

studies, subgrouping autism cases on the basis of CDDs may enhance our knowledge of etiological pathways in autism.

#### 02-6.4 IMPACT OF MATERNAL OBESITY ON STILLBIRTH AND INFANT DEATH: ABSOLUTE RISK AND TEMPORAL TRENDS

doi:10.1136/jech.2011.142976a.79

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**Introduction** UK guidelines advocate that obese pregnant women (body mass index, BMI  $\geq 30$  kg/m<sup>2</sup>) be made aware of the increased risks to them and their offspring. This study hence pooled data from several sources to derive estimates of the absolute and attributable risks of stillbirth and infant death for obese women in England, and predict changes in prevalence resulting from trends in BMI.

**Methods** The BMI profile of the maternal population of England and of the prevalence of each outcome were obtained from nationally representative sources. Trends in BMI were modelled by logistic regression. RRs for stillbirth and infant death were derived from published literature. These were equated to estimate absolute risks, attributable risks, and future prevalence rates.

**Results** The estimated absolute risk of a stillbirth or infant death for an obese pregnant woman in England is 1.5% (95% CI 1.3 to 1.8), compared to 0.9% (0.8 to 0.9) for women of recommended BMI (25–29 kg/m<sup>2</sup>). An estimated 8.1% of stillbirths and infant deaths in England are attributable to maternal obesity.

If trends in maternal BMI continue, 24.0% (22.1 to 25.9) of the maternal population of England will be obese by 2020. This is predicted to result in a 4.4% increase in the prevalence of stillbirth and infant death compared to 2010.

**Conclusion** This study provides estimates of the individual risk and population burden of stillbirth and infant death in England resulting from maternal obesity. These results have implications for public health planning and for providing clear information to obese women about their pregnancy-related risks.

#### 02-6.5 RISK OF FETAL DEATH IN WOMEN WITH PERICONCEPTIONAL INTAKE OF MULTIVITAMINS

doi:10.1136/jech.2011.142976a.80

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**Introduction** Nutrition is important in a healthy pregnancy, but little is known about the impact of multivitamins (MV) on the survival of the fetus.

**Methods** We related periconceptional MV use to early (<20 weeks) and late ( $\geq 20$  weeks) fetal death. At recruitment, women in the Danish National Birth Cohort (n=35 897) reported the number of weeks of MV use during a 12 week periconceptional period. Information about lifestyle factors came from a later telephone interview. Cox regression was used to estimate HR for the association between MV use and fetal death with follow-up starting at 8 completed weeks of gestation. Intensity of preconception use (6 weeks before conception) and postconception use (6 weeks after conception) were categorised as use in 1–2, 3–4, and 5–6 weeks. No use at any time of these weeks was reference.

**Results** Compared to women with <2 weeks of preconception MV use, risk of early fetal death increased with increasing intensity of

#### 02-6.3 THE RISK FOR AUTISM AND FOR AUTISM WITH CO-EXISTING DEVELOPMENTAL DISABILITIES IN LOW BIRTH WEIGHT CHILDREN COMPARED TO NORMAL BIRTH WEIGHT CHILDREN

doi:10.1136/jech.2011.142976a.78

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**Introduction** Recently, the autism age-specific prevalence has increased dramatically and autism prevalence estimates now range from 60 to 70/10 000. This makes autism the second most common neurodevelopmental disorder after mental retardation (MR) and a serious public health concern.

Autism is a highly heritable multifactorial disorder, but the specific etiological pathways are largely unknown. Previous studies reported ORs from 1.9 to 2.3 for autism in low birth weight children (lbw, <2500 g) when compared to normal birth weight children (nbw, 3000–3999 g), but most studies have ignored the possible impact of comorbidities. Co-existing developmental disabilities (CDDs) like MR, ADHD, and epilepsy are common in autism.

We hypothesise that lbw is a stronger risk factor for autism with CDDs than for autism without CDDs. We report HR (95% CI) for autism + CDDs and autism / noCDDs specifically in lbw children when compared to nbw children.

**Methods** We conducted a nationwide, register-based, follow-up study. 1990 to 2007 birth cohorts were identified in The Danish Medical Birth Register and followed through 2009. Linkage with national hospital registers made exposure, outcome, and covariates information available to us.

**Results** We found HR 2.0 (1.5 to 2.8) for autism + MR and HR 0.9 (0.7 to 1.1) for autism/no MR in lbw children when compared to nbw children. Analyses of more CDD subgroups are ongoing.

**Conclusion** Consistent with a recently published study we find that lbw children have a twofold increased risk for autism with MR but no increased risk for autism with normal intelligence. In etiological