Gestational Diabetes:
The importance of making the diagnosis for mother and child.

Sara J Meltzer
McGill University
Lima, Peru
September 8th, 2015
Objectives of the Presentation

- Understand the controversies surrounding the diagnosis of GDM comparing 100g/75g criteria
- Understand the short and long-term risks for mothers
- Understand the short and long-term risks for the offspring...
Why Diagnose and Treat GDM?

Maternal concerns:
- Preterm delivery
- Traumatic delivery due to macrosomia
- Caesarian section risk
- Pre-eclampsia risk increased
- Future T2DM and CV risks increased

Fetal / Offspring concerns:
- Macrosomia
- Shoulder dystocia and nerve injury
- Hyperbilirubinemia
- Neonatal hypoglycemia
- Offspring obesity (?)
- Offspring diabetes (?)
Benefits of Treatment of GDM

Horvath K et al. BMJ 2010;340:c1935

Large for gestational age

<table>
<thead>
<tr>
<th></th>
<th>Intervention</th>
<th>Control</th>
<th>Odds ratio (95% CI)</th>
<th>Weight (%)</th>
<th>Odds ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bonomo 2005</td>
<td>9/150</td>
<td>21/150</td>
<td></td>
<td>8.8</td>
<td>0.39 (0.17 to 0.89)</td>
</tr>
<tr>
<td>Crowther 2005</td>
<td>68/506</td>
<td>115/524</td>
<td></td>
<td>0.70</td>
<td>0.70</td>
</tr>
<tr>
<td>Landon 2009</td>
<td>34/477</td>
<td>66/454</td>
<td></td>
<td>0.70</td>
<td>0.70</td>
</tr>
<tr>
<td>Langer 1989</td>
<td>4/63</td>
<td>15/63</td>
<td></td>
<td>0.70</td>
<td>0.70</td>
</tr>
<tr>
<td>Total</td>
<td>115/1196</td>
<td>217/1191</td>
<td></td>
<td>100.0</td>
<td>0.48 (0.38 to 0.62)</td>
</tr>
</tbody>
</table>

Test for heterogeneity: $\chi^2=2.79$, df=3, $P=0.425$, $I^2=0\%$
Test for overall effect: $z=-5.85$, $P<0.001$, $\tau=0$

Macrosomia

<table>
<thead>
<tr>
<th></th>
<th>Intervention</th>
<th>Control</th>
<th>Odds ratio (95% CI)</th>
<th>Weight (%)</th>
<th>Odds ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bonomo 2005</td>
<td>8/150</td>
<td>16/150</td>
<td></td>
<td>8.1</td>
<td>0.47 (0.20 to 1.14)</td>
</tr>
<tr>
<td>Crowther 2005</td>
<td>49/506</td>
<td>110/524</td>
<td></td>
<td>47.8</td>
<td>0.40 (0.28 to 0.58)</td>
</tr>
<tr>
<td>Landon 2009</td>
<td>28/477</td>
<td>65/454</td>
<td></td>
<td>29.2</td>
<td>0.37 (0.23 to 0.59)</td>
</tr>
<tr>
<td>O’Sullivan 1966</td>
<td>13/307</td>
<td>40/308</td>
<td></td>
<td>15.0</td>
<td>0.30 (0.16 to 0.57)</td>
</tr>
<tr>
<td>Total</td>
<td>98/1440</td>
<td>231/1436</td>
<td></td>
<td>100.0</td>
<td>0.38 (0.30 to 0.49)</td>
</tr>
</tbody>
</table>

Test for heterogeneity: $\chi^2=0.91$, df=3, $P=0.823$, $I^2=0\%$
Test for overall effect: $z=-7.55$, $P<0.001$, $\tau=0$

Shoulder Dystocia

<table>
<thead>
<tr>
<th></th>
<th>Intervention</th>
<th>Control</th>
<th>Odds ratio (95% CI)</th>
<th>Weight (%)</th>
<th>Odds ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crowther 2005</td>
<td>7/506</td>
<td>16/524</td>
<td></td>
<td>49.2</td>
<td>0.45 (0.18 to 1.09)</td>
</tr>
<tr>
<td>Landon 2009</td>
<td>7/476</td>
<td>18/455</td>
<td></td>
<td>50.8</td>
<td>0.36 (0.15 to 0.88)</td>
</tr>
<tr>
<td>Total</td>
<td>14/982</td>
<td>34/979</td>
<td></td>
<td>100.0</td>
<td>0.40 (0.21 to 0.75)</td>
</tr>
</tbody>
</table>

Test for heterogeneity: $\chi^2=0.10$, df=1, $P=0.748$, $I^2=0\%$
Test for overall effect: $z=-2.85$, $P=0.004$, $\tau=0$
O’Sullivan & Mahan (1964) original diagnostic criteria defining GDM

Original normative data from 752 women in NY:

- Threshold values were 2 SD above the mean using whole blood glucose
- Reapplied thresholds retrospectively to a different group of 1013 subjects tested in pregnancy & followed for 5 - 10 years PP
- Diabetes developed over 7 – 8 years in 22 % (17 women) in whom 2 glucose values were ≥ 2SD above the mean.
- Criteria were accepted as assessing risk for future maternal diabetes

O’Sullivan and Mahan, 1964
An Overview of some of the different diagnostic criteria for GDM

<table>
<thead>
<tr>
<th>Plasma glucose</th>
<th>NDDG 3h 100g OGTT 1979</th>
<th>Carpenter &amp; Coustan 3h 100g OGTT</th>
<th>WHO/ IADPSG 75g OGTT</th>
<th>WHO 1999 75g OGTT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fasting</td>
<td>≥ 5.8 (105)</td>
<td>≥ 5.3 (95)</td>
<td>≥ 5.1 (92)</td>
<td>≥ 6.1 (110)</td>
</tr>
<tr>
<td>1h</td>
<td>≥ 10.6 (190)</td>
<td>≥ 10.0 (180)</td>
<td>≥ 10.0 (180)</td>
<td>-</td>
</tr>
<tr>
<td>2h</td>
<td>≥ 9.2 (165)</td>
<td>≥ 8.6 (155)</td>
<td>≥ 8.5 (153)</td>
<td>≥ 7.8 (140)</td>
</tr>
<tr>
<td>3h</td>
<td>≥ 8.0 (145)</td>
<td>≥ 7.8 (140)</td>
<td>N/A</td>
<td></td>
</tr>
</tbody>
</table>

Number of abnormal values needed for diagnosis:
- ≥ 2

O'Sullivan & Mahan numbers converted to plasma glucose and rounded up and down for ease of remembering.

Don Coustan & Marshall Carpenter recalculated more precisely the O'Sullivan & Mahan numbers and did not round them...

Acknowledged that values also indicated fetal and maternal risks in pregnancy, not only that of future maternal diabetes.

Based on HAPO OR of 1.75

Based on consensus

Values are presented in mmol/l. NDDG: National Diabetes Data Group; OGTT: oral glucose tolerance test; IADPSG: The International Association of Diabetes and Pregnancy Study Groups.
HAPO: Incidence of Adverse Outcomes for Glucose Categories (OR 1.75 or 2.0)

DM 2 is increasing in the general population – why not in pregnancy?

- SEARCH for Diabetes Study indicated a 30.5% (95% CI, 17.3%-45.1%) overall increase in type 2 diabetes between 2000 and 2009 in US in children and adolescents.

doi:10.7326/M13-2411
McGill GDM Diagnosis Study –
a prospective randomized controlled trial involving
5481 multiethnic, pregnant women in Montréal
using a 75g vs 100 g OGTT

S. Meltzer, J. Snyder, L. Morin, M. Nudi, MSc

- Comparison of NDDG criteria for diagnosis and outcome
  with Canadian criteria using a 75g 2hOGTT either with or
  without glucose screen

- Canadian values of interest because they are virtually
  equivalent to an OR of 2.0 from HAPO study

Use of McGill trial to assess impact on a multiethnic, North
America population to assess prevalence and potential
outcomes

Funded by the Canadian Diabetes Association

October 2009
Study Design

Randomization
(83% recruitment rate)

Visit 1:
Group 1
n = 1813
1h 50g GS
if Glucose Screen (GS) 7.8 - 10.2
Group 2
n = 1839
1h 50g GS
2h 75g OGTT
Group 3
n = 1836
2h 75g OGTT

Visit 2:
20.4%
3h 100g OGTT
A total of 5637 women recruited; 148 withdrawn (vomited the glucose drink, did not comply with study design or had incomplete data sets) and data analysis performed on 5481 women
3 day diet provided
21.9%
2h 75g OGTT
Normative ethnic values developed from this population
131/3753 (3.5%) missed second test or did wrong one

A total of 5637 women recruited; 148 withdrawn (vomited the glucose drink, did not comply with study design or had incomplete data sets) and data analysis performed on 5481 women
# Evaluation of cost of screening methods between 1 and 2 steps

<table>
<thead>
<tr>
<th>Cost components</th>
<th>GR1 50gGS +100gGTT</th>
<th>GR2 50gGS + 75gGTT</th>
<th>GR3 Only 75gGTT</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Direct costs</strong> (Drink, blood test costs-$CAN)</td>
<td>21.77</td>
<td>20.16</td>
<td>36.89</td>
</tr>
<tr>
<td>Mean time in clinic (hrs)</td>
<td>3.48</td>
<td>3.24</td>
<td>3.79</td>
</tr>
<tr>
<td>Transportation costs</td>
<td>14.66</td>
<td>15.32</td>
<td>11.92</td>
</tr>
<tr>
<td>Time costs</td>
<td>55.18</td>
<td>53.56</td>
<td>59.57</td>
</tr>
<tr>
<td><strong>Direct + Indirect costs ($CAN) per women screened</strong></td>
<td>91.61</td>
<td>89.03</td>
<td>108.38</td>
</tr>
<tr>
<td><strong>Average cost per case diagnosed</strong></td>
<td>1145.13</td>
<td>1112.88</td>
<td>1354.75</td>
</tr>
<tr>
<td><strong>Avg. cost per South Asian woman screened</strong></td>
<td>95.87</td>
<td>105.24</td>
<td>104.15</td>
</tr>
</tbody>
</table>

GDM diagnostic rate = 24%

Meltzer et al, BJOG 2010

*Least expensive is GS (Dx 10.3) + 75g GTT if needed*

*Except for highest risk group*
Prevalence (%) of GDM and IGT

### ADA CC 100 g 3h test (G1) vs IADPSG 75g 2h test (GR 2&3)

<table>
<thead>
<tr>
<th>Study Group, (n=)</th>
<th>GDM by GS</th>
<th>GDM by GTT</th>
<th>Total GDM</th>
<th>IGT by GTT</th>
<th>GDM and IGT</th>
</tr>
</thead>
<tbody>
<tr>
<td>CC 100g (1812)</td>
<td>2.2</td>
<td>3.9</td>
<td>6.1</td>
<td>5.2</td>
<td>11.3</td>
</tr>
<tr>
<td>GR2 (1839)</td>
<td>2.6</td>
<td>2.6*</td>
<td>5.2</td>
<td>5.0</td>
<td>10.2</td>
</tr>
<tr>
<td>GR3 (1838)</td>
<td>N/A</td>
<td>5.1</td>
<td>5.1</td>
<td>6.0</td>
<td>11.1</td>
</tr>
</tbody>
</table>

* Difference with GR1 gold standard is significant (p<0.05)

The 75 g GTT IADSPG criteria with or without GS give similar diagnostic rates of GDM/GIGT compared to ADA CC 100g GTT criteria (2014 ACOG)

Less time for the woman; easier test to tolerate
## Odds Ratios of Outcomes by group vs NDDG

Corrected for age, weight, BMI, ethnicity

<table>
<thead>
<tr>
<th>Comparison parameter</th>
<th>OR for all women</th>
<th>OR for normal women</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group 2</td>
<td>Group 3</td>
</tr>
<tr>
<td>Unplanned C/S</td>
<td>1.43</td>
<td>1.35</td>
</tr>
<tr>
<td></td>
<td>[1.04-1.89]</td>
<td>[1.05-1.80]</td>
</tr>
<tr>
<td>Pre-eclampsia</td>
<td>1.15</td>
<td>1.33</td>
</tr>
<tr>
<td></td>
<td>[0.76-1.74]</td>
<td>[0.89-1.97]</td>
</tr>
<tr>
<td>Neonatal hypoglycemia (&lt;2.2mmol/L)</td>
<td>1.40</td>
<td>1.21</td>
</tr>
<tr>
<td></td>
<td>[0.98-2.17]</td>
<td>[0.78-1.87]</td>
</tr>
<tr>
<td>NICU admission</td>
<td>1.22</td>
<td>1.15</td>
</tr>
<tr>
<td></td>
<td>[0.89-1.68]</td>
<td>[0.84-1.58]</td>
</tr>
</tbody>
</table>

Only unplanned C/S and pre-eclampsia in “normal” untreated women are significant but all trends suggest poorer outcomes with CDA criteria
Comments on “Considerations”

- Do we have sufficient evidence with respect to treatment benefit at the various thresholds to make an informed decision....
  - The ACHOIS data and the majority of the meta-analysis data was made based on old WHO criteria... the **2h value** was what diagnosed almost all of them and it was lower than both the OR for 1.75 (8.5) and 2 (9.0)... *it was 7.8mmol/L!*
  - Thus the present historical outcome data suggests even lower cutoff values would be justified...

- What is the **LONG TERM economic cost of NOT treating**, thus not recognizing women and offspring with elevated risks?
  - Prevention of DM early rather than late is a justifiable cost
  - Prevention of obesity and potentially adolescent diabetes in the offspring also would justify costs?
The cost-effectiveness of gestational diabetes screening including prevention of type 2 diabetes: application of a new model in India and Israel

- WHO has proposed that interventions costing less than the per capita GDP of a country be deemed “highly cost-effective”, and those costing up to three times per-capita GDP “cost-effective” [37].

- Screening and treating gestational diabetes, considering adverse perinatal events and future diabetes, has an incremental cost-effectiveness of $1626 per Diability Adjusted Life Years (DALY) averted for a general hospital in India, and $1830 per DALY averted for an HMO in Israel.

- Since the 2010 per-capita GDP of India and Israel are $3500 and $29 800 [38], respectively, the interventions are “highly cost-effective”.

Marseille et al, J. Matern Fetal Neonatal Med 2013: 26(8) p 802
A question of balance…

Poorer pregnancy outcomes
Obese mothers with diabetes
Obese offspring? Future patients?
Overall increased societal costs?

An overloaded medical system
Harried doctors & nurses
Exorbitant initial treatment costs
What about after the baby is born – for the Mom?

Understand the controversies surrounding the diagnosis of GDM comparing 100g/75g criteria

Understand the short and long-term risks for mothers

Understand the short and long-term risks for the offspring...
The Incidence of Diabetes (and type 2 DM in pregnancy) is Increasing

Data from Centers for Disease Control and Prevention

Pre-existing DM2 in pregnancy
Kaiser-Permanente Data

Laurence DC 2009

Data from Centers for Disease Control and Prevention

~5.8m
~17m

293% increase in 26 yrs!

Cases of Diabetes (in millions)

1980 2006

1999 2000 2001 2002 2003 2004 2005

DM /100 births

p <0.0001

125% increase in 6 years

Laurence DC 2009

Cases of Diabetes in millions

1980 2006

1999 2000 2001 2002 2003 2004 2005

DM /100 births

1.82
0.81
0.5
0

1999 2000 2001 2002 2003 2004 2005

Laurence DC 2009

Cases of Diabetes in millions

1980 2006

1999 2000 2001 2002 2003 2004 2005

DM /100 births

1.82
0.81
0.5
0

1999 2000 2001 2002 2003 2004 2005

Laurence DC 2009

Cases of Diabetes in millions

1980 2006

1999 2000 2001 2002 2003 2004 2005

DM /100 births

1.82
0.81
0.5
0

1999 2000 2001 2002 2003 2004 2005

Laurence DC 2009
Overall prevalence of Type 2 DM post GDM

<table>
<thead>
<tr>
<th>Country</th>
<th>T2DM/GDM</th>
<th>T2DM/no GDM</th>
<th>Relative risk (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Madaras et al, 21 1995</td>
<td>21/68</td>
<td>0/39</td>
<td>24.93 (1.55-400.64)</td>
</tr>
<tr>
<td>Gunderson et al, 22 1985-2006</td>
<td>43/166</td>
<td>150/2242</td>
<td>3.87 (2.87-5.22)</td>
</tr>
<tr>
<td>Vambergue et al, 23 1992</td>
<td>53/295</td>
<td>1/111</td>
<td>19.94 (2.79-142.47)</td>
</tr>
<tr>
<td>Lee A et al, 24 1971-2003</td>
<td>405/5470</td>
<td>16/783</td>
<td>3.62 (2.21-5.93)</td>
</tr>
<tr>
<td>Ferraz et al 17*</td>
<td>6/70</td>
<td>7/108</td>
<td>1.32 (0.46-3.78)</td>
</tr>
<tr>
<td>Krishnaveni et al, 25 1997-98</td>
<td>13/35</td>
<td>8/489</td>
<td>22.70 (10.09-51.08)</td>
</tr>
<tr>
<td>Morimitsu et al, 26 1999-2001</td>
<td>7/23</td>
<td>0/11</td>
<td>7.50 (0.47-120.11)</td>
</tr>
<tr>
<td>Järvelä et al 5 1984-94</td>
<td>23/435</td>
<td>0/435</td>
<td>47.00 (2.86-771.65)</td>
</tr>
<tr>
<td>Albareda et al, 27 1966-93</td>
<td>44/696</td>
<td>0/70</td>
<td>9.07 (0.56-146.25)</td>
</tr>
<tr>
<td>Åberg et al, 28 1991-99</td>
<td>21/229</td>
<td>1/61</td>
<td>5.59 (0.77-40.66)</td>
</tr>
<tr>
<td>Linné et al, 16 1964-65</td>
<td>10/28</td>
<td>0/52</td>
<td>38.38 (2.33-631.74)</td>
</tr>
<tr>
<td>Bian et al, 29 1964-65</td>
<td>15/45</td>
<td>1/39</td>
<td>13.00 (1.80-93.93)</td>
</tr>
<tr>
<td>Ko et al, 30 1988-95</td>
<td>105/801</td>
<td>7/431</td>
<td>8.07 (3.79-17.19)</td>
</tr>
<tr>
<td>Osei et al, 31 1990-91</td>
<td>10/15</td>
<td>0/35</td>
<td>47.25 (2.95-757.28)</td>
</tr>
<tr>
<td>Damm et al, 32 1978-85</td>
<td>33/241</td>
<td>0/57</td>
<td>16.06 (1.00-258.06)</td>
</tr>
<tr>
<td>Benjamin et al, 33 1961-88</td>
<td>14/47</td>
<td>3/47</td>
<td>4.67 (1.43-15.21)</td>
</tr>
<tr>
<td>O’Sullivan, 34 1954-60 and 1962-70</td>
<td>224/615</td>
<td>18/328</td>
<td>6.64 (4.19-10.52)</td>
</tr>
<tr>
<td>Persson et al, 35 1961-84</td>
<td>5/145</td>
<td>0/41</td>
<td>3.16 (0.18-55.76)</td>
</tr>
</tbody>
</table>

Overall: 7.43

O’Sullivan 1954 to 1970
36.4% vs 5.9% prevalence

Risk of Type 2 DM post GDM related to BMI changes over time

Bao et al, Diabetologia 2015 – Nurses’ Health Study II
Postpartum GDM Management Checklist

1. Encourage Breastfeeding

2. 75g OGTT between 6 weeks - 6 months postpartum to detect prediabetes or diabetes

3. Discuss increased long-term risk of diabetes – Importance of returning to pre-pregnancy weight
What about after the baby is born — for the offspring?

Understand the controversies surrounding the diagnosis of GDM comparing 100g/75g criteria

Understand the short and long-term risks for mothers

Understand the short and long-term risks for the offspring...
In-Utero Fetal programming

A stimulus or an insult at a critical and sensitive period of early life which permanently alters the organism’s physiology and metabolism

Low Birth-weight → At risk neonate → High Birth-weight

Barker

Pederson/Frienkel/Pettit

Obese adult with insulin resistance, hypertension & type 2 DM
Maternal DM leads to increased DM in offspring

- **Pima Indian Population in Southern United States** ...moved from an agrarian lifestyle to almost no activity
  - NIH began studying the diabetes risks early in the 1970’s
  - Treatment of DM and GDM was not well-established initially, so even if diagnosed, glucose control was relatively poor. (personal communication with Dr. Bennett)

- **Increased obesity in offspring of Pima women with DM2 than non-DM2 women** (Pettit 1983)

- **In Pima Indians aged 25-34** Exposure to DM in utero was strongest risk factor for DM (Dabalea 1999)
  - 70% of offspring of DM2 mothers
  - 15% of offspring of non-DM mothers
  - Ages 7 – 11 already see increased systolic BP in offspring
  - Evidence of increase MAU 4 – 6X if DM in utero
Childhood Obesity & Metabolic Imprinting

- Offspring studied (9439) 5-7 years later
- Universal screen (> 140); NDDG criteria for treatment GDM

* sex specific weights

<table>
<thead>
<tr>
<th>Mother’s Glucose test result</th>
<th>Odds Ratio For Overweight (≥ 85%ile) child *</th>
<th>Odds ratio For Obese child (≥ 95%ile)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Reference</td>
<td>Reference</td>
</tr>
<tr>
<td>+ GS, normal OGTT</td>
<td>0.98</td>
<td>0.97</td>
</tr>
<tr>
<td>+ GS, 1 abn (CC or NDDG)</td>
<td>1.37</td>
<td>1.30</td>
</tr>
<tr>
<td>+ GS, + CC, - NDDG</td>
<td>1.89</td>
<td>1.82</td>
</tr>
<tr>
<td>+GS, + NDDG - treated</td>
<td>1.29</td>
<td>1.38</td>
</tr>
</tbody>
</table>

Offspring risk lower in treated women than with milder GDM untreated

_Hillier, T et al: Diabetes Care 2007(30)2287_
### Long-term studies looking at offspring

<table>
<thead>
<tr>
<th>Author, year</th>
<th>Pays</th>
<th>Patients / Controls</th>
<th>Age of offspring</th>
<th>GI %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plagemann 1997</td>
<td>Germany</td>
<td>Offspring DM1 &amp; DM2</td>
<td>Age 1 - 4</td>
<td>9.4%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>20%</td>
</tr>
<tr>
<td>Silverman 1998</td>
<td>Chicago, USA</td>
<td>96 Offspring of DM1/DM2 &amp; GDM</td>
<td>Age 8 – 17</td>
<td>31.5%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Amnio. Ins. Lo vs hi</td>
<td></td>
<td>17% / 63%</td>
</tr>
<tr>
<td>Dabalea 1999</td>
<td>Pima USA</td>
<td>Offspring of GDM/DM or not</td>
<td>Age 25 – 34 - GDM Controls: Non GDM</td>
<td>70%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>15%</td>
</tr>
<tr>
<td>Keely 2006</td>
<td>Ottawa, Canada</td>
<td>Offspring of GDM treatment intense or not</td>
<td>Age 8 – 17</td>
<td>36%</td>
</tr>
<tr>
<td>Clausen 2007</td>
<td>Denmark</td>
<td>Offspring of + RF women + or – for GDM; O-DM1, O-Bkgd population</td>
<td>Age 18-27 O-GDM O-nonGDM</td>
<td>21%OR</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>12%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>4%</td>
</tr>
<tr>
<td>Vaarasmaki, 2009</td>
<td>Finland</td>
<td>1986 Finnish birth cohort</td>
<td>Overweight O-GDM 18.4 vs 8.4% (P&lt;0.001)</td>
<td>Not reported</td>
</tr>
<tr>
<td>Egeland, Meltzer 2010</td>
<td>Montreal Canada</td>
<td>Case-control study matched for age, social status 89 cases, 99 controls</td>
<td>Age 14-16 girls GDM Controls</td>
<td>1%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0%</td>
</tr>
</tbody>
</table>

**RR of IGT = 4.7 for ODM with elevated insulin level**

**OR O-GDM relative to O-Background for DM/GI was 7.76**

**Very low incidence of offspring GI???'***
Obese adolescent offspring of GDM more at risk for DM2/IGT conversion

Obese NGT
N= 255

NGDM
n = 210 (82.3%)

NGT
n = 192 (91.4%)

IGT/DM2
n = 18 (8.6%)

EGDM
n = 45 (17.7%)

NGT
n = 31 (68.9%)

IGT/DM2
n = 14 (31.1%)

p<0.001

OR = 5.75 (95% CI 2.19, 15.07, p < 0.001)

Holder et al, Diabetologia 2014; 57:2413
Like mother... like daughter

Mothers’ Saturated Fat Intake (%) ** predicts Daughters adjusted for mother’s case status

Mothers’ MET Score ** predicts daughters MET score adjusted for case status and daughter’s age

Meltzer, Egeland, EASD abstract, 2008
Diabetes in pregnancy begets Diabetes?

Can this circle be altered?

- Abnormal Maternal fuels
- Diabetes in pregnancy
- Increased incidence of PreDM/DM in the adult population
- Altered fetal islet / fat cells / insulin res.

- Poor Family eating & activity habits?
- Childhood obesity
- IGT in adolescence

Environmental Factors

February 2010
Lifestyle Prevention: Good Diet, Good Habits

All of these habits – less than 10% develop T2DM

- BMI < 25 (23 in Asians?)
- Diet high in cereal fiber & polyunsaturated fat and low in trans fat and glycemic load
- Exercise > 150 minutes/week of moderate intensity
- No smoking
- Consumption of low amounts of alcohol (< 9 drinks a week) may reduce risk

Nurse’s Health Study, NEJM 344:1343, 2001
Lifestyle Prevention: Activity

- Aerobic activity equal to brisk walking or more at least 3 times a week (≥150 min).
- Resistance exercises 3 times per week for 3 sets of 10 repetitions
- General increase in activity levels of any kind needs to be encouraged
Cost considerations

- Knowing lifetime excess medical costs attributable to diabetes provides a benchmark from which to measure the maximum future medical costs that could be avoided by preventing diabetes.

<table>
<thead>
<tr>
<th>Age at diagnosis</th>
<th>Discounted life-time medical spending for people with vs without diabetes</th>
</tr>
</thead>
<tbody>
<tr>
<td>40</td>
<td>$124,600</td>
</tr>
<tr>
<td>50</td>
<td>$91,200</td>
</tr>
<tr>
<td>60</td>
<td>$53,800</td>
</tr>
<tr>
<td>65</td>
<td>$35,900</td>
</tr>
</tbody>
</table>

- Younger age at diagnosis and female sex were associated with higher levels of lifetime excess medical spending attributed to diabetes.