What Guidelines to Use in Gestational Diabetes: ACOG or ADA?

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July 17, 2014
Objectives

- Why we care so much about diagnosing and treating GDM
  - Fetal programming of childhood obesity
- Why the push-back on adopting the ADA Guidelines?
  - Fetal overgrowth is caused by more than just glucose; role of obesity, GWG, lipids
- What is normal glycemia in pregnancy and at what level to adverse outcomes increase?
  - Glycemia norms and HAPO trial
- ACOG and ADA guidelines for Dx of GDM
  - Reasons for strong ACOG opposition
  - NIH Consensus Panel
- Final Thoughts
What Is GDM?

- Glucose intolerance recognized for the first time during pregnancy (brought out by the ↑ IR of normal pregnancy and ↑ demand on beta cells.
- Did not exclude women with pre-existing diabetes previously undiagnosed
  - Undiagnosed Type 2 with ↑A1C have risk for major malformations
  - Most women diagnosed before 24 weeks have prediabetes or frank Type 2 DM
New CDC Estimated Prevalence of GDM ~9.2% 
June 2014


Carla L. DeSisto, MPH; Shin Y. Kim, MPH; Andrea J. Sharma, MPH, PhD


Abstract

Introduction
The true prevalence of gestational diabetes mellitus (GDM) is unknown. The objective of this study was 1) to provide the most current GDM prevalence reported on the birth certificate and the Pregnancy Risk Assessment Monitoring System (PRAMS) questionnaire and 2) to compare GDM prevalence from PRAMS across 2007–2008 and 2009–2010.

Methods
We examined 2010 GDM prevalence reported on birth certificate or PRAMS questionnaire and concordance between the sources. We included 16 states that adopted the 2003 revised birth certificate. We also examined trends from 2007 through 2010 and included 21 states that participated in PRAMS for all 4 years. We combined GDM prevalence across 2-year intervals and conducted t-tests to examine differences. Data were weighted to represent all women delivering live births in each state.

Results
GDM prevalence in 2010 was 4.6% as reported on the birth certificate, 8.7% as reported on the PRAMS questionnaire, and 9.2% as reported on either the birth certificate or questionnaire. The agreement between sources was 94.1% (percent positive agreement = 87.0%, percent negative agreement = 90.4%). There was no significant difference in GDM prevalence between 2007–2008 (8.1%) and 2009–2010 (8.5%, P = .15).

Conclusion
Our results indicate that GDM prevalence is as high as 9.2% and is more likely to be reported on the PRAMS questionnaire than the birth certificate. We found no statistical difference in GDM prevalence between the 2 phases. Further studies are needed to understand discrepancies in reporting GDM by data source.
Why Do We Care about Dx and RX of GDM?

- Mothers have ↑ risk of C-section and preeclampsia
- Offspring have ↑ LGA, birth trauma, respiratory, cardiac and metabolic disorders
- Identifies moms who have 30-50% risk of Type 2 DM in 10-20 yrs
- Offspring have ↑ risk of childhood obesity and DM
- 2 RCTs show dx and Rx improves some outcomes
  - ACHOIS 2005 using WHO criteria (perinatal mortality)
  - MFMU 2009 using CC criteria ↓ LGA, macrosomia, PE shoulder dystocia
Poor Nutrition in Early Life

Organ Development Changes Gene Expression

Catch Up Growth

Metabolic Syndrome


Updated Hypothesis:

Overnutrition in Utero

Changes in Gene Expression

Environmental Stimuli, Continued Postnatal Growth

Metabolic Syndrome

Epigenetic Stimuli

Developmental Origins Theory—not just GDM

BW and Risk of Childhood Metabolic Syndrome

Low Birth Weight = 7.6% of all births
Followed by rapid catch up growth
Large for Gestational Age = 20%

The Hungry Gene

The Science of Fat and the Future of Thin
ELLEN RUPPEL SHELL
High birth weight and increased adiposity at age 12 months increases risk of metabolic syndrome at age 17 yrs old in girls.

Obese infants are 2-9 times as likely to be obese as adults. 

Baird J, BMJ 2005;331:929
Increased Neonatal Adiposity in Offspring of GDM

Table III. Neonatal body composition

<table>
<thead>
<tr>
<th></th>
<th>GDM (n = 195)</th>
<th>NGT (n = 220)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>FFM (g)</td>
<td>2962 ± 405</td>
<td>2975 ± 408</td>
<td>.74</td>
</tr>
<tr>
<td>Fat mass (g)</td>
<td>436 ± 206</td>
<td>362 ± 198</td>
<td>.0002</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>12.4 ± 4.6</td>
<td>10.4 ± 4.6</td>
<td>.0001</td>
</tr>
</tbody>
</table>

Data are presented as mean ± SD.

B.W. = 2893 gms; Body Fat = 16.8% by DXA

Catalano PM; 2003; Am J Obstet Gynecol; 189:1698
Newborn Intrahepatic Fat Increased in GDM/Obese Offspring
Brumbaugh, Pediatrics 2013

- Visceral fat associated with severe insulin resistance
- Hepatic fat is associated with NAFLD
- N=13 infants of obese GDM and 12 NW mothers
- Infants of Obese GDM moms had 68% more intrahepatic fat
- Genesis of NAFLD?
But Maternal Glucose is not the only Risk Factor for Childhood Obesity

- Factors Associated with high BMI at 2-3 yr:
  - Maternal BMI, LGA, GWG, Glucose (GDM or DM), Lipids, Dietary fat
  - Rate of Infant Weight Gain
    0-6 mo infants triple their fat mass
    - Rapid wt gain birth-2 yrs; Catch up-growth in IUGR
  - Feeding mode
    - BF protective in most studies

Poston L. Curr Opin Clin Nutr Metab Care 2012
Offspring from OW/Obese Moms Account for Most LGA Births

Delaney Buzzell “Big Enchilada” 13 lb 12 oz Mom without GDM

***Obesity prevalence was 9.3% in 1960...32% in 2010

Ogden, 2006, JAMA, 295(13): 1549
Why Do Obese Mothers without GDM Deliver Big Babies?

Table 3. Neonatal Body Composition of Infants of Women With Pregravid Body Mass Index (BMI) Less Than 25 Compared With Those With BMI of 25 or More

<table>
<thead>
<tr>
<th>Pregravid Body Mass Index</th>
<th>Less Than 25 (n=144)</th>
<th>25 or More (n=76)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth weight (g)</td>
<td>3,284±534</td>
<td>3,436±507</td>
<td>.061</td>
</tr>
<tr>
<td>Body composition (TOBEC)</td>
<td>2,951±406</td>
<td>3,023±410</td>
<td>.22</td>
</tr>
<tr>
<td>Lean body mass (g)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fat mass (g)</td>
<td>331±179</td>
<td>406±221</td>
<td>.008</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>9.6±4.3</td>
<td>11±4.7</td>
<td>.006</td>
</tr>
</tbody>
</table>

TOBEC, total body electrical conductivity.

Catalano PM 2007;109:419
Glucoses higher in Obese/non-GDM women compared to NW throughout day and night both early and late in pregnancy on Controlled Diet by CGM
TGs and FFAs Correlate Strongest with Infant Body Fat

Harmon, Gerard, Jensen, Kealey, Hernandez, Reece, Barbour, Bessessen Diabetes Care 2011;34:1

TG early was strongest correlate of % body fat ($r=0.67$); FFA late ($r=0.54$)

Early Maternal BMI $r=0.55$

Nothing added to TG in regression model

BW not correlated with any metabolic variables
Change in TGs Early to Late in Pregnancy Correlates with %Fat at Birth by DXA (NIH R01DK78645)

Preliminary data, Barbour LA, Regulation of Maternal Fuel Supply and Neonatal Adiposity
Maternal Obesity is Much Stronger Risk for Childhood Obesity than GDM

Catalano PM Am J Clin Nutr 2009;90:1303

89 women with GDM or NGT; Offspring evaluated at birth; 6-11 yrs by DXA

No diff in ~ 9 yr old Wt Percentile or % Fat in Offspring of GDM vs NGT

Strongest predictor for offspring % Body Fat at ~9 yrs was Mat BMI >30 (OR=5.5). Explained 18% of variance in childhood adiposity

Correlation Between % Fat Birth and Child % Fat

r=0.3

**FIGURE 1.** Correlation between percentage body fat in neonates at birth and percentage body fat in children at follow-up. $r = 0.29, P = 0.02 (n = 63)$. 
Gestational Weight Gain Contributes More to LGA than Overweight or GDM

*Kim SY Obstet Gynecol 2014;123:737*

Population Attributable Risk to LGA

BC data Florida 2004-2008 in Florida to assess influence of BMI vs GWG vs GDM on LGA

**Excessive GWG contributed the most to LGA**

Target GWG more than Pre-Pregnancy BMI or GDM.
GDM Only Part of the Problem:
Predictive Value of BW, Obesity and GDM on Metabolic Syndrome Age 6-11

Boney CM 2005 Peds 115:e290

<table>
<thead>
<tr>
<th>Variables</th>
<th>Hazard Ratio</th>
<th>( P ) Value</th>
<th>95% CI for Hazard Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>LGA versus AGA</td>
<td>2.19</td>
<td>.006</td>
<td>1.25–3.82</td>
</tr>
<tr>
<td>Maternal obesity* versus nonobese</td>
<td>1.81</td>
<td>.039</td>
<td>1.03–3.19</td>
</tr>
<tr>
<td>GDM versus control</td>
<td>1.44</td>
<td>.191</td>
<td>0.83–2.50</td>
</tr>
<tr>
<td>Male versus female</td>
<td>1.52</td>
<td>.133</td>
<td>0.88–2.61</td>
</tr>
</tbody>
</table>

* Prepregnancy BMI of >27.3 mg/m²
But the Sugar is Obviously Important

Patterns of Glycemia in Normal Pregnancy
Should the current therapeutic targets be challenged?

Teri L. Hernandez, PhD, RN
Rachel E. Van Pelt, PhD
Jacob E. Friedman, PhD
Linda A. Barbour, MD, MSPh

19 lbs 2oz Baby Boy; Mother 41 with DM
Medan, North Sumatra Indonesia

- 12 studies
- 1975-2008
- N=168-255
- 33.8±2.3 wks
- BMI 22-28
Normal Glycemia In Pregnancy is Lower than Recognized

Hernandez T,
Barbour LA et al, Diabetes Care Oct 2011
What Diagnostic Criteria is Used for GDM?
ACOG Criteria Predicts T2 DM, Not Adverse Outcomes

Practice Bulletin #137 Aug 2013

Two Step:

- 50 gm glucose screen non fasting
  - 130-140 cut-off
- 100 gm OGTT (Carpenter and Coustan)
  - FBG ≥ 95 mg/dl
  - 1 hr ≥ 180 mg/dl
  - 2 hr ≥ 155 mg/dl
  - 3 hr ≥ 140 mg/dl
- National Diabetes Data Group
  - FBG ≥105
  - 1 hr ≥190; 2 hr ≥165; 3 hr ≥135
  - Used less often; different conversion from whole blood to plasma from O’Sullivan criteria)
25,505 women in 15 centers in 9 countries

Objective: Clarify risk of adverse outcome with degrees of maternal glucose intolerance

75 gm 2 hr OGTT; Blinded if FBG <105; 2hr <200

Primary outcomes: LGA, C sec, Neonatal hypoglycemia, Cord blood C-peptide >90th %
International Association of Diabetes and Pregnancy Study Groups Recommendations on the Diagnosis and Classification of Hyperglycemia in Pregnancy

Diabetes Care 2010;33:676-682

Diagnosis and Classification of Diabetes Mellitus

American Diabetes Association

Diabetes Care 2011;34 S62-S69
**DX of GDM and Overt DM per IADPSG**

**Diagnosis of hyperglycemia in pregnancy**

**Table 1—Threshold values for diagnosis of GDM or overt diabetes in pregnancy**

To diagnose GDM and cumulative proportion of HAPO cohort equaling or exceeding those thresholds

<table>
<thead>
<tr>
<th>Glucose measure</th>
<th>Glucose concentration threshold*</th>
<th>Above threshold (%)</th>
<th>Cumulative</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mmol/l</td>
<td>mg/dl</td>
<td></td>
</tr>
<tr>
<td>FPG</td>
<td>5.1</td>
<td>92</td>
<td>8.3</td>
</tr>
<tr>
<td>1-h plasma glucose</td>
<td>10.0</td>
<td>180</td>
<td>14.0</td>
</tr>
<tr>
<td>2-h plasma glucose</td>
<td>8.5</td>
<td>153</td>
<td>16.1†</td>
</tr>
</tbody>
</table>

**Incidence of GDM = 17.8%**

To diagnose overt diabetes in pregnancy

<table>
<thead>
<tr>
<th>Measure of glycemia</th>
<th>Consensus threshold</th>
</tr>
</thead>
<tbody>
<tr>
<td>FPG‡</td>
<td>≥7.0 mmol/l (126 mg/dl)</td>
</tr>
<tr>
<td>A1C‡‡</td>
<td>≥6.5% (DCCT/UKPDS standardized)</td>
</tr>
<tr>
<td>Random plasma glucose</td>
<td>≥11.1 mmol/l (200 mg/dl) + confirmation§</td>
</tr>
</tbody>
</table>

*One or more of these values from a 75-g OGGT must be equaled or exceeded for the diagnosis of GDM. †In addition, 1.7% of participants in the initial cohort were unblinded because of FPG > 5.8 mmol/l (105 mg/dl) or 2-h OGGT values > 11.1 mmol/l (200 mg/dl), bringing the total to 17.8%. ‡One of these must be met to identify the patient as having overt diabetes in pregnancy. §If a random plasma glucose is the initial measure, the tentative diagnosis of overt diabetes in pregnancy should be confirmed by FPG or A1C using a DCCT/UKPDS-standardized assay.
Screening and Diagnosis of Gestational Diabetes Mellitus

The recent studies on GDM and its increasing incidence in the United States underscore the need for the development of uniform screening and diagnostic criteria. (9, 10), there is no evidence that the identification and treatment of women based on the new International Association of Diabetes in Pregnancy Study Group recommendations will lead to clinically significant improvements in maternal and neonatal outcomes and it would lead to a significant increase in health care costs.
FOR DEBATE

Diagnosing gestational diabetes

E. A. Ryan

Canadian representative on IADPSG Consensus Panel who did not join authorship

Reproducibility of OGTT poor (25% could be re-classified)

Increased diagnostic rate of 17.8% would prevent only 140 cases LGA and 16 cases of birth injury in 23,000 pregnancies

78% LGA infants were not born to GDM mothers; maternal Obesity is a stronger predictor of LGA

Rec: Screening with 50 gm and using higher cutoffs on a 75 gm OGTT using a **2-fold risk** of LGA which would give a prevalence of 10.5%

FBG ≥ 95 mg/dl or 1 hr ≥191 OR 2 hr ≥162 (5.3, 10.6, and 9 mmol/l)
*Variability in 2 hr OGTT: differing results in ~25% of women if performed at different times. 1 step testing may result in more FPs

*Pooled meta-analysis of 5 RCTs: Rx of GDM resulted in BW difference of <150 gm; ~6% risk reduction of LGA; May ↑ C-section and NICU rates

*Obesity contributes most to LGA. Treating milder GDM may not benefit

*Adopting ADA criteria: projected 1,000,000 more clinic visits and prenatal tests per yr; $636 million to $2 billion?
True Benefits of Rx of Mild GDM?

- No difference in obesity risk in 4-5 yr old offspring in ACHOIS. *Diabetes Care* 2010;33:964
- 78% of LGA babies in HAPO no GDM; >90% shoulder dystocia no GDM
- ACHOIS; 5 perinatal deaths but 2/5 due to lethal anomaly or IUGR
  - Rx: ↓ BW 140 gm ↓ PIH by 6%; ↑ IOL, Resp distress, neonatal hypoglycemia
- MFMU; N.S. in perinatal mortality or composite outcomes
  - Rx ↓ BW 100 gm, PIH by 5%, shoulder dystocia (1.5 vs 4%)
  - 5 RCTs and 6 Cohort Studies
  - Women who are treated for GDM have more perinatal visits
  - Moderate evidence shows ↓ PIH, shoulder dystocia, LGA/ Macrosomia; No diff C-section, IOL, neonatal hypoglycemia, long term infant outcomes
More Controversy: IADPSG Early Screening Paradigm Will Miss DM/GDM Cases

Screening for Diabetes and Pre-Diabetes With Proposed A1C-Based Diagnostic Criteria

Diabetes Care 2010;33:2184-89

The A1C criteria misses 70% of individuals with DM (≥6.5) and 82-94% with prediabetes (≥5.7) compared to OGTT data in non-Hispanic White or Black adults.

HAPO did not advocate using an A1C of 5.7 to diagnose GDM early or proceed with 75 gm testing.

Unless a fasting or 75 gm or 2 hr OGTT is performed on high risk women for GDM in the first trimester, many early cases of GDM and up to 1/3 cases of DM will be missed!
## Pros and Cons - You Decide

<table>
<thead>
<tr>
<th><strong>ACOG</strong></th>
<th><strong>ADA</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>CC or NDDG criteria predict maternal DM risk, not pregnancy outcomes</td>
<td>Criteria based on HAPO fetal outcomes; 1.75 ↑ OR risk arguable</td>
</tr>
<tr>
<td>Fasting not required for 50gm screen</td>
<td>Requires fasting; ↓ blood draws</td>
</tr>
<tr>
<td>Missed/delay in dx 2 step approach</td>
<td>No delay; precision of single value?</td>
</tr>
<tr>
<td>Only used in U.S.</td>
<td>Global use of 75 gm OGTT</td>
</tr>
<tr>
<td>Clear criteria to screen early for GDM</td>
<td>Early GDM missed with A1C, gluc</td>
</tr>
<tr>
<td>Unclear diagnosis of overt DM but most OBs use A1C of 6.5</td>
<td>All/High Risk women receive A1C, FBG, or random glucose early</td>
</tr>
<tr>
<td>Prevalence ~ 6-9%</td>
<td>Prevalence ~18%</td>
</tr>
<tr>
<td>Some benefit supported by MFMU data which used C/C</td>
<td>Unclear benefit of treating IADPSG criteria without large RCTs</td>
</tr>
<tr>
<td>Inadequate resources to treat if prevalence tripled; target obesity</td>
<td>Prediabetes 26% by NHANES; Why not treat in pregnancy?</td>
</tr>
</tbody>
</table>
Gestational Diabetes Mellitus

Gestational diabetes mellitus (GDM) is one of the most common medical complications of pregnancy. Debate continues to surround both the diagnosis and treatment of GDM despite several recent large-scale studies addressing these issues. The purpose of this document is to 1) provide a brief overview of the understanding of GDM, 2) provide management guidelines that have been validated by appropriately conducted clinical research, and 3) identify gaps in current knowledge toward which future research can be directed.

A COUNTRY DIVIDED — Lack of consistent criteria for Research Trials; Patient and Practitioner Confusion

Diabetes and Pregnancy: An Endocrine Society Clinical Practice Guideline

Ian Blumer, Eran Hadar, David R. Hadden, Lois Jovanović, Jorge H. Mestman, M. Hassan Murad, and Yariv Yogev

Diabetes Care 2013;33(5):964
## Guidelines for Gestational Diabetes (GDM)

### Screening and Diagnosis

**OGTT = Oral Glucose Tolerance Test, 3-hour**

<table>
<thead>
<tr>
<th>First Prenatal Encounter: Universal Risk Assessment</th>
<th>24-28 Weeks: Universal Screening</th>
</tr>
</thead>
<tbody>
<tr>
<td>High-risk if any of the following:</td>
<td></td>
</tr>
<tr>
<td>- Advanced maternal age (&gt;35 y.o.)</td>
<td></td>
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<tr>
<td>- Obesity (BMI ≥ 25 kg/m² based on prep)</td>
<td></td>
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<tr>
<td>- High-risk ethnic background</td>
<td></td>
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<tr>
<td>- T1DM or T2DM</td>
<td></td>
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<tr>
<td>- Previous macrovascular or microvascular complications</td>
<td></td>
</tr>
<tr>
<td>- Family history of diabetes</td>
<td></td>
</tr>
<tr>
<td>- PCOS</td>
<td></td>
</tr>
<tr>
<td>- Glycosuria</td>
<td></td>
</tr>
</tbody>
</table>

**OGTT = Oral Glucose Tolerance Test, 3-hour**

<table>
<thead>
<tr>
<th>1-hour OGGT</th>
<th>2-hour OGGT</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥ 135 mg/dl</td>
<td>&lt; 130-140 mg/dl</td>
</tr>
<tr>
<td>&lt; 135 mg/dl</td>
<td>&lt; 120 mg/dl</td>
</tr>
</tbody>
</table>

**OGTT Diagnostic Criteria for Gestational Diabetes**

- If 2 or more values meet or exceed thresholds, diagnosis of GDM.

<table>
<thead>
<tr>
<th>Time</th>
<th>mg/dl</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-hour</td>
<td>≤ 160</td>
</tr>
<tr>
<td>2-hour</td>
<td>≤ 155</td>
</tr>
<tr>
<td>3-hour</td>
<td>≤ 140</td>
</tr>
</tbody>
</table>

### Medical Nutrition Therapy (MNT) and Physical Activity

**Meal Planning**
- Educate on healthy food choices and smaller, frequent meals throughout the day.
- Teach portion control (plate method or carbohydrate counting) and reading food labels.
- Refer to an RDN or CDEx if available, or an RN trained in community health worker.

**Physical Activity**
- Recommend regular physical activity 30 min/day, 5 days/week.
- Consult with MNU or any contraindications.

### Blood Glucose Monitoring

**Self-Monitoring Blood Glucose Goals**

- Check and record BG 4x/day: fasting and 1 or 2 hours postprandial for a minimum of 2 weeks.
- Never discontinue SMBG during GDM. Remain vigilant as glucose intolerance increases as pregnancy progresses. If frequency is decreased, rotate SMBG at different times each day.
- If 20% of BG values exceed the target while following prescribed nutrition and physical activity plan, consider medication therapy.

### Medication Management

**Oral**
- Glyburide is the only oral hypoglycemic agent that may be considered as an alternative to insulin.
- Metformin should not be initiated in pregnancy. If used to manage PCOS risks, discontinue after 1st trimester.

**Insulin**
- Use SMBG to guide the dosage and timing of the insulin regimen.
- Aspart and Lispro are the most effective at reducing postprandial glycemic excursions.
- Regular and NPH have also been used safely in pregnancy.

### Prenatal Surveillance and Delivery Management

**Surveillance**
- A fetal based strategy (AC > 75%tile at 28-33 weeks) may help identify women that may benefit from more intensive medical management.
- Prenatal surveillance may include NST, AFI, Biophysical Profile or Contraction Stress Test. Selection of the prenatal test is at the discretion of the practitioner.

**Diet Controlled**
- Euglycemic: initiate surveillance at 40 weeks.
- Not euglycemic: initiate surveillance at 36 weeks.

**Medication Controlled**
- If pregnancy is not otherwise complicated, initiate surveillance at 33-34 weeks.

**Delivery**
- There is no data to support delivery at <38 wks or cesarean delivery purely on the basis of GDM.

### Postpartum Follow-Up

Due to the increased risk of developing type 2 diabetes, it is crucial that women return to their provider to receive the appropriate postpartum counseling, testing, and follow-up after a GDM pregnancy. See reverse for GDM Postpartum Algorithm.

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*These clinical guidelines were approved 9/22/06 and are adapted from the American Diabetes Association (ADA) Standards of Medical Care in Diabetes 2006. They are designed to assist clinicians in managing women with gestational diabetes and are not intended to replace a clinician’s judgment or withhold care for all women with gestational diabetes. For references, important updates, additional copies of guidelines, go to [http://www.cdphe.state.co.us/pp/womens/gestationaldiabetes.html](http://www.cdphe.state.co.us/pp/womens/gestationaldiabetes.html).*
diagnostic criteria for GDM. GDM screening can be accomplished with either of two strategies:

1. “One-step” 2-h 75-g OGTT or
2. “Two-step” approach with a 1-h 50-g (nonfasting) screen followed by a 3-h 100-g OGTT for those who screen positive (Table 6)

The conflicting recommendations from these two consensus panels underscore several key points:

1. There are insufficient data to strongly demonstrate the superiority of one strategy over the other.
2. The decision of which strategy to implement must therefore be made based on the relative values placed on currently unmeasured factors (e.g., cost-benefit estimation, willingness to change practice based on correlation studies rather than clinical intervention trial results, relative role of cost considerations, and available infrastructure).
3. Further research is needed to resolve these uncertainties.
Final Thoughts

- Lower maternal glycemia than previously recognized contributes to infant adiposity, adverse outcomes
- Obesity, GWG, diet, lipids, AAs, GFs, placental function, oxidative stress, inflammation all contribute to LGA
- Whether adopting the IADPSG criteria without modifying above will succeed in improving outcomes is unclear; Resources/cost?
- As an Endocrinologist practicing in an OB setting, ACOG criteria used; I would favor 75 mg OGTT with 2-fold OR cutoff
  - Move to Canada OR
  - Intensify glucose targets if fetal AC is >90% on US (fetal based strategy)
  - Target Pre-Preg Wt Loss, Lower GWG than IOM Recs, Low Fat/Complex Carb diet, TG lowering (diet, Omega-3) if high, ↑Physical Activity (↓LGA)
Diabesity Begets Diabesity: Halting the Cycle

Genetics

INSULIN RESISTANCE

Central Obesity
Hyperlipidemia
Glucose Intolerance
Inflammation/Ox Stress
High Fat Diet

Metabolic Syndrome

TYPE 2 DIABETES

Environment

Epigenetics

Fetal Metabolic Programming

Catch-up Growth

Childhood Obesity, IGT

Your Genes
Your Health