a Stratified According to ASD Status

<table>
<thead>
<tr>
<th>Assessment</th>
<th>ASD (n = 315)</th>
<th>No Conditions</th>
<th>P&lt; b</th>
<th>ASD (n = 276)</th>
<th>No Conditions</th>
<th>P&lt; b</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Diabetes</td>
<td>No Conditions</td>
<td></td>
<td>Diabetes</td>
<td>No Conditions</td>
<td></td>
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<td></td>
<td>LS Mean</td>
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<td>LS Mean</td>
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<tr>
<td>VABS</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Communication standard score</td>
<td>61.74</td>
<td>2.19</td>
<td>66.07</td>
<td>1.24</td>
<td>87.08</td>
<td>3.25</td>
</tr>
<tr>
<td>Socialization standard score</td>
<td>64.43</td>
<td>1.62</td>
<td>66.82</td>
<td>1.04</td>
<td>87.84</td>
<td>2.70</td>
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<tr>
<td>Motor skills standard score</td>
<td>74.86</td>
<td>2.57</td>
<td>74.37</td>
<td>1.57</td>
<td>88.50</td>
<td>4.00</td>
</tr>
<tr>
<td>Composite standard score</td>
<td>60.82</td>
<td>1.95</td>
<td>62.89</td>
<td>1.21</td>
<td>85.36</td>
<td>3.68</td>
</tr>
</tbody>
</table>

a Diabetes group includes mothers with T2D or GDM; no conditions group consists of mothers with no diabetes (T2D or GDM) or hypertension and who have BMI <25; this comparison group consisted of 267 in ASD and 236 in non-ASD strata.

b Adjusted for mother’s age at delivery, race/ethnicity, education level, delivery payer, calendar time, child’s age at enrollment and gender, and catchment area.

TABLE 4 Assessment Scores of Children of Mothers With and Without any MCs, a Stratified According to ASD Status

<table>
<thead>
<tr>
<th>Assessment</th>
<th>ASD (n = 315)</th>
<th>No Conditions</th>
<th>P&lt; b</th>
<th>ASD (n = 276)</th>
<th>No Conditions</th>
<th>P&lt; b</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Any MC</td>
<td>No Conditions</td>
<td></td>
<td>Any MC</td>
<td>No Conditions</td>
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<td></td>
<td>LS Mean</td>
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<td>VABS</td>
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<td></td>
</tr>
<tr>
<td>Communication standard score</td>
<td>66.71</td>
<td>1.47</td>
<td>68.64</td>
<td>1.23</td>
<td>85.87</td>
<td>2.11</td>
</tr>
<tr>
<td>Socialization standard score</td>
<td>67.16</td>
<td>1.32</td>
<td>68.50</td>
<td>1.04</td>
<td>87.63</td>
<td>1.78</td>
</tr>
<tr>
<td>Motor skills standard score</td>
<td>75.00</td>
<td>1.83</td>
<td>74.35</td>
<td>1.57</td>
<td>86.15</td>
<td>2.51</td>
</tr>
<tr>
<td>Composite standard score</td>
<td>62.62</td>
<td>1.55</td>
<td>62.87</td>
<td>1.20</td>
<td>84.09</td>
<td>3.23</td>
</tr>
</tbody>
</table>

a Any MCs group includes mothers with T2D or GDM, hypertension, or a BMI ≥30; no conditions group consists of mothers with no diabetes (T2D or GDM) or hypertension and who have a BMI <25; this comparison group consisted of 267 in ASD and 236 in non-ASD strata.

b Adjusted for mother’s age at delivery, race/ethnicity, education level, delivery payer, calendar time, child’s age at enrollment and gender, and catchment area.

Our findings relating to impairments in cognitive and language development are consistent with some13,15,16 but not all previous studies.17,18 Hultman et al17 included numerous maternal and pregnancy characteristics in their multivariable model; as such, the temporal and causal interrelationships among these risk factors were effectively ignored. Hence, it is plausible that the association with ASD may have been attenuated as a consequence of including complications downstream of diabetes. Furthermore, although Dodds et al18 reported higher proportions of diabetes (preexisting and GDM) among mothers of ASD children compared with controls in unadjusted analyses that only nearied statistical significance, key confounders were not considered. Interestingly, prepregnancy obesity (≥90 kg) and excessive weight gain (≥18 kg) during pregnancy were significantly associated with ASD; however, the final model also included intermediary maternal characteristics (eg, labor type) potentially on the causal pathway between these risk factors and the outcome, thus limiting the interpretability of these findings. Nevertheless, obesity is a significant risk factor for both hypertension and diabetes (T2D and GDM) and is characterized by increased insulin resistance and chronic inflammation, as are the other 2 MCs we examined.19,20,22 Therefore, in our study, we constructed a causal diagram to investigate the interrelationships among maternal MCs, covariates, and the outcome. We also compared the risk of an adverse outcome in children whose mothers had a given condition (eg, diabetes) relative to those whose mothers had neither condition nor the risk factors for it (eg, no hypertension and with BMI associated with impairments in visual reception, motor skills, and receptive and expressive language, as well as adaptive communication and socialization.

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previously published population-based studies investigating maternal risk factors and ASD.16–18 Secondly, whereas previous studies have examined maternal T2D or GDM in relation to neurodevelopmental disorders or developmental measures, to our knowledge, this is the first to also examine a broader group of conditions highly predictive of insulin resistance and these forms of diabetes. In a diabetic and possibly prediabetic pregnancy, poorly regulated maternal glucose can result in adverse fetal development. Prolonged fetal exposure to elevated glucose levels results in chronic fetal hyperinsulinemia, which in turn triggers the fetus to increase oxygen consumption and metabolism, inducing chronic intrauterine tissue hypoxia.37 Further biological responses may result in fetal iron deficiency.38 Both fetal hypoxia and iron deficiency can profoundly affect neurodevelopment in humans, including alterations in myelination and cortical connectivity and aberrations in hippocampal neurons.39 Fetal iron deficiency has also been associated with reduced recognition memory performance in infants of diabetic mothers at 1 year.40

CONCLUSIONS

The prevalence of obesity and diabetes among US women of childbearing age is 34% and 8.7%, respectively.24,27 Our findings raise concerns that these maternal conditions may be associated with neurodevelopmental problems in children and therefore could have serious public health implications.

REFERENCES


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DOI: 10.1542/peds.2011-2583

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