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- Developed for local health professionals, by local health professionals
- Clear, local and relevant referral options
Trusted information at the point of care

Register and access ACT & SNSW HealthPathways today

https://actsnsww.healthpathways.org.au
Prof Chris Nolan
Director of Endocrinology, ACT Health
Type 1 and type 2 diabetes in pregnancy
Pre-pregnancy planning and medical management

Capital Health *Education* - 14\(^{th}\) Oct 2017

Christopher Nolan
Canberra Hospital & Health Services
Australian National University Medical School
Prevalence of diabetes in pregnancy (Australia)

• Pre-existing type 1 diabetes about (0.3-0.4%)
• Pre-existing type 2 diabetes (>1.0 %)
• Gestational diabetes mellitus (8-13%)
Diabetic fetopathy

Mother

Glucose
Lipids
Amino acids

Placenta

Fetus

Excessive fetal growth

Hyperinsulinemia
Infant of diabetic mother
Overview

• Outcomes of pregestational T1D and T2D
• Importance of pregnancy planning
• Fetal-glucose steal phenomenon
• Pre-pregnancy care and medical care during pregnancy
Overview

- Outcomes of pregestational T1D and T2D
- Importance of pregnancy planning
- Fetal-glucose steal phenomenon
- Pre-pregnancy care and medical care during pregnancy
The St Vincent Declaration 1989

- To reduce (within 5 years) adverse pregnancy outcomes in women with type 1 diabetes (T1D) to a level equal to that of women without diabetes
Failure to achieve goals of the St Vincent Declaration

- Assessment of 12 population-based studies in Europe
  - 14,099 women T1D
  - 4,035,373 women from background population

- T1D (%) vs BKG (%) and Relative risk for:
  - Cong malformation: 5.0% vs 2.1%; RR 2.4
  - Perinatal mortality: 2.7% vs 0.72%; RR 3.7
  - Preterm infants: 25.2% vs 6.0%; RR 4.2
  - Large for GA infants: 54.2% vs 10.0%, RR 4.5

- Early pregnancy HbA1c was positively associated with adverse pregnancy outcomes

Failure to achieve goals of the St Vincent Declaration

“There was a higher occurrence of first trimester HbA1c > 8.0% in women not receiving pre-conception care compared with women who did receive pre-conception care (55% versus 4.3%).”

Type 1 and 2 diabetes in pregnancy (Australia-NSW)

- Audit of 180 pregnancies- pre-existing diabetes
- T1D (45%) and T2D (55%) pregnancies
- Perinatal mortality T1D 1.2%; T2D 5.1%
- Congenital malf T1D 6%; T2D 10%

Pregestional diabetes outcomes- ACT

• Work of medical student - Jacqui Jones
• Restrospective clinical audit 2009-1013
• 146 pregnancies (120 women) with type 1 or type 2 diabetes
• x2 twin, x1 triplet pregnancies- analysed separately
• Of singleton pregnancies
  – 90 type 1 diabetes
  – 53 type 2 diabetes
<table>
<thead>
<tr>
<th>Variable</th>
<th>Type 1 diabetes (n=90)</th>
<th>Type 2 diabetes (n=53)</th>
<th>Sig. p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
</tr>
<tr>
<td><strong>Maternal characteristics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean age at delivery (years)</td>
<td>29.32 (±6.17)</td>
<td>33.37 (±5.35)</td>
<td>0.000*</td>
</tr>
<tr>
<td>Advanced maternal age (≥35)</td>
<td>21</td>
<td>23.1</td>
<td>21</td>
</tr>
<tr>
<td>Mean duration of diabetes (years)</td>
<td>12.17 (±7.20)</td>
<td>4.42 (±3.69)</td>
<td>0.000</td>
</tr>
<tr>
<td>Nulliparous</td>
<td>44</td>
<td>48.4</td>
<td>34</td>
</tr>
<tr>
<td>Nulligravida</td>
<td>31</td>
<td>34.1</td>
<td>12</td>
</tr>
<tr>
<td>Multigravida (G3+)</td>
<td>31</td>
<td>34.1</td>
<td>30</td>
</tr>
<tr>
<td>Mean pre-pregnancy BMI (kg/m²)</td>
<td>27.40 (±6.00)</td>
<td>32.65 (±6.35)</td>
<td>0.000*</td>
</tr>
<tr>
<td>Overweight/obese (BMI ≥25kg/m²)</td>
<td>50</td>
<td>61.7</td>
<td>47</td>
</tr>
<tr>
<td>Current smoker</td>
<td>9</td>
<td>10.1</td>
<td>4</td>
</tr>
</tbody>
</table>
## Glycaemic control/ blood pressure

<table>
<thead>
<tr>
<th>Variable</th>
<th>Type 1 diabetes (n=90)</th>
<th>Type 2 diabetes (n=53)</th>
<th>Sig. p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
</tr>
<tr>
<td>Mean first trimester HbA1c (%)</td>
<td>7.75 (±1.50)</td>
<td>6.86 (±1.10)</td>
<td>0.001*</td>
</tr>
<tr>
<td>First trimester HbA1c &gt;7.0% †</td>
<td>48</td>
<td>55.8</td>
<td>20</td>
</tr>
<tr>
<td>Late pregnancy HbA1c (%)</td>
<td>6.85 (±1.02)</td>
<td>5.90 (±0.72)</td>
<td>0.000 ‡</td>
</tr>
<tr>
<td>Late pregnancy HbA1c &gt;7.0% †</td>
<td>29</td>
<td>34.1</td>
<td>3</td>
</tr>
<tr>
<td>Systolic blood pressure &gt;130mmHg</td>
<td>30</td>
<td>35.7</td>
<td>13</td>
</tr>
<tr>
<td>Diastolic blood pressure &gt;80mmHg</td>
<td>39</td>
<td>46.4</td>
<td>20</td>
</tr>
</tbody>
</table>
### Major pregnancy outcomes

<table>
<thead>
<tr>
<th>Variable</th>
<th>Type 1 diabetes (n=90)</th>
<th>Type 2 diabetes (n=53)</th>
<th>Sig. p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
</tr>
<tr>
<td><strong>Major outcomes</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Perinatal death</td>
<td>3</td>
<td>3.4</td>
<td>1</td>
</tr>
<tr>
<td>Gender male</td>
<td>49</td>
<td>57.0</td>
<td>34</td>
</tr>
<tr>
<td>Mean gestation (weeks)</td>
<td>36.90 (±2.32)</td>
<td></td>
<td>37.51 (±2.70)</td>
</tr>
<tr>
<td>Preterm delivery (&lt;37 weeks)</td>
<td>31</td>
<td>36.0</td>
<td>10</td>
</tr>
<tr>
<td>Mean birth weight (g)</td>
<td>3598.3 (±0.77)</td>
<td></td>
<td>3222.8 (±0.75)</td>
</tr>
<tr>
<td>Large for gestational age</td>
<td>53</td>
<td>61.6</td>
<td>11</td>
</tr>
<tr>
<td>Macrosomia</td>
<td>28</td>
<td>32.6</td>
<td>8</td>
</tr>
<tr>
<td>Major birth trauma</td>
<td>2</td>
<td>2.3</td>
<td>0</td>
</tr>
<tr>
<td>Major congenital abnormality</td>
<td>7</td>
<td>8.1</td>
<td>3</td>
</tr>
<tr>
<td>Admission to SCN / NICU</td>
<td>38</td>
<td>42.7</td>
<td>11</td>
</tr>
<tr>
<td>Neonatal hypoglycaemia</td>
<td>34</td>
<td>42.5</td>
<td>12</td>
</tr>
</tbody>
</table>
### Birth outcomes

#### Table 1: Details of birth status from singleton pregnancies

<table>
<thead>
<tr>
<th>Birth status</th>
<th>Count</th>
<th>Gestation</th>
<th>Reason / cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Live birth</td>
<td>137</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Miscarriage</td>
<td>3</td>
<td>16</td>
<td>Suspected Trisomy 8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>18</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>7+</td>
<td></td>
</tr>
<tr>
<td>Still birth</td>
<td>2</td>
<td>21</td>
<td>Trisomy 18</td>
</tr>
<tr>
<td></td>
<td></td>
<td>30</td>
<td></td>
</tr>
<tr>
<td>Neonatal death</td>
<td>1</td>
<td>23</td>
<td>Prematurity</td>
</tr>
<tr>
<td>Termination</td>
<td>2</td>
<td>20</td>
<td>Trisomy 18</td>
</tr>
<tr>
<td></td>
<td></td>
<td>13</td>
<td>Unstable diabetic retinopathy</td>
</tr>
</tbody>
</table>
ACT vs NSW vs Europe

- **NSW**
  - 180 pregnancies, 10 teaching hospitals
  - Type 1 - 45%  Type 2 – 55%

- **Europe**
  - 14,099 women T1D, 4,035,373 background population
## ACT vs NSW vs International

<table>
<thead>
<tr>
<th></th>
<th>ACT</th>
<th>NSW</th>
<th>Europe T1D</th>
<th>International Background</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pregnancies (n)</strong></td>
<td>143 (T1D/T2D)</td>
<td>180 (T1D/T2D)</td>
<td>14,099 T1D</td>
<td>4,035,373</td>
</tr>
<tr>
<td><strong>Major congenital malformations</strong></td>
<td>7.0% (8.1%/5.7%)</td>
<td>8.1%</td>
<td>5.0%</td>
<td>1.5%</td>
</tr>
<tr>
<td><strong>Perinatal mortality</strong></td>
<td>2.8% (3.4%/1.9%)</td>
<td>3.4% (1.2%/5.1%)</td>
<td>2.7%</td>
<td>0.72%</td>
</tr>
<tr>
<td><strong>Preterm delivery &lt;37 weeks</strong></td>
<td>29% (36%/19%)</td>
<td>-</td>
<td>25%</td>
<td>6%</td>
</tr>
<tr>
<td><strong>Large for gestational age</strong></td>
<td>53% (62%/21%)</td>
<td></td>
<td>54%</td>
<td>10%</td>
</tr>
<tr>
<td><strong>Neonatal hypoglycaemia</strong></td>
<td>34% (43%/24%)</td>
<td>25%</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>
Overview

• Outcomes of pregestational T1D and T2D
• Importance of pregnancy planning
• Fetal-glucose steal phenomenon
• Pre-pregnancy care and medical care during pregnancy
## Importance of pre-pregnancy care (Type 1 Diabetes)

<table>
<thead>
<tr>
<th></th>
<th>Pre-pregnancy care</th>
<th>No pre-pregnancy care</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>140</td>
<td>149</td>
<td></td>
</tr>
<tr>
<td>1&lt;sup&gt;st&lt;/sup&gt; trimester HbA1c &gt;8%</td>
<td>6 (4.3%)</td>
<td>82 (55%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Perinatal mortality</td>
<td>1 (0.7%)</td>
<td>12 (8.1%)</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>Congenital malformation</td>
<td>1 (0.7%)</td>
<td>12 (8.1%)</td>
<td>&lt;0.005</td>
</tr>
</tbody>
</table>

Diabetes in pregnancy group, France. Diabetes Care 2003; 11: 2990-2993
Pregnancy outcomes of women with diabetes according to pre-pregnancy care attendance (type 1 and type 2 diabetes) East England 2004-2006

<table>
<thead>
<tr>
<th>Pre-preg care</th>
<th>YES</th>
<th>NO</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>181</td>
<td>495</td>
<td></td>
</tr>
<tr>
<td>Cong malf</td>
<td>1 (0.7%)</td>
<td>23 (5.6%)</td>
<td>0.02</td>
</tr>
<tr>
<td>Stillbirth</td>
<td>1 (0.7%)</td>
<td>6 (1.5%)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Neonatal death</td>
<td>0 (0%)</td>
<td>3 (0.7%)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Any serious adverse outcome</td>
<td>2 (1.3%)</td>
<td>32 (7.8%)</td>
<td>0.009</td>
</tr>
</tbody>
</table>

Overview

• Outcomes of pregestational T1D and T2D
• Importance of pregnancy planning
• Fetal-glucose steal phenomenon
• Pre-pregnancy care and medical care during pregnancy
Diabetic fetopathy

Mother

Placenta

Fetus
Excessive fetal growth

Hyperinsulinemia

Glucose
Lipids
Amino acids

Pedersen hypothesis
Diabetic fetopathy

Mother

- Glucose
- Lipids
- Amino acids

Placenta

Fetus

- Excessive fetal growth
- Hyperinsulinemia
- Fetal beta-cell dysfunction

Maternal beta-cell dysfunction

Pedersen hypothesis
Diabetic fetopathy

Mother

Maternal beta-cell dysfunction

Glucose
Lipids
Amino acids

Placenta

Hyperinsulinemia
Fetal beta-cell dysfunction

Fetus
Excessive fetal growth

Pedersen hypothesis
The fetal glucose steal: an underappreciated phenomenon in diabetic pregnancy

Gernot Desoye¹ · Christopher J. Nolan²³

Diabetologia (2016) 59:1089-1094
Maternal and fetal components to the maternal-fetal glucose gradient in diabetes

Desoyme G, Nolan CJ
Diabetologia (2016) 59:1089-1094
Effect of fetal hyperinsulinemia on maternal glucose tolerance

Adapted from: Weiss PA et al Am J Obstet Gynecol 184: 470-475
Effect of fetal hyperinsulinemia on maternal glucose tolerance

- Birth weight >4,000 g
- 7 of 21 (33%) if AFI was >7 microU/L
- 0 of 11 (0%) if AFI <7 microU/L

Fetal glucose steal and diabetes

- Will be exaggerated in hyperinsulinemic fetuses
- Will maintain a maternal-fetal glucose gradient even during periods of maternal normoglycemia
- Could be an explanation for normal glucose tolerance in late pregnancy of mothers with diabetes affected fetuses
Overview

• Outcomes of pregestational T1D and T2D
• Importance of pregnancy planning
• Fetal-glucose steal phenomenon
• Pre-pregnancy care and medical care during pregnancy
Type 1 and 2 guidelines before conception

- Strict control of blood glucose levels should be pursued (HbA1c level as close as possible to the reference range)
- Folate (1 mg/day) should be commenced
- T2D- Metformin often continued
- T2D- Sulphonylureas ceased
- Other oral medications should be optimized for pregnancy (e.g. antihypertensives changes, lipid lowering meds stopped)
- Diabetes complications screening/ assessment/ stabilisation should take place
Type 1 and 2 guidelines before conception

- Strict control of blood glucose levels should be pursued (HbA1c level as close as possible to the reference range)
- Folate (1 mg/day) should be commenced
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- T2D- Sulphonylureas ceased
- Other oral medications should be optimized for pregnancy (e.g. antihypertensives changes, lipid lowering meds stopped)
- Diabetes complications screening/ assessment/ stabilisation should take place

Family planning advice
Type 1 and 2 guidelines
Maternal diabetes complications- before conception

• Retinopathy – can progress in pregnancy – should be stabilized prior to conceiving
• Nephropathy- can significantly increase the risk of severe pre-eclampsia- can worsen - blood pressure control very important- ACEI and A2RB contraindicated in pregnancy
• Neuropathy- autonomic neuropathy can increase risk of adverse outcomes
• Macrovascular disease- exclude coronary artery disease
Care during pregnancy
Improved pregnancy outcomes in women with type 1 and type 2 diabetes but substantial clinic-to-clinic variations: a prospective nationwide study

Helen R. Murphy¹,² • Ruth Bell³ • Cher Cartwright⁴ • Paula Curnow⁴ • Michael Maresh⁵ • Margery Morgan⁶ • Catherine Sylvester⁴ • Bob Young⁴ • Nick Lewis-Barned⁷
National Pregnancy in Diabetes (NPID) 2015 audit in the UK

• Lower stillbirth rates reported in centres involved in NPID (2015) compared to the Confidential Enquiry into Maternal and Child Health (CEMACH) audit from 2002-3
  – T1D 10.7 vs 25.8/1000, $p = 0.0012$
  – T2D 10.5 vs 29.2/1000 births, $p = 0.0091$

• Improvement is possible

• Indicative of the value of national audit programs
Continuous glucose monitoring in pregnant women with type 1 diabetes (CONCEPTT): a multicentre international randomised controlled trial

Denice S Feig, Lois E Donovan, Rosa Corcoy, Kellie E Murphy, Stephanie A Amiel, Katharine F Hunt, Elisabeth Asztalos, Jon F R Barrett, J Johanna Sanchez, Alberto de Leiva, Moshe Hod, Lois Jovanovic, Erin Keely, Ruth McManus, Eileen K Hutton, Claire L Meek, Zoe A Stewart, Tim Wysocki, Robert O’Brien, Katrina Ruedy, Craig Kollman, George Tomlinson, Helen R Murphy, on behalf of the CONCEPTT Collaborative Group*

Published Online
September 15, 2017
http://dx.doi.org/10.1016/S0140-6736(17)32400-5
CONCEPTT Clinical Trial

- CGM was associated with:
  - Lower HbA1c in 3rd trimester (mean difference -0.19%; p=0.0207).
  - Less time hyperglycaemic (27% vs 32%; p=0.0279)
  - Comparable severe hypoglycaemia episodes (18 CGM and 21 control)
CONCEPTTT Clinical Trial

• CGM compared to capillary blood glucose monitoring resulted in:
  • Less LGA (odds ratio 0.51, 95% CI 0.28 to 0.90)
  • Less neonatal intensive care admissions >24 h (odds ratio 0.48; CI 0.26 to 0.86)
  • Less occurrence of neonatal hypoglycaemia (odds ratio 0.45; CI 0.22 to 0.89)
Take home messages

• Type 1 and type 2 diabetes in pregnancy
  – Pregnancy planning
  – Pregnancy planning
  – Pregnancy planning
  – Pregnancy planning
  – Pregnancy planning
  – Pregnancy planning
Thank you
Dr Peter Scott
Staff Specialist, Obstetrician and Gynaecologist
Diabetes in Pregnancy

Obstetrician’s perspective

Dr Peter Scott
14th October 2017
Objectives

- Brief overview of diabetes in pregnancy
- Dilemmas in obstetric management
- Take-home messages
Diabetes in Pregnancy

- 6 – 8% of pregnancies
- Pre-gestational
- Gestational - 90% of cases
- The “intersection”
  - Risk factors & co-morbidities, especially high BMI
  - Outcomes with type 2 diabetes
Effects of Pregnancy on Diabetes

- Hypoglycaemia in first trimester

- Placental “anti-insulin” hormones
  - Progesterone, HPL, cortisol

- Deterioration in renal function

- Progression of retinopathy
Effects of Diabetes on Pregnancy

- Miscarriage & congenital anomalies
- LGA & Macrosomia/ shoulder dystocia
- Stillbirth & intra-uterine demise
- Polyhydramnios
- Hypertensive disease
- PTL & prematurity
- Caesarean delivery and other interventions
- Neonatal complications – low apgars/ NICU admission
Effects of diabetes on pregnancy

- Almost all these complications improved or eliminated with GOOD GLYCEMIC CONTROL
Background

- Until fairly recently usefulness of screening (and managing) was questioned

  - NICE UK 2005
  - PSTSF US 2008
Background

Testing was originally “screening” for those at risk of later diabetes

1960’s – O’Sullivan first noticed association between gestational diabetes and subsequent Type 2 diabetes

- WHO – based on non-pregnant levels
- ADIPS 1991 - based on expert opinion
Gestational Diabetes

- Should we bother?
Gestational Diabetes

- Should we bother?
- ACHOIS, MFM
Gestational Diabetes

- Should we bother?
- ACHOIS, MFM
- HAPO

HAPO: Incidence of Adverse Outcomes Increases Along Continuum – No Threshold

Benefits of managing maternal hyperglycaemia

- **Maternal**
  - Hypertensive disease/ PET
  - LSCS/operative delivery
  - Birth trauma
  - PPH
  - ACHOIS: QOL & less depression

- **Fetal and neonatal**
  - Macrosomia, Shoulder dystocia
  - Hypoglycaemia
  - Respiratory distress (Insulin inhibits surfactant)

- **Longer term health of mother and child**
Poolsup et.al meta-analysis 2014

- Ten studies involving 3,800 women
- Treatment of GDM significantly reduced risk of:
  - Macrosomia
  - Shoulder dystocia
  - Gestational hypertension

NO increase in SGA babies
No difference in
- *Perinatal/ neonatal mortality*
- Neonatal hypoglycemia
- Pre term birth
- PET
- Caesarean section and induction of labour
Cost effectiveness

- Several studies demonstrate cost-effectiveness of treating GDM, especially across the life-course of the mother

- St Carlos GDM Study Diabetes Care Sept 2014
  - Improved pregnancy outcomes and “markedly reduced healthcare costs”
  - Main savings were due to fewer caesareans & NICU adm.
  - Estimated saving of €14,000/ 100 women
Harms of treating gestational diabetes

- Recent meta-analysis concluded that *harm* of treating GDM was “limited to an increase in resource use and related costs”

- Hartling et al Ann Intern Med May 28 2013
Diabetes Dilemmas
Diabetes dilemmas

- Diabetes and *stillbirth*
- Stillbirth a *rare* but *catastrophic* event
- Related to *suboptimal* glycemic control
- Metabolic/hypoxic & cardiac effects
- Maternal vasculopathy – poor placental perfusion
Stillbirth & GDM

- O’Sullivan in 1960’s noted increased rate of S/B

- *Recent* studies less clear about association
  - due to improved detection and management

- Macrosomia
  - Conflicting results re association
Diabetes dilemmas

- How should we monitor?
  - 2009 NIH: unclear as to best method of screening
Diabetes dilemmas

Dilemma with *ultrasound*

- No accurate method of predicting birthweight
- Birthweight per se not the only factor in the nature of labour and birth
- “False positives” – especially in high BMI women
- Growth *impairment*
Diabetes dilemmas

- **CTG monitoring**

  No benefit in NIRGDM

  May alter management in Type 1&2, IRGDM, and those with *secondary complications*

  ? When to start and how often

  ? Cost implications / “false positives”
Decision re birth

• Timing of birth?

• Consensus when sugars optimal, normal fetal weight & no other complication – IOL @ 39- 40/40
“Pros” of Induction

- Avoid late stillbirth
- Avoid complications of continued fetal growth
Decision re birth

- **Rosenstein et al** AmJOG 2012 – 39 weeks optimal – lower infant mortality with IOL than with expectant Mx – R.R 1.8 (but 39 weeks < 36 weeks)

Comment that *absolute* risk of S/B is low.

NNTD to prevent one excess death at 39 weeks is 1500

?impact on cost, C/S rates, neonatal & maternal outcomes
Decision re birth


- IRGDM: IOL 38-39 weeks vs expectant mx (40 weeks) ➔

  shoulder dystocia 1.4% vs 10%
“Cons” of Induction

- Failed induction
  - *Primiparous* women

- Fetal lung maturity

- “Interventional”
  - Need for CTG monitoring, IV line etc
Decision re birth

- **Mode** of birth
  - Shoulder dystocia more likely at a given weight with diabetic vs non-diabetic pregnancies
    - EFW 4,500g - >500 caesars to prevent brachial plexus injury
    - EFW 4,000g - >900 caesars
  - ACOG guidelines: offer C/S if EFW > 4,500g
Caesarean section

- Higher risk of neonatal complications - esp HMD
  - Insulin inhibits surfactant
  - Longer recovery time, infection etc
- Future pregnancies
- ?Microbiome
Individualize management

- Parity, previous C/S
- Glycemic control
- EFW
- Favourability of cervix
- Co-morbidities
  - BMI
  - Maternal age, IVF
- Patient preference
Postpartum

- Follow up
  - GTT ?@ 6/52

- Contraception
  - LARC’s

- Encourage breast feeding
  - May reduce likelihood of developing T2DM

- Postnatal depression more common
Epigenetics of gestational diabetes

- Intrauterine hyperglycemia/hyperinsulinemia is associated with lifelong risks:
  - Obesity and metabolic disease
  - Cardiovascular disease and hypertension
  - Malignancy
“Take-Home” Messages

- Multi-disciplinary and *pre-pregnancy* care
  
  “societal” problem with lifestyle, obesity

- Good *glycemic* control

- *Individualise* obstetric management plan
Thank you

“IT IS BETTER TO KEEP YOUR MOUTH CLOSED AND LET PEOPLE THINK YOU ARE A FOOL THAN TO OPEN IT AND REMOVE ALL DOUBT.”

MARK TWAIN

© Lifehack Quotes
Obesity and Pregnancy
BuMP Clinic

Dr. Farah Sethna

Capital Health Education Day
14 October 2017
“Obesity is arguably the one of the biggest challenges facing maternity services today. It is a challenge not only because of the magnitude of the problem…but also because of the impact that obesity has on the women’s reproductive health and that of their babies”
DOHD
The first nine months shape the rest of your life
Take a moment to reflect upon your individual practice?

Do you routinely discuss lifestyle changes, contraception / conception plans with women of child-bearing age whom you encounter?
Does the woman’s BMI influence the likelihood of you having this conversation?
Does it alter the advice you give in any way?
Once pregnant, what information do you provide the overweight / obese woman about the impact of her weight on her pregnancy?
Learning Objectives

1. To develop an awareness of the prevalence of maternal obesity

2. To develop knowledge of the influence of obesity on pregnancy outcomes

3. To develop a practical approach for managing the obese parturient

4. To introduce the BuMP clinic
Defining Maternal Obesity

No pregnancy specific categories
BMI may be inaccurate for assessing healthy weight in certain groups (WHO)
Health Effects of Overweight and Obesity in 195 Countries over 25 Years

The GBD 2015 Obesity Collaborators
Globally: The Top Ten

1. American Samoa: 74.6
2. Nauru: 71.1
3. Cook Islands: 63.4
4. Tokelau: 63.4
5. Tonga: 57.6
6. Samoa: 54.1
7. Palau: 48.9
8. Kiribati: 46
9. Marshall Islands: 45.4
10. Kuwait: 42
World’s Fattest Countries

Overall percentage of obesity in 100 most populated nations

- Saudi Arabia
- Egypt
- United States
- South Africa
- Iraq
- Mexico
- United Arab Emirates
- Australia
- Turkey
- Chile
Australia

Seventeen in ten Australian men are overweight or obese.

One in two women are overweight or obese.

One in four children are overweight or obese.

These health stats are from the Australian Bureau of Statistics National Health Survey 2014-15.
Prevalence of Maternal Obesity
Centenary Hospital

![Bar chart showing the prevalence of maternal obesity from 2010 to 2016. The chart is color-coded to indicate different BMI categories: ≥50, ≥40 and <50, ≥35 and <40, and ≥30 and <35.](image)
Maternal Obesity Across Australia

Maternal BMI ≥ 40 Kg/m²

Average for all hospitals was 3.23%

3.27% amongst Level 6 hospitals

Maternal BMI > 50 Kg/m² or weight > 140 Kg (at any point in pregnancy)

Prevalence 2.1 per 1000 women giving birth in Australia in 2010 (n=370)

WA>QLD>NSW

BMI 52.8 Kg/m² (40.9 - 79.9 Kg/m²)

Weight 156 Kg (108 - 204 Kg)
<table>
<thead>
<tr>
<th>Risk to Mother</th>
<th>Risk to Baby</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Depression and anxiety</strong>&lt;br&gt; Anxiety OR 1.41; PND OR 1.30; Depression in pregnancy OR 1.43</td>
<td>↑ Risk congenital anomaly</td>
</tr>
<tr>
<td><strong>GDM</strong>&lt;br&gt; Risk increased by 0.82% with each 1Kg/m² increase in BMI (3.76 increase on average)</td>
<td>x2-3 fold increase macrosomia</td>
</tr>
<tr>
<td><strong>Gestational HTN</strong>&lt;br&gt; OR 2.5-3.2</td>
<td>Increased risk SGA</td>
</tr>
<tr>
<td><strong>PET</strong>&lt;br&gt; Double risk with each 5-7Kg/m² increase in BMI</td>
<td>↑ Lifetime risk of DM, heart disease, obesity</td>
</tr>
<tr>
<td><strong>VTE</strong></td>
<td>X2 fold risk FDIU in 3rd trimester</td>
</tr>
<tr>
<td><strong>Sleep disordered breathing</strong></td>
<td>x1.5-2 fold increase in risk of <strong>spontaneous</strong> preterm delivery, dose-dependent by BMI</td>
</tr>
<tr>
<td><strong>Prolonged pregnancy</strong>&lt;br&gt; Double risk (&gt;41 wk)</td>
<td>x1.5-2.7 fold increase in risk of <strong>induced</strong> preterm delivery, dose-dependent by BMI</td>
</tr>
</tbody>
</table>
Pregnancy may precipitate or exacerbate OSA. CPAP is safe in pregnancy.
Adverse Childhood Events

- May be implicated in obesity
- Can impact upon relationships, compliance with care, intimate examinations, breastfeeding
<table>
<thead>
<tr>
<th>Variable</th>
<th>Non-Obese N (%)</th>
<th>Obese Proportion N (%)</th>
<th>OR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Emotional Abuse</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None to minimal</td>
<td>150 (63.0)</td>
<td>34 (22.7%)</td>
<td>1.00</td>
</tr>
<tr>
<td>Low to moderate</td>
<td>42 (17.7)</td>
<td>10 (23.8%)</td>
<td>1.07 (0.48, 2.34)</td>
</tr>
<tr>
<td>Moderate to severe</td>
<td>46 (19.3)</td>
<td>19 (41.3%)</td>
<td>2.40* (1.19, 4.84)</td>
</tr>
<tr>
<td>Physical Abuse</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None to minimal</td>
<td>172 (72.3)</td>
<td>40 (23.3%)</td>
<td>0.92 (0.32, 2.63)</td>
</tr>
<tr>
<td>Low to moderate</td>
<td>23 (9.7)</td>
<td>5 (21.7%)</td>
<td></td>
</tr>
<tr>
<td>Moderate to severe</td>
<td>43 (18.1)</td>
<td>18 (41.9%)</td>
<td>2.38* (1.18, 4.79)</td>
</tr>
<tr>
<td>Sexual Abuse</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None to minimal</td>
<td>179 (75.2)</td>
<td>42 (23.5%)</td>
<td></td>
</tr>
<tr>
<td>Low to moderate</td>
<td>11 (4.6)</td>
<td>5 (45.5%)</td>
<td>2.72 (0.79, 9.36)</td>
</tr>
<tr>
<td>Moderate to severe</td>
<td>47 (19.7)</td>
<td>25 (31.9%)</td>
<td>1.53 (0.76, 3.09)</td>
</tr>
<tr>
<td>Emotional Neglect</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None to minimal</td>
<td>158 (66.4)</td>
<td>37 (23.4%)</td>
<td></td>
</tr>
<tr>
<td>Low to moderate</td>
<td>51 (21.4)</td>
<td>19 (37.3%)</td>
<td>1.94 (0.99, 3.82)</td>
</tr>
<tr>
<td>Moderate to severe</td>
<td>28 (11.8)</td>
<td>6 (21.4%)</td>
<td>0.89 (0.37, 2.36)</td>
</tr>
<tr>
<td>Physical Neglect</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None to minimal</td>
<td>192 (80.7)</td>
<td>47 (24.5%)</td>
<td></td>
</tr>
<tr>
<td>Low to moderate</td>
<td>19 (8.0)</td>
<td>7 (36.8%)</td>
<td>1.80 (0.67, 4.84)</td>
</tr>
<tr>
<td>Moderate to severe</td>
<td>19 (8.0)</td>
<td>7 (26.3%)</td>
<td>1.37 (0.56, 3.36)</td>
</tr>
</tbody>
</table>

* = p<.05.

Problems During Labour and Birth

- Anaesthetic
- Difficulties monitoring fetal wellbeing
- Unsuccessful IOL
- Operative / assisted delivery
- Perineal trauma / OASIS
- PPH
- Shoulder dystocia
Postpartum Issues

- Infection
- VTE
- Postnatal depression
- Difficulties with breastfeeding
- Weight retention
- SIDS risk if bed-sharing / co-sleeping
Maternal Death

- Obesity is associated with higher odds of maternal death
- Effect primarily manifested through medical comorbidities

Table 2.16: Selected medical conditions and characteristics identified amongst women who died 2012–14

<table>
<thead>
<tr>
<th>Medical condition/characteristic</th>
<th>Direct (n=81) Frequency (%)</th>
<th>Indirect (n=119) Frequency (%)</th>
<th>Total (n=200) Frequency (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body mass index (BMI)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;18</td>
<td>1 (1)</td>
<td>4 (3)</td>
<td>5 (3)</td>
</tr>
<tr>
<td>18–24</td>
<td>27 (33)</td>
<td>50 (42)</td>
<td>77 (38)</td>
</tr>
<tr>
<td>25–29</td>
<td>17 (21)</td>
<td>20 (17)</td>
<td>37 (18)</td>
</tr>
<tr>
<td>≥30</td>
<td>27 (33)</td>
<td>38 (32)</td>
<td>65 (33)</td>
</tr>
<tr>
<td>Missing</td>
<td>9 (11)</td>
<td>7 (6)</td>
<td>16 (8)</td>
</tr>
</tbody>
</table>

MBRRACE-UK Maternal Report 2016
Maternal Weight

- BMI should be measured at the first antenatal consultation and should not be reflective of a self-reported weight and height

“I maintained my weight at 290 lb throughout pregnancy”
# Healthy Weight Gain in Pregnancy

## ISOM 2009 Guidelines

**BMI and the risk of complications during pregnancy.**

<table>
<thead>
<tr>
<th>BMI</th>
<th>Weight class</th>
<th>Risk of complications during pregnancy</th>
<th>Singleton pregnancy: Total weight-gain goal during pregnancy</th>
</tr>
</thead>
<tbody>
<tr>
<td>18.5 or less</td>
<td>Underweight</td>
<td>Increased risk</td>
<td>12.5–18 kg</td>
</tr>
<tr>
<td>18.5–24.9</td>
<td>Normal range/healthy weight</td>
<td>No increased risk</td>
<td>11.5–16 kg</td>
</tr>
<tr>
<td>25–29.9</td>
<td>Overweight</td>
<td>No increased risk</td>
<td>7–11.5 kg</td>
</tr>
<tr>
<td>30–34.9</td>
<td>Obese class 1</td>
<td>Mildly increased risk</td>
<td>5–9 kg</td>
</tr>
<tr>
<td>35.0–39.9</td>
<td>Obese class 2</td>
<td>Moderately increased risk</td>
<td>5–9 kg</td>
</tr>
<tr>
<td>40 or more</td>
<td>Obese class 3</td>
<td>Severely increased risk</td>
<td>5–9 kg</td>
</tr>
</tbody>
</table>

The caloric requirement only goes up in the third trimester.
Pregnancy weight gain chart for BMI ≥ 25kg/m²

Congratulations
Pregnancy is an exciting time for you and your family. It is a great time to focus on your health. Weight gain is an important part of any healthy pregnancy. Gaining too much weight or not enough weight can affect your health and the health of your baby, not just during pregnancy but also for many years to come.

Monitoring your weight during pregnancy can help keep you on track for a healthy weight gain. This weight gain chart can be customised just for you. Bring this chart with you to each visit to discuss with your health care provider what your weight gain goals for this pregnancy should be and to monitor your progress.

Why your weight is important?
Women who are underweight or do not gain enough weight have a risk of preterm birth and a baby small for its gestational age. Women who are overweight or gain too much weight during pregnancy have a higher risk of:
- high blood pressure
- gestational diabetes
- a large baby (macrosomia)
- caesarean sections
- difficulty losing weight after their baby is born. This can also increase your long-term risk of diabetes, heart disease and some cancers
- a baby who is overweight in childhood and as an adult.

How much weight gain is recommended?
The weight you should aim to gain depends on what your weight (and body mass index - BMI) was before you became pregnant. BMI is the number used to work out the recommended amount of weight gain for you. If you were given a hand-drawn record from antenatal clinic you may find your pre-pregnancy BMI in here. If not ask your health care provider to help you work it out.

Following is the recommended amount of weight to gain based on BMI numbers. Choose the weight gain range that matches your pre-pregnancy BMI:

- **Pre-pregnancy BMI**
  - 25 to 29.9 kg/m²
  - Gain 7 to 11.5 kg
  - Pre-pregnancy BMI
  - Above 30 kg/m²
  - Gain 5 to 9 kg

If you are having twins or triplets the recommendations are a bit more. Talk with your health care provider about how much is right for you.

How to use this tracker:
Every pregnancy is different. What worked for your last pregnancy or for your mum may not work for this pregnancy. This tracker will help you work with your health care provider to customise a weight gain plan that is right for you.

1. Write down your weight before pregnancy in the two spaces provided: first in the box inside the chart and second at the starting point at the left hand side of the chart.
2. Ask your health care provider two things: your height, and your pre-pregnancy BMI; you can work this out yourself using a BMI calculator found online at http://www.health.gov.au/internet/healthyactive/publishing.nsf/Content/your-bmi
3. Starting from your pre-pregnancy weight add 1kg and write this number on the line above where you wrote your pre-pregnancy weight, follow this pattern until you reach the top of the chart and all the empty lines are filled.
4. Start recording your weight as early as you can. Place a dot at your current weight and your week gestation. Connect the dots every week to track and compare your weight with the recommended weight gain chart lines for you.
5. Discuss your progress when you have a health care visit. If you are falling above or below the chart turn over for some quick tips to get back on track.

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Version: 1 Effective date: 12/2014 Review date: 11/2017

Great state. Great opportunity.
And a plan for the future.
Physical Activity

Lifestyle interventions are safe in pregnancy and help control weight gain.
Pregnancy can be a powerful motivator for behaviour change.
Available Information Leaflets
Aneuploidy Screening and Imaging
Concentrations of PAPP-A and βhCG are Affected by Maternal Weight

Not adjusting for maternal weight can lead to misinterpretation of:

- Combined first trimester screening result
- PET screening risk assessment
- Women with a low PAPP

<table>
<thead>
<tr>
<th>PAPP-A level</th>
<th>IUGR (birth weight &lt;10th centile)</th>
<th>Delivery &lt;34 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;0.45 MoM (5th centile)</td>
<td>14% risk (odds ratio 2.7)</td>
<td>2.3% risk (odds ratio 2.3)</td>
</tr>
<tr>
<td>&lt;0.29 MoM (1st centile)</td>
<td>24% risk (odds ratio 5.4)</td>
<td>2.5% risk (odds ratio 2.5)</td>
</tr>
</tbody>
</table>
Screening with cfDNA

↑Maternal weight  ↓fetal fraction

↑GA ↑fetal fraction

Risk of non-reportable test due to insufficient fetal fraction

### Table 3

Adjusted odds ratios for “no call” compared to normal-weight women stratified by body mass index class

<table>
<thead>
<tr>
<th></th>
<th>Underweight N = 27</th>
<th>Normal weight N = 965</th>
<th>Overweight N = 686</th>
<th>Class I obesity N = 347</th>
<th>Class II obesity N = 192</th>
<th>Class III obesity N = 168</th>
</tr>
</thead>
<tbody>
<tr>
<td>“No call”, n (%)</td>
<td>1 (0.95)</td>
<td>17 (16.19)</td>
<td>27 (25.17)</td>
<td>20 (19.05)</td>
<td>20 (19.05)</td>
<td>20 (19.05)</td>
</tr>
<tr>
<td>aOR (95% CI)</td>
<td>2.28 (0.28—18.73)</td>
<td>Referent</td>
<td>2.31 (1.21—4.42)</td>
<td>3.66 (1.76—7.61)</td>
<td>7.52 (3.60—15.74)</td>
<td>8.55 (4.16—17.56)</td>
</tr>
<tr>
<td>P&lt;sub&gt;trend&lt;/sub&gt;</td>
<td>&lt;.01</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

aOR, adjusted odds ratio; CI, confidence interval.
* Adjusted for maternal age.


**Important pretest counselling point**
Scanning: A Real Challenge!

Include BMI on all requests for ultrasound imaging
Visualisation

More difficult in obese women
Linearly correlated with degree of obesity

*There was a significant decrease in proportion of pregnancies with complete visualization of fetal anatomy with increasing BMI category, P < 0.001.*

Visualisation in obese women: linearly correlated with degree of obesity.

Journal of Ultrasound in Medicine 2009; 28(8): 1025-1030
Increased Risk of Congenital Anomalies

- Neural tube defects
- Anorectal atresia
- Cardiac defects
- Orofacial clefts
- Limb reduction
- Hydrocephalus

Persson et al. BMJ 2017;357
Recommendations

• In obese pregnant women, it is advisable to perform an early anatomy evaluation, ± transvaginally, at 12–14 weeks of gestation.

• Gently inform the patient and her partner that maternal obesity/being overweight and/or high weight gain in pregnancy are all associated with decreased image resolution and so a reduced detection rate for fetal anomalies.
Incomplete Study: What Next?

Organise a rescan 2-3 weeks following incomplete FAS

Warn the woman that visualisation may remain suboptimal

The Fetal Medicine team cannot work miracles

<table>
<thead>
<tr>
<th>anatomical structure</th>
<th>Mean GA (weeks)</th>
<th>Suboptimal visualization rate (%) for BMI category:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Normal weight &lt; 30 kg/m²</td>
</tr>
<tr>
<td>Heart</td>
<td>19</td>
<td>19</td>
</tr>
<tr>
<td>Heart; rescan</td>
<td>21</td>
<td>2</td>
</tr>
<tr>
<td>Craniospinal</td>
<td>36</td>
<td>29</td>
</tr>
</tbody>
</table>

Adapted from Hendler *et al.*34, 35. *P < 0.001* for all differences vs. normal weight (BMI < 30 kg/m²).

* Rate of suboptimal visualization at recall ultrasound 2 weeks later35.
† Includes cerebellar diameter and posterior fossa, midline, cavum septi pellucidi, lateral ventricle atrial width and spine. GA, gestational age.
Third trimester growth scan required to aid detection of late onset FGR or macrosomia
Blood Pressure
Blood Pressure Assessment

Appropriate cuff

Validated automated device, calibrated at regular intervals

Box 1. Automated devices validated for use in pregnancy (including pre-eclampsia)

- OMRON MIT Elite
- OMRON MIT
- OMRON Hem 705CP
- OMRON M7
- Microlife WatchBP Home
- Microlife BP 3BTO-A
- Microlife BP 3AS1-2
- Welch Allyn Spot Vital Signs
- Dinamap ProCare 400

http://www.dableducational.org/sphygmomanometers/devices_1_clinical.html#ClinTable
Blood Pressure Assessment

- Upper arm bare
- Cuff at heart level
- Arm supported
- Back supported
- Feet flat on floor

<table>
<thead>
<tr>
<th>Variance (mmHg)</th>
<th>Cause of Variance</th>
<th>Variance (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>10-40</td>
<td>Cuff is too small</td>
<td>10-40</td>
</tr>
<tr>
<td></td>
<td>Cuff over clothing</td>
<td>10-40</td>
</tr>
<tr>
<td></td>
<td>Back/feet unsupported</td>
<td>5-15</td>
</tr>
<tr>
<td></td>
<td>Legs crossed</td>
<td>5-8</td>
</tr>
<tr>
<td></td>
<td>Not resting 3-5 minutes</td>
<td>10-20</td>
</tr>
<tr>
<td></td>
<td>Patient talking</td>
<td>10-15</td>
</tr>
<tr>
<td></td>
<td>Labored breathing</td>
<td>5-8</td>
</tr>
<tr>
<td></td>
<td>Full bladder</td>
<td>10-15</td>
</tr>
<tr>
<td></td>
<td>Pain</td>
<td>10-30</td>
</tr>
<tr>
<td>1.8/inch</td>
<td>Arm below heart level</td>
<td>1.8/inch</td>
</tr>
<tr>
<td></td>
<td>Arm above heart level</td>
<td>1.8/inch</td>
</tr>
</tbody>
</table>
Supplements, Medications and Vaccinations
Pregnancy After Weight Loss Surgery
Additional Considerations

• Timing of pregnancy following surgery
• Nutritional deficiencies
• Issues related to the prior surgery
• Screening for GDM

Rates of bariatric surgery in women are predicted to increase
Timing

- No firm guidelines
- Stabilisation and optimisation of weight loss
- Minimise effect of nutritional deficiencies

Advise delaying pregnancy for at least 1y following surgery

Screen and treat nutritional deficiencies pre-pregnancy

Caution with oral contraceptives
These women need a dietetics referral

Start your journey to a healthy weight
### Types of Bariatric Surgery

<table>
<thead>
<tr>
<th></th>
<th>LAGB</th>
<th>Gastric Sleeve</th>
<th>RNY Gastric Bypass</th>
<th>Biliopancreatic diversion / DS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Type</strong></td>
<td>Restrictive</td>
<td>Restrictive</td>
<td>Restrictive &amp; Malabsorptive</td>
<td>Restrictive &amp; Malabsorptive</td>
</tr>
<tr>
<td><strong>Risk of Dumping Syndrome</strong></td>
<td>✗</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td><strong>Nutritional Deficiencies</strong></td>
<td>+/-</td>
<td>+/-</td>
<td>++</td>
<td>++</td>
</tr>
</tbody>
</table>
## Nutrient Monitoring in Pregnancy

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>LAGB</th>
<th>Gastric Sleeve</th>
<th>RNY Gastric Bypass</th>
<th>Biliopancreatic diversion / DS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Folate</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Iron</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Vitamin D</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Calcium</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>B1 (Thiamin)</td>
<td></td>
<td></td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>B12</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Vitamin A</td>
<td></td>
<td></td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Vitamin E</td>
<td></td>
<td></td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Vitamin K</td>
<td></td>
<td></td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Zinc</td>
<td></td>
<td></td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Copper</td>
<td></td>
<td></td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Selenium</td>
<td></td>
<td></td>
<td>✓</td>
<td>✓</td>
</tr>
</tbody>
</table>
Pregnancy Risks

• Post Surgery
  ➣ Risk obesity related complications
  ➣ Risk GDM / HT / LFD infants (but risk still higher cf. non obese women)
  ➤ Risk SGA and prematurity (more likely following malabsorptive operation)

• Deflation vs. maintenance of inflation of AGB
  ➣ Risk SGA; ➤ Wt gain and risk of PIH (UKOSS; unpublished)

• Surgical complications
Screening for GDM

- No formal guidelines
- Suggest HbA1C and BGL at booking
- Capillary home glucose monitoring

Risk of early and late dumping syndrome with OGTT
It’s time to change the conversation about obesity and pregnancy.
BuMP

- Multi-professional
- Respectful care throughout pregnancy
- Provision of relevant, honest and timely information
- Emotional support and advice
- Collaborative and woman centered
Availability of Appropriate Equipment

Maintain the woman’s dignity
Avoid embarrassment and injury (physical and psychological)
• Pathology (routine for pregnancy + metabolic +/- micronutrients)
• Imaging (aneuploidy and fetal anomaly screening / fetal growth surveillance)
• Medications, supplements and vaccinations
• Lifestyle advice (weight at every visit)
• Screening & management of medical co-morbidities and pregnancy complications
• Multidisciplinary assessment and review as required (obstetrics / midwifery / anesthesics / sleep physician / dietetics / diabetes educators / physiotherapy / PNMH / lactation consultant)
• Planning for a safe delivery and beyond
Working in Partnership With You

BuMP → Obesity Management Service
Referral
Results (bloods & scans)
Height, Weight, BMI
Co-morbidities
Bariatric surgery / Optifast

Refer women early
Try and avoid the 11th hour referral
TIME FOR QUESTIONS
Thank You For Your Attention
Dr Sumathy Perampalam
Senior Staff Specialist, Endocrinologist
Thyroid Disorders in Pregnancy

Dr Sumathy Perampalam
Department of Endocrinology
Canberra Hospital
Thyroid and Pregnancy

- Thyroid hormone is essential for normal pregnancy and fetal brain development.
- Fetus is dependent on maternal T4 for the first part of pregnancy
Physiologic changes in Pregnancy

• Pregnancy is a stress test for the thyroid.
• Thyroid gland must produce 30-50% more thyroid hormone
• Physiologic changes:
  • Peak HCG at 8-10 weeks -> suppression of TSH
  • Estrogen increases TBG by almost 2 fold → Total T4 and total T3 rise in parallel
  • Increased plasma volume
  • Placental DIO3 activity (iodothyronine deiodinase Type 3 activity)
  • Increased iodine clearance -> doubles the need of iodine
FT4, FT3, Total T4 levels

FIG. 2. Variation in serum levels of thyroid function test and pregnancy-related hormones according to course of gestation. TBG, thyroxine-binding globulin; T4, thyroxine; T3, triiodothyronine; hCG, human chorionic gonadotropin; TSH, thyroid-stimulating hormone. (Adapted from Glinoer and Brent, with permission.)
2017 Guidelines of the American Thyroid Association for the Diagnosis and Management of Thyroid Disease During Pregnancy and the Postpartum

Erik K. Alexander,1,* Elizabeth N. Pearce,2,* Gregory A. Brent,3 Rosalind S. Brown,4 Herbert Chen,5 Chrysoula Dosiou,6 William A. Grobman,7 Peter Laurberg,8,* John H. Lazarus,9 Susan J. Mandel,10 Robin P. Peeters,11 and Scott Sullivan12
What’s new?

• Re-assessment of the normal TSH reference ranges
• Synergistic effect of subclinical hypothyroidism and autoimmunity on adverse pregnancy outcomes.
• Some data on treatment of subclinical hypothyroidism
• Congenital anomalies with anti-thyroid drugs, including Propylthiouracil.
Normal TFTs in Pregnancy

• Local trimester-specific ranges should be used, when available.

• 2011 ATA Guidelines:
  – first trimester TSH: 0.1 to 2.5 mIU/L
  – second and third trimesters upper threshold 3.0mIU/L

• 2017 ATA Guidelines : Recommendation 26:
  – For the first trimester,
    • the lower limit can be reduced by approximately 0.4 mU/L,
    • the upper limit reduced by approximately 0.5mU/L.
  – TSH : 0.1 to 3.5-4.0mIU/L
  – From weeks 7–12, with a gradual return towards the non pregnant range in the second and third trimesters
<table>
<thead>
<tr>
<th>Author, country (reference) (analyzing method)</th>
<th>N</th>
<th>Gestation (week)</th>
<th>TSH, mU/L</th>
<th>FT4, pmol/L (ng/dL)</th>
<th>Population characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Median</td>
<td>2.5th–97.5th</td>
<td>Median</td>
</tr>
<tr>
<td>Bestwick et al., Italy (24) (AutoDELFIA)</td>
<td>5005</td>
<td>&lt;16</td>
<td>1.07</td>
<td>0.04–3.19</td>
<td>9.3</td>
</tr>
<tr>
<td>Bestwick et al., UK (24) (Advia Centaur)</td>
<td>16,334</td>
<td>&lt;16</td>
<td>1.11</td>
<td>0.06–3.50</td>
<td>13.9</td>
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<tr>
<td>Bocos-Terraz et al., Spain (264) (Architect)</td>
<td>481</td>
<td>&lt;14</td>
<td>0.94</td>
<td>0.41–2.63</td>
<td>13.9</td>
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<tr>
<td>Gilbert et al., Australia (271) (Architect)</td>
<td>1817</td>
<td>9–13</td>
<td>0.74</td>
<td>0.02–2.15</td>
<td>13.5</td>
</tr>
<tr>
<td>Lambert-Messertian et al., USA (270) (Immulite 2000)</td>
<td>8351</td>
<td>T1</td>
<td>1.00</td>
<td>0.12–3.37</td>
<td>14.2</td>
</tr>
<tr>
<td>La’ulua et al., USA (139, 265) (Architect)</td>
<td>8415</td>
<td>T2</td>
<td>1.19</td>
<td>0.35–3.35</td>
<td>13.0</td>
</tr>
<tr>
<td>Li et al., China (17) (Cobas Elesys 601)</td>
<td>640</td>
<td>7–12</td>
<td>1.47</td>
<td>0.10–4.34</td>
<td>15.8</td>
</tr>
<tr>
<td>Männisto et al., Finland (266) (Architect i2000)</td>
<td>4333</td>
<td>T1</td>
<td>1.11</td>
<td>0.08–3.54</td>
<td>15.3</td>
</tr>
<tr>
<td>Medici et al., the Netherlands (267) (Vitro ECI)</td>
<td>747</td>
<td>T2</td>
<td>1.37</td>
<td>0.11–4.24</td>
<td>14.6</td>
</tr>
<tr>
<td>Pearce et al., USA (142) (Advia Centaur)</td>
<td>585</td>
<td>&lt;14</td>
<td>1.1</td>
<td>0.04–3.60</td>
<td>2.1&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Quinn et al., Russia (272) (Abbott AxSYM)</td>
<td>380</td>
<td>T1</td>
<td>1.66</td>
<td>0.09–4.67</td>
<td>—</td>
</tr>
<tr>
<td>Springer et al., Czech Republic (268) (ADVIA Centaur)</td>
<td>549</td>
<td>T2</td>
<td>2.00</td>
<td>0.20–4.68</td>
<td>—</td>
</tr>
<tr>
<td>Stricker et al., Switzerland (262) (Architect i2000SR)</td>
<td>4337</td>
<td>9–11</td>
<td>1.21</td>
<td>0.06–3.67</td>
<td>—</td>
</tr>
<tr>
<td>Vaidya et al., UK (274) (Modular E 170)</td>
<td>1089</td>
<td>&lt;12</td>
<td>1.08</td>
<td>0.14–3.19</td>
<td>14.6</td>
</tr>
</tbody>
</table>

Studies were selected according to the following criteria: N ≥ 500, exclusion of thyroid peroxidase antibody (TPOAb)-positive women and availability of data from the manuscript or via personal communication. Iodine status was estimated based on references from article, WHO iodine status reports or from the Vitamin and Mineral Nutrition Information System (VMNIS).
Assessment of thyroid function during pregnancy: first-trimester (weeks 9–13) reference intervals derived from Western Australian women

Rhonda M Gilbert, Narelle C Hadlow, John P Walsh, Stephen J Fletcher, Suzanne J Brown, Bronwyn G Stuckey and Ee Mun Lim

### 2 Demographic and thyroid function details for pregnant women at 9–13 weeks’ gestation*

<table>
<thead>
<tr>
<th></th>
<th>All women</th>
<th>Antibody-positive women</th>
<th>Antibody-negative women (R)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>2155</td>
<td>338</td>
<td>1817</td>
</tr>
<tr>
<td>Age (years)</td>
<td>30.9 (5.3)</td>
<td>30.7 (5.4)</td>
<td>30.9 (5.2)</td>
</tr>
<tr>
<td>Gestation (weeks)</td>
<td>11.2 (1.0)</td>
<td>11.1 (1.0)</td>
<td>11.3 (1.0)</td>
</tr>
<tr>
<td>TSH* (mU/L)</td>
<td>0.78 (0.03, 2.78)</td>
<td>1.13† (0.06, 6.11)</td>
<td>0.74† (0.02, 2.15)</td>
</tr>
<tr>
<td>fT₄ (pmol/L)</td>
<td>13.5 (2.1)</td>
<td>13.0† (2.4)</td>
<td>13.5† (2.0)</td>
</tr>
<tr>
<td>fT₃ (pmol/L)</td>
<td>4.30 (0.67)</td>
<td>4.24† (0.85)</td>
<td>4.35† (0.64)</td>
</tr>
</tbody>
</table>

R = reference group. TSH = serum thyrotropin. fT₄ = free thyroxine. fT₃ = free triiodothyronine.

*Values are mean (SD) except for TSH, which are median (2.5th, 97.5th percentiles). † P < 0.001 from a Wilcoxon test of shift effect between the antibody-positive and antibody-negative groups.
Targeted Screening

• RECOMMENDATION 93: There is insufficient evidence to recommend for or against universal screening for abnormal TSH concentrations in early pregnancy.

• Case Finding:
1. A history of hypothyroidism/hyperthyroidism or current symptoms/signs of thyroid dysfunction
2. Known thyroid antibody positivity or presence of a goiter
3. History of head or neck radiation or prior thyroid surgery
4. Age >30 years
5. Type 1 diabetes or other autoimmune disorders
6. History of pregnancy loss, preterm delivery, or infertility
7. Multiple prior pregnancies (≥2)
8. Family history of autoimmune thyroid disease or thyroid dysfunction
9. Morbid obesity (BMI ≥40 kg/m2)
10. Use of amiodarone or lithium, or recent administration of iodinated radiologic contrast
11. Residing in an area of known moderate to severe iodine insufficiency
Hypothyroidism
Overt Hypothyroidism

• Overt hypothyroidism: low FT4 with high TSH levels.
• Incidence 2 in 1 000
• Negative outcomes in the mother
  • spontaneous miscarriage, gestational hypertension, pre-eclampsia, preterm delivery, still birth
    • 2002 Abalovich et al, who showed that early fetal loss is significantly lower (4%) with thyroxine, in compared with inadequately treated hypothyroidism (31%).
• Negative outcomes in the baby
  • decreased IQ by 7 points in the offspring of hypothyroid mothers
    – Haddow et al. in 1999:
  • impaired psychomotor development
Case 1

- 25 y.o female with Hashimoto’s Thyroiditis diagnosed 5 years ago.
- 7 weeks pregnant.
- TSH = 18mIU/L (0.1-2.5?3.5), FT4 = 7 (9-17)
- Anti TPO +ve
- Diagnosis: Overt Hypothyroidism
Overt Hypothyroidism Management

• Restore euthyroidism as soon as possible.
  – 150 mcg of levothyroxine a day for 3-5 days, and thereafter reducing the dosage 100-150 mcg/day, according to serum TSH and FT4.
  – Repeat TFTs in 2-3 weeks
  – Aim for TSH <2.5 mIU/L
  – Refer to clinic
Subclinical hypothyroidism

• Elevated TSH, normal FT4
  – 3-18% pregnancies, depending definition
  – 60% have autoimmune positive state : TPO+ve

• Definition is not standardised:
  • Population based reference range
  • TSH cut off ? TSH >2.5 vs >4

• Associated with pregnancy adverse effects
• Adverse fetal cognitive outcomes unlikely
• Sparse data on effects of treatment
Case 2

• 25y.o female, G1P0, otherwise well
• 7 weeks pregnant with TSH 3.6
• Repeat TSH 3.2 mIU/L
• Commenced on Thyroxine 50mcg mane
• Referred to Antenatal Endocrine Clinic
• Seen in Antenatal Endocrine clinic at 11 weeks
  – TSH 1.5mIU/L
  – Anti TPO, anti TG negative
• What is the evidence that Thyroxine therapy is beneficial?
<table>
<thead>
<tr>
<th>Pregnancy outcome</th>
<th>Pooled RR [95% CI]</th>
<th>$I^2$ (%)</th>
<th>Studies used for meta-analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pregnancy loss</td>
<td>2.01 [1.66–2.44]</td>
<td>0</td>
<td>(6,7,10–12,14,18–20,35)</td>
</tr>
<tr>
<td>Preterm labor</td>
<td>0.93 [0.58–1.51]</td>
<td>0</td>
<td>(18,32,34)</td>
</tr>
<tr>
<td>Preterm delivery</td>
<td>1.20 [0.97–1.50]</td>
<td>39</td>
<td>(6–8,11–14,18–20,31,33–35)</td>
</tr>
<tr>
<td>Gestational hypertension</td>
<td>1.22 [0.84–1.78]</td>
<td>52</td>
<td>(11,12,14,18,20,21,32,33)</td>
</tr>
<tr>
<td>Preeclampsia</td>
<td>1.30 [1.00–1.68]</td>
<td>0</td>
<td>(12,13,18,21,33,34)</td>
</tr>
<tr>
<td>Gestational diabetes</td>
<td>1.28 [0.90–1.81]</td>
<td>44</td>
<td>(12,14,18,20,21,32–35)</td>
</tr>
<tr>
<td>Placental abruption</td>
<td>2.14 [1.23–3.70]</td>
<td>0</td>
<td>(12–14,18,21,32,34)</td>
</tr>
<tr>
<td>Placenta previa</td>
<td>0.78 [0.19–3.18]</td>
<td>0</td>
<td>(14,18,34)</td>
</tr>
<tr>
<td>PROM</td>
<td>1.43 [1.04–1.95]</td>
<td>9</td>
<td>(8,14,18,32,34,35)</td>
</tr>
<tr>
<td>Caesarean delivery</td>
<td>1.06 [0.94–1.19]</td>
<td>0</td>
<td>(12,13,19,20,31,32)</td>
</tr>
<tr>
<td>IUGR</td>
<td>1.70 [0.83–3.50]</td>
<td>47</td>
<td>(14,20,32,35)</td>
</tr>
<tr>
<td>Low birth weight</td>
<td>1.34 [0.98–1.82]</td>
<td>52</td>
<td>(7,11,12,14,18,19,35)</td>
</tr>
<tr>
<td>Low Apgar score</td>
<td>1.08 [0.71–1.65]</td>
<td>0</td>
<td>(11,19,34)</td>
</tr>
<tr>
<td>Small for gestational age</td>
<td>1.17 [0.65–2.09]</td>
<td>43</td>
<td>(7,19,34,35)</td>
</tr>
<tr>
<td>Neonatal death</td>
<td>2.58 [1.41–4.73]</td>
<td>0</td>
<td>(7,12,18,19,34,35)</td>
</tr>
</tbody>
</table>

RR, relative risk; CI, 95% confidence interval; PROM, premature rupture of membranes; IUGR, intrauterine growth restriction.

Maraka, S et al; SCH in Pregnancy: A Systematic Review and Meta-analysis
THYROID ;Volume 26, Number 4, 2016
FIG. 2. Forest plots of odds ratios and confidence intervals of women comparing thyroid abnormalities according to the risk of miscarriage.

Liu H; Maternal SCH, autoimmunity and miscarriage; A Prospective Cohort Study

THYROID
Volume 24, Number 11, 2014
SCH and Interventional Studies in Pregnancy

• Composite pregnancy outcome improved in TSH>2.5mIU/L and antiTPO +ve women with Thyroxine treatment

• A lower rate of preterm delivery among antiTPO +ve women treated with levothyroxine than among controls, mainly with TSH >4mIU/L (7.1% vs 23.7%).

• No benefit with levothyroxine in SCH/ hypothyroxinaemia with regard to cognitive function in children at 3.5 years of age. TPOab not assessed
SCH and Interventional Studies

- Treatment for subclinical hypothyroidism or hypothyroxinemia between 8 and 20 weeks of gestation did not result in significant differences in neurocognitive outcomes at 5 years.

- 2 randomized clinical trials are currently ongoing:
  - The Thyroid AntiBodies and LEvoThyroxine study (TABLET) trial in the United Kingdom
  - T4Lifetrial in the Netherlands
In HIGH RISK women, check TSH as soon as pregnancy confirmed, with reflex TPOAb if TSH is 2.5-10 mU/L

- TSH <2.5th percentile or < 0.1 mU/L
  - See thyrotoxicosis section

- TSH 0.1-2.5 mU/L
  - No further workup

- TSH 2.5-10 mU/L
  - TPOAb positive
    - TSH 2.5 mU/L - ULRR
      - Consider treatment with levothyroxine
    - TSH ULRR-10 mU/L
      - Treat with levothyroxine
  - TPOAb negative
    - TSH 2.5 mU/L - ULRR
      - No treatment
    - TSH ULRR-10 mU/L
      - Consider treatment with levothyroxine

FIG. 1. Testing for thyroid dysfunction in pregnancy. ULRR, upper limit of the reference range.
SCH and thyroxine treatment

• Recommended thyroxine dosing:
  – TSH 2.5 to 4.0mIU/L, +ve ab \( \rightarrow \) consider Tx (Thyroxine 50mcg mane)
  – TSH >4.0 to 10.0mIU/L, +ve ab \( \rightarrow \) Tx (Thyroxine 75mcg -100mcg mane)
  – TSH 2.5 to 4.0mIU/L, -ve ab \( \rightarrow \) no Tx
  – TSH >4.0 to 10.0mIU/L, -ve ab \( \rightarrow \) Tx (Thyroxine 75mcg -100mcg mane)
    • Controversial, poor evidence
  – TSH>10, irrespective of ab status \( \rightarrow \) Tx (Thyroxine100-150mcg mane)

• Thyroxine dose may be altered, depending on TSH and FT4 performed 4 weekly until 20 weeks and 1-2 times thereafter
• Dose requirements stabilize by 20 weeks
• Post delivery Thyroxine may be ceased if small dose requirement, TFTs re-assessed about 6-8 weeks post partum
Pre-existing Hypothyroidism

• With confirmation of pregnancy,
  – proactive increment of 30 to 50% or doubling the daily dose 2 days per week, aiming for TSH<2.5mIU/L is recommended.

• Further adjustments may be required, depending on the TSH performed 4-6 weekly.

• Post delivery, reduce thyroxine to pre-pregnancy dose and re-check levels about 6 weeks post partum.
ART and subclinical hypothyroidism

• RECOMMENDATION 20
• Subclinically hypothyroid women undergoing IVF or ICSI should be treated with LT4.
• The goal of treatment is to achieve a TSH concentration <2.5 mU/L.

Use of T3 or desiccated thyroxine in pregnancy

- RECOMMENDATION 31
  - The recommended treatment of maternal hypothyroidism is administration of oral LT4. Other thyroid preparations such as T3 or desiccated thyroid should not be used in pregnancy.

- T4 : T3 = 14:1 ratio in human
- T4: T3 =4:1 in desiccated preperations
- This relative excess of T3 leads to supraphysiologic maternal levels of T3 and relatively low levels of T4
- Fetal T3 present in the CNS during pregnancy is derived from maternal T4 actively transported into this space
Isolated Thyroxinaemia

• FT4 mildly low, normal TSH
• Ensure nil pituitary disorder
• Adequate iodine, iron supplementation
• Likely due to FT4 immunoassay with dilution in latter half of pregnancy

• RECOMMENDATION 30
• Isolated hypothyroxinemia should not be routinely treated in pregnancy
• ? Association with neuro behavioural disorders. Watch this space!
Iodine in pregnancy

• Iodine requirement increases by about 50%
  • needs to produce more thyroid hormone
  • renal loss of iodine is exacerbated increased GFR
  • the fetus needs to produce thyroid hormone during the second half of pregnancy

• Prenatal vitamin preparations contain 150 μg/day

• The WHO recommends 250 mcg/d for both pregnant women and lactating women
Hyperthyroidism in Pregnancy
Hyperthyroidism

• Overt hyperthyroidism
  – occurs in 0.1-0.4% of pregnant women
  – serum TSH level below the trimester-specific reference range with elevated levels of fT3 and/or fT4.

• Subclinical hyperthyroidism
  – TSH level below the trimester-specific reference range with normal peripheral thyroid hormone levels.
  – not associated with adverse maternal or fetal outcomes
  – treatment for this condition is not recommended.

Causes of thyrotoxicosis in pregnancy

Panel 1: Causes of hyperthyroidism in pregnancy

<table>
<thead>
<tr>
<th>Excessive TSH-receptor stimulation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Graves’ disease (TSH-receptor autoantibodies)</td>
</tr>
<tr>
<td>Gestational transient thyrotoxicosis (hCG induced)</td>
</tr>
<tr>
<td>Familial gestational hyperthyroidism (TSH-receptor mutation)</td>
</tr>
<tr>
<td>Trophoblastic disease (hCG induced)</td>
</tr>
<tr>
<td>TSH-producing pituitary adenoma</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Autonomous thyroid hormone secretion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multinodular toxic goitre</td>
</tr>
<tr>
<td>Solitary toxic thyroid adenoma</td>
</tr>
<tr>
<td>Genomic activating TSH-receptor mutation</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Destruction of follicles with release of hormone</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subacute (granulomatous, de Quervain’s) thyroiditis (viral infection)</td>
</tr>
<tr>
<td>Painless (silent) thyroiditis (autoimmunity)</td>
</tr>
<tr>
<td>Acute thyroiditis (bacterial infection)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Extrathyroidal sources of thyroid hormone</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overtreatment with thyroid hormone</td>
</tr>
<tr>
<td>Factitious intake of thyroid hormone</td>
</tr>
<tr>
<td>Functional thyroid cancer metastases</td>
</tr>
<tr>
<td>Struma ovarii</td>
</tr>
</tbody>
</table>

TSH—thyroid stimulating hormone; hCG—human chorionic gonadotropin.
Gestational Transient Thyrotoxicosis (GTT)

- GTT is the most frequent cause of hyperthyroidism in the first trimester
- Due to elevated serum hCG levels
  - hyperemesis gravidarum
  - multiple pregnancies
- GTT does not require anti-thyroid drug treatment
- Resolves spontaneously as hCG levels fall after 10-12w of gestation, normal by 14-18 weeks
- Need to distinguish from Graves’ Disease
Graves’ Disease (GD)

• The most common cause of hyperthyroidism in women of reproductive age.
• GD is autoimmune in nature and presence of thyroid receptor antibodies (TRab) is the hallmark.
• Natural history in pregnancy:
  – Early pregnancy - exacerbate GD
  – Latter half of pregnancy - remission of GD
  – Post partum period – exacerbation of GD
  – Due to pregnancy related immunosuppression.
• Graves’ Disease is likely:
  • presence of a diffuse goitre,
  • history of hyperthyroid symptoms prior to pregnancy
  • presence of ophthalmopathy
Figure 1
Incidence of hyperthyroidism before and after giving birth in 403,958 Danish women studied during 1997–2010. Only the woman’s first birth in the period was included. The time periods before, during, and after pregnancy were split into 3-month intervals as indicated. The horizontal broken line indicates the overall incidence of hyperthyroidism in women over the time period (65.0/100,000/year; asterisk indicates statistical significant difference ($P<0.05$) from this average). Reproduced with permission from Andersen SL et al. (12).
Case 2

• 30 year old female 9 weeks pregnant, unwell with a small goitre, family hx of B12 deficiency
  – TSH<0.001 mIU/L (0.1 to 4.0)
  – FT4 =30 pmol/L (10-20)
  – FT3=10pmol/L (3.5-5.5)
• ?Gestational Thyrotoxicosis ?Graves Disease
• Repeat TFTs and TRAb soon
• Refer to Antenatal Endocrine Clinic – urgent
• Consider ATD if thyrotoxicosis with +ve TRAb
Graves’ Disease in Pregnancy

• Graves’ disease in pregnancy
  – Uncontrolled hyperthyroidism
    → adverse pregnancy and maternal outcomes
    - miscarriage, preeclampsia, preterm birth, placental abruption, low birth weights, still birth, maternal cardiac failure and thyroid storm
    - Improved prognosis with control of hyperthyroidism
  – Effect of TRAb crossing placenta → fetal hyperthyroidism
  – Passage of antithyroid drugs across placenta
    → fetal hypothyroidism: antithyroid drugs should be carefully adjusted
    → risk of congenital malformation

TRAb and the fetus

- Fetal hyperthyroidism 1-5%
- Fetal TSH receptors are responsive to TRAb at 20 weeks
- Fetal hyperthyroidism occurs after 20 weeks.
- If TRAb >3x upper limit of normal fetal hyperthyroidism (100% sensitivity; 43% specificity)
- Ultrasound used to assess for signs:
  - fetal hyperthyroidism (fetal tachycardia, accelerated bone maturation, fetal goiter, intrauterine growth restriction, and signs of congestive heart failure)
  - fetal hypothyroidism (goitre)

Anti-thyroid Drugs

• Graves’ Disease in pregnancy
  – Propylthiouracil (PTU) is recommended until 16 weeks and thereafter Carbimazole.
  – lowest possible doses of antithyroid drugs, keep the free T4 in the high-normal to slightly thyrotoxic range. Do not aim to normalise TSH. Fetal thyroid is more sensitive to ATD.
  – serum TSH and free T4 should be assessed every 2-4 weeks until euthyroidism is achieved
  – Short-term use of propranolol will improve symptoms.
  – RAI is contra-indicated

• Carbimazole is associated congenital anomalies:
  – cutis aplasia, choanal, esophageal atresia, omphalocele, dysmorphic facies [11,12].

• Propylthiouracil :
  – Also associated with birth defects: urinary tract and face and neck malformations
  – fulminant maternal hepatotoxicity (0.1- 0.01%)

• In women who are unable to tolerate or resistant to antithyroid drugs, thyroidectomy in the second trimester may be required.
The 8-10% prevalence of birth defects associated with the use of PTU and MMI/CMZ in weeks 6–10 of pregnancy. AOR: 1.4 to 1.6
Fig. 3. Patient 2 at 4 years of age showing broad, flared eyebrows, short, upslanting palpebral fissures, broad nasal bridge, and double inferior eyelids.

Fig. 4. Patient 2 at 4 years of age showing small ears with overfolded helices.
Post partum

• Propylthiouracil and Carbimazole for lactating mothers
  • Carbimazole < 20 mg or PTU < 300 mg been shown to be safe for the infant
  • To be taken after breast feeding, 3 hour prior to next feed
• Neonate may need TFTs and TRAb monitored
Post partum thyroiditis

• 50% TPOAb-positive pregnant women develop abnormal TFT
• % of clinical post partum thyroiditis is unclear
• β-blockers can be used
• Antithyroid drugs will not improve thyroid function
• 30-50% with postpartum thyroiditis develop permanent hypothyroidism during the first year post partum
Pregnancy planning

• Consider TFTs before cessation of contraception in at risk women

• Hypothyroidism
  – Optimise Thyroxine dose prior to cessation of contraception
  – Proactive increment of Thyroxine by 30% with confirmation of pregnancy

• Hyperthyroidism – Graves’ Disease or nodular disease
  – Stabilise thyroid levels prior to pregnancy
Women's Health

In This Section

Breasts and Breastfeeding
Contraception and Sterilisation
Gynaecology
Pregnancy
Women's Health Referrals

See Also

Breast Cancer
Breast Imaging
Female Genital Mutilation (FGM)
High TSH in Pregnancy

Hypothyroidism in pregnancy

**Considerations**

1. If **subclinical hypothyroidism** with TSH > 2.5 mIU/L in the first trimester, treatment with thyroxine is recommended (as below).

2. If **overt hypothyroidism**:
   - Start thyroxine as soon as possible (see below).
   - Recommend referral to an **antenatal endocrinology clinic** or **endocrinologist**.

3. If pre-existing hypothyroidism:
   - Increase **thyroxine dosing** in pregnancy to maintain euthyroidism.
   - Monitor **FT4s** and maintain TSH between 0.5 to 2.5 mIU/L for pre-conception and first trimester.
   - After delivery, reduce thyroxine to pre-pregnancy dose and recheck TSH around 6 weeks post-partum.

**Thyroxine dosing**

1. Advise patient that commonly used calcium and iron supplements in pregnancy can reduce thyroxine absorption and should be taken separately.

2. Start thyroxine dose depending on TSH level:
   - TSH 2.5 to 5.0 mIU/L: 50 micrograms in the morning.
   - TSH 5.1 to 10.0 mIU/L: 75 to 100 micrograms in the morning.
   - TSH > 10 mIU/L:
     - loading dose of 150 micrograms/day for 3 to 5 days, then
     - ongoing dose of 100 to 150 micrograms/day.

   Thyroxine is safe in pregnancy (category A).

3. Check **FTTs** regularly. Monitor closely with the FT4 and FT3 – these may be a more useful guide to thyroid hormone levels in the short term as the TSH can take up to 6 weeks to reach a steady state.

4. Aim for TSH to be < 2.5 mIU/L in the first trimester and ideally preconception, and within trimester-specific normal ranges throughout pregnancy.

5. Individualise **thyroxine management after delivery**.
Referral

- Refer urgently to an antenatal endocrinology clinic or an endocrinologist during pregnancy if:
  - overt hypothyroidism
  - persistently elevated TSH despite thyroxine treatment
  - persistently suppressed TSH
  - Graves' disease (TRAb positive hyperthyroidism)
  - significant nodular thyroid disease, if:
    - symptoms of local invasion
    - significant size
    - suspicious ultrasound features, or
    - confirmed malignancy

- Refer to an antenatal endocrinology clinic or endocrinologist if:
  - Post-partum thyroiditis
  - Post-partum exacerbation of undiagnosed Graves' disease (TRAb positive hyperthyroidism)

- If Graves' disease and the patient is not being seen at a multidisciplinary clinic, also refer to an obstetrician. If TFTs are not stable, refer urgently to an obstetrician as early as possible.
Summary

- 2017 ATA Guidelines
- Normal TSH levels during pregnancy:
  - Upper limit upto 3.5 to 4.0mIU/L
  - Population based normal ranges
- Subclinical hypothyroidism with positive antibody positive state carries higher risk of pregnancy adverse outcomes
- Hyperthyroidism in pregnancy
  - recognise Graves’ Disease
  - Managing risks: uncontrolled Graves’ Disease vs fetal hypothyroidism vs congenital anomalies
- Importance of pregnancy planning in pre-existing thyroid disease.
THANK YOU
Prof Chris Nolan
Director of Endocrinology, ACT Health

Capital Health Network
ACT’s primary health network
Screening for GDM in the ACT

Capital Health Education - 14th Oct 2017

Christopher Nolan
Canberra Hospital & Health Services
Australian National University Medical School
Gestational diabetes mellitus (GDM)

- Increased risk of adverse pregnancy outcomes
- Increased long-term risk of cardio-metabolic disease for mothers
- Increased long-term risk of obesity and diabetes for offspring
History of GDM screening

• Detection of future risk of diabetes in mothers
• Clinical trials in GDM
• Hyperglycaemia and Adverse Pregnancy Outcomes Study (HAPO study)
• Guidelines IADPSG/ADIPS/WHO
• Role of early screening?
GDM- does diagnosis and management improve perinatal outcomes?

- ACHOIS trial
- MFMUN-GDM trial

Crowther CA et al    NEJM 2005; 352: 2477-2486
Landon MB et al      NEJM 2009; 361: 1339-1348
Treatment of GDM Reduces Adverse Outcomes

<table>
<thead>
<tr>
<th>OUTCOME</th>
<th>ROUTINE CARE (N = 510)</th>
<th>INTERVENTION (N = 490)</th>
<th>P</th>
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<tbody>
<tr>
<td>Birth Weight</td>
<td>3482 ± 660</td>
<td>3335 ± 551</td>
<td>&lt; .001</td>
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<tr>
<td>LGA</td>
<td>22%</td>
<td>13%</td>
<td>&lt; .001</td>
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<tr>
<td>Macrosomia</td>
<td>21%</td>
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<td>&lt; .001</td>
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<tr>
<td>Preeclampsia</td>
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<tr>
<td>SGA</td>
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<td>7%</td>
<td>ns</td>
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*Crowther CA, et al. NEJM 352:2477-86, 2005*
Treatment of GDM Reduces Adverse Outcomes

<table>
<thead>
<tr>
<th>Outcome</th>
<th>NICHD RCT</th>
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<tr>
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<td>Not treated</td>
<td>Treated</td>
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<td>BW &gt;90th percentile</td>
<td>14.5</td>
<td>7.1</td>
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<tr>
<td>C-peptide &gt;95th percentile</td>
<td>22.8</td>
<td>17.7</td>
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<tr>
<td>NICU admission</td>
<td>11.6</td>
<td>9.0</td>
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<tr>
<td>Shoulder Dystocia</td>
<td>4.0</td>
<td>1.5</td>
</tr>
<tr>
<td>Preeclampsia</td>
<td>5.5</td>
<td>2.5</td>
</tr>
</tbody>
</table>

*Landon MB et al. NEJM 361:1339-48, 2009*
Diagnostic Criteria and Classification of Hyperglycaemia First Detected in Pregnancy

WHO- Hyperglycaemia first detected in pregnancy

• Should be classified as either:
  – Diabetes mellitus in pregnancy
    • 2006 WHO guidelines for diabetes
  – Gestational diabetes mellitus
    • Any time of pregnancy (75 g OGTT)
      Fasting glucose 5.1-6.9 mmol/L
      1-hr glucose ≥ 10.0 mmol/L
      2-hr glucose 8.5-11.0 mmol/L

Flowchart for Gestational Diabetes ACT (October 2017)

Unless high risk (see below)

ALL Women: GTT at 24-28 weeks

Normal:
- Fasting < 5.1
- 1hr < 10
- 2hr < 8.5

GDM:
- Fasting ≥ 5.1
- 1hr ≥ 10.0
- 2hr ≥ 8.5

No GDM
- Routine antenatal care

GDM
- For management of GDM:
  - ACT: Fax referral with GTT results to Diabetes Service for GDM Education
    (Fax: 6244 3834)
  - NSW: Discuss with GP
**HIGH RISK:**

- BMI ≥ 30 kg/m²
- Maternal age ≥ 40 years
- Family history DM (1st degree relative with diabetes)
- Previous elevated blood glucose
- Ethnicity: Asian, Indian subcontinent, Aboriginal, Torres Strait Islander, Pacific Islander, Maori, Middle Eastern, non-white African
- Previous perinatal loss
- Previous GDM
- Previous macrosomia (birth weight > 4500 g)
- Polycystic Ovarian Syndrome
- Medications: corticosteroids, antipsychotics

Order random BSL with antenatal bloods- if ≥ 7.0 mmol/L - proceed to early OGTT

- **No GDM**
  - Repeat OGTT at 24-28 weeks
  - If no GDM
    - Routine antenatal care

- **GDM**
  - For management of GDM:
    - ACT: Fax referral with GTT results to Diabetes Service for GDM Education
    - NSW: Discuss with GP
    - (Fax: 6244 3834)
Health pathways
Early screening for GDM

• Which test?
• Should criteria be same as for 24-28 wk?
• Does treatment improve pregnancy outcomes?
• Are there risks of early treatment?
• Cost – benefit?
TREATMENT OF BOOKING GESTATIONAL DIABETES MELLITUS STUDY
TOBOGM: RCT

• To test whether treatment of ‘Booking GDM’ will reduce the sequelae of maternal ‘hyperglycaemia’ without increasing the risk of fetal under-nutrition.
TOBOGM Study

- <20 wks gestation
- One or more risk factors for GDM
  - Previous GDM or hyperglycaemia
  - Previous macrosomia/large for gestational age baby
  - High risk ethnicity
  - BMI $\geq 30$ kg/m$^2$
  - 1st degree relative with diabetes
Local PIs: Profs Chris Nolan and Michael Peek

Clinical Trials Coordinator: Lori Grlj

Telep 02 61747586 lori.grlj@act.gov.au
Thank you
Lynelle Boisseau
Credentialled Diabetes Educator, ACT Health Diabetes Service
LEARNING OUTCOMES

Increased knowledge of:

- Services provided by ACT Health Diabetes Maternity team
- Gestational Diabetes Mellitus (GDM) rates in the region
- Referral process for women with GDM and pre-existing diabetes
- Diabetes education specific to women with GDM
- Management goals during pregnancy and postpartum
- Importance of multidisciplinary team approach
- Follow-up postpartum
SERVICES PROVIDED

Gestational diabetes
1. Education
2. Monitor glycaemic control during pregnancy
3. Outpatient management
4. Postpartum considerations

Pre-existing diabetes
1. Preconception counselling
2. Glycaemic control during pregnancy
3. Outpatient management
4. Postpartum considerations
NDSS National Data (as at March 2017)

- Total 34,424
- 103 Women daily
- 34,248 < 40 years
- 3,176 > 40 years
- 29% currently registered GDM required insulin to manage
Data: Gestational  Area: ACT  (as at June 2017)

% of Pop.: 4.1% (16,937 / 413,619)
% Registrants: 4.7% (789 / 16,937)
NDSS Registrant percentages:

<table>
<thead>
<tr>
<th>Diabetes Type</th>
<th>ACT %</th>
<th>National %</th>
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<tr>
<td>Type 2</td>
<td>83.6</td>
<td>87.2</td>
</tr>
<tr>
<td>Type 1</td>
<td>11.1</td>
<td>9.2</td>
</tr>
<tr>
<td><strong>Gestational</strong></td>
<td>4.7</td>
<td><strong>3.0</strong></td>
</tr>
<tr>
<td>Other</td>
<td>0.6</td>
<td>0.6</td>
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</table>
GDM Diagnostic Criteria

ADIPS

• All women should have 75gram OGTT at 24-28 weeks
• One step approach

• GDM diagnosis based on 1 abnormal value
• Fasting PG ≥ 5.1 mmol/L
• 1 hour PG ≥ 10.0 mmol/L
• 2 hour PG ≥ 8.5 mmol/L
Referral for GDM Education

Following diagnosis -> education is vital
Optimal -> within 1/52 of diagnosis
Reduce maternal anxiety
Correct information – up to date
Encourage partner or support person to attend
Challenge of working within a limited timeframe
Referral Process

**Internal**
- ACT Health Diabetes in pregnancy referral form

**External**
- GP or Obstetrician to fax referral
- Diabetes in Pregnancy Service
  - Antenatal clinic 6244 3834
  - Including the referrers contact details

- Email - diabetes.pregnancy@act.gov.au
Gestational Diabetes Mellitus (GDM) Education pathway

Gungahlin Community Health Centre

GDM Group Education Location

Centenary Hospital for Women & Children

Diabetes Educator

Diabetes Dietitian

Diabetes Educator

Diabetes Dietitian

Exercise Physiologist

Dietitian Individual Review
TCH or GCHC

Patient with well controlled GDM or intermittent elevated BGL’s:
Advised to call Diabetes in Pregnancy on ph. 6174 7601 if 3 elevated BGL’s in a 5 day period

Patient likely to require insulin due to elevated BGL’s and will need endocrine registrar review:
Booked into insulin group start Tuesday & Thursday
GDM Management Guidelines

- Team approach
- Self-management education
- Dietary therapy primary strategy
- Glycaemic targets
- Activity levels secondary strategy
- Insulin treatment
Key Components of Education

- Overview of GDM
- Implications for mother and baby
- Home blood glucose monitoring
- Review by Dietitian 1 week post group education
- NDSS – National Diabetes supplies scheme
GDM Management

- Perform Self Monitoring of blood glucose levels, both fasting and 2 hours postprandial

- Glycaemic Targets during pregnancy:
  - cBGL Fasting < 5.3 mmol/L
  - cBGL 2 Hours Post Main Meals < 7.0 mmol/L

- Nutrition counselling

- Physical activity
  20-30 minutes per day, most effective after a meal appropriate for pregnancy
Psychosocial Issues

- Pregnancy journey now altered
- Heightened anxiety and stress at diagnosis
- Impedes ability to learn
- Guilt
- Concern for baby
- Potential separation from baby at birth
- Will my baby have diabetes?
- Cultural considerations
Alerts

- Need to look at the whole picture
- Sometimes clinical scenario does not match GDM
- What to consider
- Blood Glucose Levels – good glycaemic control?
- Self-reported dietary modifications and increased physical activity?
- Clinically Large for Gestational Age (LGA)
- Significant maternal weight gain
## Log Book Record

<table>
<thead>
<tr>
<th>Breakfast</th>
<th>Lunch</th>
<th>Dinner</th>
<th>Before Supper or Bed</th>
<th>Over night</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before</td>
<td>After</td>
<td>Before</td>
<td>After</td>
<td>Before</td>
</tr>
<tr>
<td>4:25:6</td>
<td>6.3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4:55:8</td>
<td>6.2</td>
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<td>6.6</td>
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<tr>
<td>4:16:0</td>
<td>6.4</td>
<td>6.0</td>
<td></td>
<td></td>
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<tr>
<td>4:05:4</td>
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<td>5.8</td>
<td></td>
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</tr>
<tr>
<td>4:65:8</td>
<td>6.5</td>
<td>6.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4:56:1</td>
<td>6.0</td>
<td>6.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4:36:0</td>
<td>6.2</td>
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</table>
### Monitoring Blood Glucose

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<tr>
<td>Before</td>
<td>After</td>
<td>Before</td>
<td>After</td>
<td>Remains</td>
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<td>4.85</td>
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<td>4.56</td>
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<td>6.6</td>
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<td>4.36</td>
<td>7.5</td>
<td>6.0</td>
<td></td>
<td>LUNCH OUT</td>
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<td>6.3</td>
<td>8.2</td>
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<td>PIZZA FOR DINNER</td>
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<td>5.26</td>
<td>6.6</td>
<td>8.0</td>
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<td>CHINESE TAKEAWAY</td>
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<td>5.37</td>
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<td>6.2</td>
<td></td>
<td>CAFE BREAKFAST</td>
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<td>4.56</td>
<td>4.3</td>
<td>5.4</td>
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<td>WALK AFTER BREAKFAST</td>
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### Glucose: Logbook/table

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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Average (0)</td>
</tr>
</tbody>
</table>
CAN I HAVE A LITTLE TASTE?
I AM EATING FOR TWO NOW.

NOT THE GREEN BEANS.
GO FOR THE PIE! THE PIE!
Postpartum care

- Women with GDM should have an OGTT 6-12 weeks postpartum
- Follow-up appointment with GP
- Discuss – lifestyle issues, weight management, diet, exercise, future pregnancy, contraception
- All women should be encouraged to breastfeed, since this may reduce offspring obesity, especially in the setting of maternal obesity
- Metformin may be used when breastfeeding
Conclusion

- When a pregnancy is complicated by diabetes a multidisciplinary team approach provides the best care for a mother and her baby to achieve an optimal outcome
Useful Websites

• http://adips.org/
Questions
Nutrition approach to GDM

Rosemary Young APD CDE
Dietitian, ACT Health Diabetes Service
Learning Objectives

• Increased understanding of the usual nutrition education and review provided to women with GDM in ACT
• Review the nutritional requirements of pregnancy
• Develop awareness of the evidence around diet advice for GDM
Our Nutrition Education...
Overview.

Nutrition Education occurs in a group. Topics include:

• Expected weight gain in pregnancy
• Nutritional requirements of pregnancy
• Food safety
• Carbohydrates - how much / distribution / how to count
• Glycaemic index
• Need to minimise dietary fat
Teaching CHO Estimation

- 1/3 - 1/2 cup cooked pasta
- ¼ - 1/3 cup cooked rice

= 2 smaller fruits e.g. mandarins, kiwifruits or nectarines
What “cup” did you use for your third cup of rice?

“And that’s how you determine the number of net carbs per serving.”

Source: DIABETease
The answers..........

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<tr>
<th>Food</th>
<th>Grams of Carbohydrate</th>
<th>Grams of Fat</th>
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<tr>
<td>Domino’s® Pizza Traditional Supreme Classic Crust</td>
<td>165</td>
<td>44</td>
</tr>
<tr>
<td>McDonalds BLT McMuffin®</td>
<td>26.8</td>
<td>6.5</td>
</tr>
<tr>
<td>KFC ® Chicken Salad Twister [like a wrap]</td>
<td>44.6</td>
<td>4.4</td>
</tr>
<tr>
<td>Hungry Jacks® Chicken Royale Burger</td>
<td>42</td>
<td>26.7</td>
</tr>
<tr>
<td>Subway® Foot long Chicken Schnitzel</td>
<td>92.6</td>
<td>19.8</td>
</tr>
<tr>
<td>McDonald’s – Big Mac®, large fries and Medium coke</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Big Mac 37.4 g CHO</td>
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</tr>
<tr>
<td></td>
<td>Large Fries 41.7 g CHO</td>
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<td>Medium Coke 36 g CHO</td>
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<td></td>
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<tr>
<td></td>
<td>Big Mac 28.6 g fat</td>
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<tr>
<td></td>
<td>Large Fries 18.5 g fat</td>
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<tr>
<td></td>
<td>Total fat = 47.1</td>
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</tr>
<tr>
<td>Banana Bread</td>
<td>56.1</td>
<td>16.4</td>
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<tr>
<td>Sushi large piece</td>
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<tr>
<td>Gloria Jeans large skim latte</td>
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<td>0.4</td>
</tr>
<tr>
<td>Boost Juice All Berry Bang medium smoothie 450 ml</td>
<td>55.6</td>
<td>3.9</td>
</tr>
<tr>
<td>Muffin Break Blueberry Muffin</td>
<td>66.4</td>
<td>21.1</td>
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<tr>
<td>Wok in a Box Singapore Noodles Regular serving 380 g</td>
<td>144.8</td>
<td>17.1</td>
</tr>
<tr>
<td>Subway 6” Veggie Delite no dressing</td>
<td>37.7</td>
<td>2.3</td>
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<tr>
<td>Kebab Felafel with salad</td>
<td>62.9</td>
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# Carbohydrate Distribution

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<td>g</td>
<td>g</td>
<td>g</td>
</tr>
</tbody>
</table>

- If eat 7.30
- If eat 12.30
- If eat 6.30
- Test 9.30
- Test 2.30
- Test 8.30
After Group Education

• Home blood glucose monitoring 4 x per day – fasting and 2 hours post prandial. Record BGL’s in the monitoring diary provided.
• Keep food diary and look for response to different foods.
• Return for individual review appointments following week with the dietitian.
The review with the dietitian

Consider food, beverage and nutrient intake with attention to:

• Types and amounts of carbohydrate, fat and protein foods and nutritional requirements pregnancy
• Meal and snack patterns including frequency, duration and serving sizes.
• Pregnancy related issues such as hyperemesis, heartburn and constipation that may affect or be improved by food choices
• Cultural and psychosocial considerations
• Factors affecting access to food e.g. lack of money
Old Habits ..........
Nutritional requirements..
Pregnant women have altered nutritional requirements

In Australia we are guided by the NHMRC Australian Dietary Guidelines when educating patients on an adequate diet.

Food modelling based on the NRVs and DGs has produced for us this a guide with recommended number of serves from each food group.
<table>
<thead>
<tr>
<th>Food Group</th>
<th>Percentage Increase</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protein (46 → 60 g/day)</td>
<td>10.00%</td>
</tr>
<tr>
<td>Dietary fibre (25→28 g/day)</td>
<td>20.00%</td>
</tr>
<tr>
<td>Linoleic acid (8 →10 g/day)</td>
<td>30.00%</td>
</tr>
<tr>
<td>α-Linoleic acid (0.8 →1.0 g/day)</td>
<td>40.00%</td>
</tr>
<tr>
<td>total LC n-3 (90→115 mg/day)</td>
<td>50.00%</td>
</tr>
<tr>
<td>Vitamin A (700→800 µg/day retinol equivalents)</td>
<td>60.00%</td>
</tr>
<tr>
<td>Thiamin (1.1→1.4 mg/day)</td>
<td>10.00%</td>
</tr>
<tr>
<td>Riboflavin (1.1 →1.4 mg/day)</td>
<td>20.00%</td>
</tr>
<tr>
<td>Niacin (14→18 mg/day niacin equivalents)</td>
<td>30.00%</td>
</tr>
<tr>
<td>Vitamin B6 (1.3→1.9 mg/day)</td>
<td>40.00%</td>
</tr>
<tr>
<td>Vitamin B12 (2.4→2.6 ug/day)</td>
<td>50.00%</td>
</tr>
<tr>
<td>Folate (400→600 ug/day as dietary folate equivs)</td>
<td>60.00%</td>
</tr>
<tr>
<td>Pantothenic Acid (4→5 mg/day)</td>
<td>10.00%</td>
</tr>
<tr>
<td>Biotin (25→30 ug/day)</td>
<td>20.00%</td>
</tr>
<tr>
<td>Choline (425→440 mg/day)</td>
<td>30.00%</td>
</tr>
<tr>
<td>Vitamin C (45→60 mg/day)</td>
<td>40.00%</td>
</tr>
<tr>
<td>Iodine (150→220 ug/day)</td>
<td>50.00%</td>
</tr>
<tr>
<td>Iron (18→27 mg/day)</td>
<td>60.00%</td>
</tr>
<tr>
<td>Magnesium (320→360 mg/day)</td>
<td>10.00%</td>
</tr>
<tr>
<td>Molybdenum (45→50 ug/day)</td>
<td>20.00%</td>
</tr>
<tr>
<td>Selenium (60→65 ug/day)</td>
<td>30.00%</td>
</tr>
<tr>
<td>Zinc (8→11 mg/day)</td>
<td>40.00%</td>
</tr>
<tr>
<td>Chromium (25→30 ug/day)</td>
<td>50.00%</td>
</tr>
<tr>
<td>Copper (1.2→1.3 mg/day)</td>
<td>60.00%</td>
</tr>
</tbody>
</table>
A diet with recommended serves from all food groups

<table>
<thead>
<tr>
<th>Breakfast</th>
<th>M/Tea</th>
<th>Lunch</th>
<th>A/Tea</th>
<th>Dinner</th>
<th>Supper</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Food</strong></td>
<td><strong>Quantity</strong></td>
<td><strong>Food</strong></td>
<td><strong>Quantity</strong></td>
<td><strong>Food</strong></td>
<td><strong>Quantity</strong></td>
</tr>
<tr>
<td>Sanitarium Weet-Bix</td>
<td>2 biscuits</td>
<td>Arnott’s Vita-Weat</td>
<td>4 crackers</td>
<td>2 slices</td>
<td>200 g</td>
</tr>
<tr>
<td>Lowfat milk</td>
<td>0.5 cup</td>
<td>Margarine</td>
<td>2 teasp</td>
<td>Fresh blueberry</td>
<td>¼ cup</td>
</tr>
<tr>
<td>Banana</td>
<td>Half large</td>
<td>Tuna, canned in brine, drained</td>
<td>Small can</td>
<td>Pasta boiled</td>
<td>1 ½ cups</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lettuce, common</td>
<td>1 cup</td>
<td>Pasta sauce tomato based</td>
<td>0.5 cup</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Raw tomato</td>
<td>1 med</td>
<td>Steamed broccoli</td>
<td>1/2 cup</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mayonnaise</td>
<td>2 teasp</td>
<td>Carrot</td>
<td>½ cup</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Apple</td>
<td>1</td>
<td>Green beans</td>
<td>½ cup</td>
</tr>
</tbody>
</table>

~45 g CHO: 15 g CHO | 45 g CHO | ~23 g CHO | ~65 g CHO | 22 g CHO

<table>
<thead>
<tr>
<th>AGHE serves</th>
<th>Breads &amp; Cereals</th>
<th>Fruit</th>
<th>Vegetables</th>
<th>Protein</th>
<th>Dairy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>8 ½</td>
<td>2</td>
<td>5</td>
<td>3.5</td>
<td>3</td>
</tr>
</tbody>
</table>
## What about vegetarian Indian style?

<table>
<thead>
<tr>
<th>Breakfast</th>
<th>M/Tea</th>
<th>Lunch</th>
<th>A/Tea</th>
<th>Dinner</th>
<th>Supper</th>
</tr>
</thead>
<tbody>
<tr>
<td>Food</td>
<td>Quantity</td>
<td>Food</td>
<td>Quantity</td>
<td>Food</td>
<td>Quantity</td>
</tr>
<tr>
<td>Bread, roti</td>
<td>2 piece</td>
<td>Apple</td>
<td>1 med</td>
<td>Rice Basmati cooked</td>
<td>2 cups</td>
</tr>
<tr>
<td>Boiled egg</td>
<td>2</td>
<td>Almonds</td>
<td>15</td>
<td>Lentil curry</td>
<td>1 cup</td>
</tr>
<tr>
<td>Eggplant</td>
<td>½ cup</td>
<td>Oil</td>
<td>2 teasp</td>
<td>Peanut butter</td>
<td>1 tbsp</td>
</tr>
<tr>
<td>Oil</td>
<td>2 teasp</td>
<td>Carrot</td>
<td>1 med</td>
<td>Vegetable curry</td>
<td>1 cup</td>
</tr>
<tr>
<td>Okra cooked</td>
<td>0.25</td>
<td>Spinach</td>
<td>½ cup</td>
<td>Paneer</td>
<td>100 g</td>
</tr>
<tr>
<td>Milk FC</td>
<td>1 cup</td>
<td>Yoghurt regular</td>
<td></td>
<td>Oil</td>
<td>2 teasp</td>
</tr>
<tr>
<td>~75 g CHO</td>
<td>15 g CHO</td>
<td>~130 g CHO</td>
<td>30 g CHO</td>
<td>90 g CHO</td>
<td>15 g CHO</td>
</tr>
</tbody>
</table>

### AGHE serves
- **Breads & Cereals**: 13
- **Fruit**: 1
- **Vegetables**: 7
- **Protein**: 3
- **Dairy**: 2 ½
The Evidence....
AND Gestational Diabetes (2016) Evidence-Based Nutrition Practice Guidelines

• Refer to a Dietitian [strong]
• Assess the food and nutrition-related history of women [consensus]
• Weigh and measure [consensus]
• Assess biochemistry and SMBG [consensus]
• Provide medical nutrition therapy (MNT) that includes an individual nutrition prescription and nutrition counselling [strong]
• Frequency and duration of MNT minimum 3 visits [consensus]
• Individualise KJ prescription so adequate for growth of baby and maternal health and weight gain [fair]
• Daily minimum of 175g CHO, 71g protein (or 1.1g per kg per day) and 28g fibre [consensus]
• Individualize both the amount and type of CHO for women with GDM [fair]
• Distribute the total calories and carbohydrate (CHO) into smaller meals and multiple snacks per day [consensus]
The ‘best’ diet for GDM?

2017 Cochrane review

• 19 RCT trials involving 1398 women with GDM and their babies.

• Ten different diet comparisons included

• DASH diet ONLY standout

“Dietary advice is the main strategy for managing GDM, however it remains unclear what type of advice is best.”
True or False?

• Women with GDM need to avoid carbohydrate
• Avoiding CHO will avoid the need for insulin
• The woman with GDM only needs to ‘eat healthy’ until delivery.
• Obese women with GDM need to lose weight in pregnancy.
Healthy Eating during your Pregnancy can be found at: https://www.eatforhealth.gov.au/guidelines

Nutrient reference values can be found at: https://www.nrv.gov.au/


Academy of Nutrition and Dietetics http://www.eatrightpro.org/

GDM Evidence Based Nutrition Practice Guidelines from AND Evidence Analysis library can be found at: https://www.andea.org/topic.cfm?menu=5288

http://www.cochrane.org/CD009275/PREG_different-types-dietary-advice-women-gestational-diabetes-mellitus

Prof Michael Peek
Associate Dean, ANU & Professor, Maternal Fetal Medicine
Obstetric management of diabetes in pregnancy: Fetal side

Professor Michael Peek
michael.peek@anu.edu.au
The majority of women with diabetes in pregnancy have successful pregnancies ending in a normal delivery of a normal infant.
• Fetal abnormality
• Fetal growth and wellbeing
• Implications of timing of delivery
• Long term effects
Fetal Abnormality
Risk of fetal abnormality

- Associated with pre-existing diabetes
- Increased risk compared to the general population
- Related to glucose control at the time of conception and in the first trimester
- The abnormalities are structural ones, not chromosomal
Congenital anomalies in infants of diabetic mothers

- **Skeletal and CNS**
  - Caudal regression syndrome
  - Neural tube defects
- **Cardiac**
  - Transposition of the great vessels
  - Ventricular septal defects
- **Renal**
- **Gastrointestinal**
Correlation between glycaemic control and malformation rate

Poor control v Good control

13.6% 2.6%

Ultrasound is the mainstay of diagnosis

- Dating scan
- Nuchal translucency scan
  - First look at anatomy
- 18-20 week ultrasound
- 24-26 week fetal echocardiography
What can you do about it?

• Prepregnancy advice on what to do:
  – Periconception glucose control
  – Folic acid
  – Low dose aspirin
  – Need for ultrasounds

• Endocrine and obstetric counselling in the multidisciplinary Diabetes in Pregnancy Clinic on Tuesdays at TCH
Fetal growth and wellbeing
Infant of diabetic mother
Diabetic Fetopathy

Mother

Placenta

Fetus

Hyperinsulinemia

Excessive fetal growth

Glucose
Lipids
Amino acids

Blue arrow from placenta to fetus
Fetal Effects

• Macrosomia with possible heart, lung and other organ damage
• Polyhydramnios
• Preterm delivery
• Fetal death in utero
Pathophysiology of fetal death

• Exact mechanism unknown
• Rare when glucose levels maintained within physiologic limits
• Probably related to fetal hyperinsulinaemia which may increase fetal metabolic rate and oxygen requirements
What can you do about it?

• Good glucose control
• Serial growth scans
  – Insulin requiring at least 28/40, 32/40 and 36/40
  – Individualised
• Antenatal corticosteroids for fetal lung maturation
• Magnesium sulphate for neuroprotection
Implications of timing of delivery
• Risks of prematurity vs stillbirth
• Risks of delivering large baby
• Increasing evidence of even early term delivery may be associated with poorer neurodevelopmental outcomes and increased risk of allergies
• Pressures of a busy service
Timing of Delivery

• Delivery before full term not indicated unless:
  – Macrosomia
  – Polyhydramnios
  – Difficult glycaemic control; on insulin
  – Other obstetric complications: pre-eclampsia, IUGR
Method of Delivery

• Uncomplicated diet controlled GDM await spontaneous labour. Caesarean section only for other obstetric reasons
• Insulin requiring GDM usually induction of labour 39+/40. Earlier depending upon complications.
• Consider Caesarean Section if EFW > 4.0kg
Neonatal Period

- Close Paediatric review
- Hypoglycaemia
  - Promote breastfeeding
- Polycythaemia and Jaundice
- Rarer problems
Long term risks
GDM and Future Health of Babies

- Prevalence of T2D/prediabetes at 22 years of age
  - Offspring of diet-treated GDM mothers - 21%
  - Offspring of T1D mothers - 11%
  - Offspring of control mothers - 4%

- Risk for metabolic syndrome
  - Offspring of diet-treated GDM mothers - 4.1 (95% CI, 1.7-10)
  - Offspring of T1D mothers - 2.6 (95% CI, 1.0-6.5%)

Clausen et al JCEM 2009; 94: 2464-2470
Prof Chris Nolan

Director of Endocrinology, ACT Health
GDM Follow-up & TOBOGM study

Capital Health *Education* - 14\(^{th}\) Oct 2017

Christopher Nolan
Canberra Hospital & Health Services
Australian National University Medical School
GDM Follow-up

- Mother
- Offspring
GDM and future health of mothers

- Follow-up of 5470 GDM patients and 783 control subjects
- Cumulative incidence of T2D - 25.8% at 15 years
- Risk for T2D - 9.6 X in GDM compared to control women

Lee et al. Diabetes Care 2007; 30: 878-883
GDM- does diagnosis and management improve long term health of the offspring?

- ACHOIS trial
- MFMUN-GDM trial

Landon et al Diabetes Care (2015) 38:445–452
ACHOIS follow up offspring and obesity at age 4.7 years

- Follow up of 199 (of 1000) mother child pairs at 4.7 years (South Australian Cohort)
- BMI > 85th centile for treated GDM 33% vs untreated GDM 29%
MFMUN-GDM trial
- follow up of offspring and obesity at age 5-10 years

- Follow up of 905 (55%) mother child pairs at 5-10 years of age
- BMI > 95th centile for treated GDM 20.8% vs untreated GDM 22.9% (not significant)
- BMI > 85th centile for treated GDM 32.6% vs untreated GDM 38.6% (not significant)

Landon et al Diabetes Care 2015;38:445–452
T1D & GDM and future health of offspring

• Prevalence of T2D/prediabetes at 22 years of age
  – Offspring of diet-treated GDM mothers - 21%
  – Offspring of T1D mothers - 11%
  – Offspring of control mothers - 4%

• aORs for metabolic syndrome
  – Offspring of diet-treated GDM mothers - 4.1 (95% CI, 1.7-10)
  – Offspring of T1D mothers - 2.6 (95% CI, 1.0-6.5%)

Clausen et al JCEM 2009; 94: 2464-2470
Treatment of GDM and offspring obesity

• Why has it not worked to reduce childhood obesity?
  – Treatment started too late
  – The wrong treatments
  – It is the neonatal/early childhood environment that is most important?
Timelines for prevention of gestational and permanent diabetes
Role of primary care:

• Prepare women for pregnancy
• Interpregnancy is a time of opportunity
• Follow up of mothers (CVD risk)
• Follow up of children
• Family approach
TOBOGM Study

TREATMENT OF BOOKING GESTATIONAL DIABETES MELLITUS STUDY
• To test whether treatment of ‘Booking GDM’ will reduce the sequelae of maternal ‘hyperglycaemia’ without increasing the risk of fetal under-nutrition.
TOBOGM- inclusion criteria

- <20 wks gestation
- One or or risk factors for GDM
  - Previous GDM or hyperglycaemia
  - Previous macrosomia/ large for gestational age baby
  - High risk ethnicity
  - BMI >= 30 kg/m²
  - 1st degree relative with diabetes
Local PIs: Profs Chris Nolan and Michael Peek

Clinical Trials Coordinator: Lori Grlj

Telep 02 61747586 lori.grlj@act.gov.au
Thank You!