Assisting Women with Obesity to Have Their Best Perinatal Outcomes

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Conflict of Interest—none

- Member Perinatal Services BC Advisory Board
- No financial interest in organizations or corporations related to issues discussed in this presentation

Photos of individuals with obesity are provided by the Rudd Center for Food Policy and Obesity, University of Connecticut

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Objectives

– Understand obesity as adipose tissue disease with multiple origins including broken food systems, endocrine disrupting chemicals and chronic stress
– Describe the physiological origins for common obesity related complications of the perinatal period
– List maternal and fetal morbidity associated with prepregnancy obesity
– Use the Edmonton Obesity Staging System to triage which women with obesity are at the highest risk for perinatal morbidity
– Apply the physiology of adipose tissue disease to develop measures that ameliorate risk for obesity related morbidities
– Support improved nutrition through advocacy that promotes equitable food distribution, production of nutritious foods, and reduction of environmental pollutants.
White adipose tissue is the largest & most productive endocrine organ.

Figure 1. The most significant physiological functions of white adipose tissue such as coagulation, appetite regulation, immunity, glucose and lipid metabolism, reproduction, angiogenesis, fibrinolysis, body weight homeostasis and vascular tone control.

OBESITY IS AN ENDOCRINE ORGAN DISEASE
Broken Food Systems

- Industrial monoculture dependent on external fertilizers, pesticides, hormones and water sources
- Foods manufactured for long shelf lives: high sodium, high sugars, preservatives
- Foods manufactured and distributed to maximize profits: long shelf lives, hyper-palatable
- Foods maldistributed with 50% of world’s population starving but the other 50% exposed to high quantities of non-nutritious foods
**Endocrine Disrupting Chemicals (EDCs)**

Highly persistent, bioaccumulative synthetic compounds

<table>
<thead>
<tr>
<th>EDCs</th>
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</thead>
<tbody>
<tr>
<td>- Organochlorines (DDT, DDE)</td>
</tr>
<tr>
<td>- Bisphenol A (BPA)</td>
</tr>
<tr>
<td>- Phthalates</td>
</tr>
<tr>
<td>- Polychlorinated biphenyls (PCBs)</td>
</tr>
<tr>
<td>- Polybrominated diphenylethers</td>
</tr>
<tr>
<td>- Parabens</td>
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<tr>
<td>- Phytoestrogens</td>
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</tbody>
</table>

**Environmental sources:**

- Pesticides
- Household plastics (food storage containers, baby bottles)
- Flame retardants
- Electric insulation
- Plasticizers, adhesives, lubricants, hydraulic fluids
- Coolants
- Soaps, lotions, cosmetics
Obesity has less to do with personal choice than with socioeconomic limits on choices.

Fig. 1. The effects of a single environmental exposure can be transmitted transgenerationally. An adverse maternal environment (F0) affects not only the development of the fetus (F1) but can also affect the germ cells which form the F2 generation.
From Fleming, et al., *Lancet*

**Paternal Obesity and Undernutrition**
- Reduced sperm motility, increased DNA damage
- Altered sperm epigenome and RNA profile
- Altered seminal plasma composition
- Reduced embryo potential
- Epigenetic reprogramming
- Endocrinal misregulation
- Postnatal cardiometabolic disease risk

**Maternal Obesity and Overnutrition**
- Metabolite and lipid accumulation in follicles and eggs
- Mitochondrial damage
- Cellular and ER stress
- Epigenetic and metabolic reprogramming
- Postnatal cardiometabolic disease risk
- Adverse programming evident after embryo transfer

**Maternal Undernutrition**
- Preimplantation embryo sensing of maternal nutrients
- Extra-embryonic (TE, PE) compensatory responses
- Epigenetic and metabolic reprogramming
- Resetting fetal growth rate through regulating ribosome biogenesis
- Endocrinal misregulation
- Postnatal cardiometabolic disease risk
- Adverse programming evident after embryo transfer
MATERNAL SMOKING DURING PREGNANCY

- Nicotine exposure downregulates genes responsible for nutrition and lipid metabolism
- Growth restricted fetus gains weight easily when exposed to excess calories, increasing risk for future obesity
- Eggs in female fetus also exposed to nicotine
“You can’t do anything; the women are already obese when they come for prenatal care!”

Oh, yes, we can!
PERINATAL MORBIDITY & MORTALITY

**EPIGENETIC CHANGES:**
- fetal
- ova in female fetus

**ECONOMIC & EDUCATIONAL DISPARITIES:**
- access to quality education
- access to living wage
- access to health care
- access to health information

**NUTRITIONAL DISPARITIES:**
- incidence & duration of breastfeeding
- income for quality nutrition
- availability of nutritious foods
- exposure to calorie dense, high glycemic index foods

**ENVIRONMENTAL DISPARITIES:**
- exposure to endocrine disrupting chemicals
- increased tobacco product exposure
- safety of physical activity

**OBESITY:**
- white adipose tissue cytokines & hormones
- satiety signaling from gut microbiome
FACTORS CONTRIBUTING TO OBESITY

- Grandmother obese during mother’s gestation
- Mother obese during pregnancy
- Grandmother or mother smoked during pregnancy
- Formula fed instead of breastmilk
- High glycemic index, calorie dense, highly processed foods
- Lack of control over foods
  - Public food programs, food pantry use
  - Family groups sharing foods
- Chronic stress, racism
- Night shift work, inadequate sleep
- Exposure to endocrine disrupting chemicals
  - Canned foods, plastic food containers
  - Pesticides
  - Environmental pollutants
- Neighborhood unsafe for physical activity
- Starvation
  - In utero growth restriction
  - Food deprivation
    - Poverty
    - War, relocation
  - Long term dieting to maintain low weight: ballet, athletics, clothing models
PERINATAL-RELATED PHYSIOLOGIC EFFECTS OF OBESITY

- INSULIN RESISTANCE (growth hormone)
  - Gestational diabetes
  - Fetal macrosomia
  - Newborn hypoglycemia
- LEPTIN RESISTANCE (leptin is a tocolytic)
  - Prolonged pregnancy (fetal macrosomia, shoulder dystocia)
  - Prolonged stages of labor
  - Postpartum uterine atony/hemorrhage
  - Impaired milk ejection reflex
- INFLAMMATION
  - Postpartum DVT
Evidence about Risk & Obesity

- Increase with increasing BMI:
  - Hypertensive disorders of pregnancy
  - Gestational diabetes
  - Macrosomia
  - Stillbirth
- Risk constant with increasing BMI or clinically insignificant increases:
  - Shoulder dystocia
  - Postpartum hemorrhage
  - The largest increases in risk start at a BMI of 36.

BUT MANY WOMEN HAVE NO PREGNANCY RELATED COMPLICATIONS
Not all obese women are at risk-
EOSS Predicts Mortality in NHANES III

www.drsharma.ca

Padwal R, Sharma AM et al. CMAJ 2011
The 2 most common pregnancy complications:

Edmonton Obesity Staging System (EOSS)

**Type 2 Diabetes**
- Stage 2: Type 2 Diabetes
- Stage 3: Micro-/macro-vascular disease
- Stage 4: Blindness, ESRD

**Hypertension**
- Stage 2: Hypertension
- Stage 3: Pre-hypertension, LVH, CHD
- Stage 4: Heart Failure

References:
- Sharma AM & Kushner RF, *Int J Obes 2009*
ASSESS

Assess Obesity Class and Stage

- Obesity Class (1-III) is based on BMI and is a measure of how BIG the patient is.
- Obesity Stage (0-4) is based on the MEDICAL, MENTAL, and FUNCTIONAL impact of obesity and is a measure of how HEALTHY the patient is.
- Waist circumference provides additional information regarding CARDIOMETABOLIC risk.

**Obesity Class**

<table>
<thead>
<tr>
<th>BMI</th>
<th>kg/m²</th>
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<tbody>
<tr>
<td>Underweight</td>
<td>≤18.5</td>
</tr>
<tr>
<td>Normal Weight</td>
<td>18.6 - 24.9</td>
</tr>
<tr>
<td>Overweight</td>
<td>25.0 - 29.9</td>
</tr>
<tr>
<td>Obesity Class I</td>
<td>30.0 - 34.9</td>
</tr>
<tr>
<td>Obesity Class II</td>
<td>35.0 - 39.9</td>
</tr>
<tr>
<td>Obesity Class III</td>
<td>≥ 40</td>
</tr>
</tbody>
</table>

**Obesity Stages (EOSS*)**

- Stage 4: End-Stage
- Stage 3: End-Organ Damage
- Stage 2: Established Co-Morbidity
- Stage 1: Preclinical Risk Factors
- Stage 0: No Apparent Risk Factors

*Edmonton Obesity Staging System

Waist Circumference Risk Threshold: Europid: ø ≥ 94 cm; ø ≥ 80 cm; Asian and Hispanic: ø ≥ 90 cm; ø ≥ 80 cm

www.drsharma.ca
A 10% reduction in prepregnancy BMI was associated with at least a 10% lower risk of preeclampsia, gestational diabetes, macrosomia and stillbirth. To reduce risks of cesarean birth and shoulder dystocia, a 20-30% reduction in BMI was needed.

<table>
<thead>
<tr>
<th></th>
<th>BMI 18.5-24.9 Referent group</th>
<th>BMI 25-29.9</th>
<th>BMI 30-34.9</th>
<th>BMI 35.39.9</th>
<th>BMI &gt; 40</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preeclampsia</td>
<td>3.4</td>
<td>6.6</td>
<td>10.0</td>
<td>12.8</td>
<td>16.3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Gestational diabetes</td>
<td>6.1</td>
<td>9.7</td>
<td>13.7</td>
<td>16.6</td>
<td>20.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Cesarean birth</td>
<td>26.5</td>
<td>33.1</td>
<td>38.2</td>
<td>43.1</td>
<td>49.7</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Macrosomic newborn</td>
<td>1.4</td>
<td>2.8</td>
<td>3.8</td>
<td>4.5</td>
<td>6.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Shoulder dystocia</td>
<td>3.5</td>
<td>4.1</td>
<td>4.1</td>
<td>4.4</td>
<td>4.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Postpartum hemorrhage</td>
<td>0.7</td>
<td>0.7</td>
<td>0.8</td>
<td>0.7</td>
<td>0.3</td>
<td>0.010</td>
</tr>
<tr>
<td>Stillbirth</td>
<td>0.3</td>
<td>0.3</td>
<td>0.4</td>
<td>0.4</td>
<td>0.6</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>
Respectful Care in Pregnancy

- Recognize that women’s choices for purchasing nutritious food, preparing fresh foods and being physically active can be limited
- Respectful counseling relies on information sharing and patient-centered decision making
- Information dumping with expectations for compliance is hierarchical, privileged and demeaning
- Have appropriate equipment: large gowns, large BP cuffs, armless chairs, bariatric beds in hospital
- Use patient-centered language:
  - Women with obesity not obese women, large women or fat women
  - Stop the use of morbid obesity. Use Class III obesity.
Optimizing labor and birth begins with PRENATAL SELF-MANAGEMENT

<table>
<thead>
<tr>
<th>HEALTH GUIDANCE</th>
<th>PHYSIOLOGIC ACTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>NUTRITION:</td>
<td></td>
</tr>
<tr>
<td>Low glycemic index, low fat, DASH</td>
<td>Fosters healthier gut microbiome, improved satiety signaling</td>
</tr>
<tr>
<td>Vitamin supplementation</td>
<td>Improves micronutrient intake if nutritious are unavailable</td>
</tr>
<tr>
<td>Weight gain within US IOM guidelines</td>
<td>Reduces accumulation of excess white adipose</td>
</tr>
<tr>
<td>PHYSICAL ACTIVITY:</td>
<td></td>
</tr>
<tr>
<td>30 minutes of walking 5 days a week (2 x 15, 3 x 10)</td>
<td>Increases peripheral insulin activity, improves cardio-respiratory fitness for labor</td>
</tr>
<tr>
<td>SMOKING CESSATION</td>
<td>Decreases DNA methylation-decreasing obesity in offspring</td>
</tr>
<tr>
<td>SLEEP:</td>
<td></td>
</tr>
<tr>
<td>7-8 hours of nighttime sleep Day time work shifts</td>
<td>Decreases insulin resistance, decreases ghrelin, increases leptin</td>
</tr>
</tbody>
</table>
1990 Institute of Medicine
Recommended Prenatal Weight Gains
Re-affirmed in 2009 by IOM

<table>
<thead>
<tr>
<th>Height for Weight Category</th>
<th>BMI Range</th>
<th>Recommended Weight Gain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Underweight women</td>
<td>(BMI &lt;19.8)</td>
<td>12.5-18.0</td>
</tr>
<tr>
<td>Normal weight women</td>
<td>(BMI 19.8-26.0)</td>
<td>11.5-16.0</td>
</tr>
<tr>
<td>Overweight women</td>
<td>(BMI 26.1-29.9)</td>
<td>7.0-11.5</td>
</tr>
<tr>
<td>Obese women</td>
<td>(BMI &gt;29.9)</td>
<td>at least 6.0</td>
</tr>
</tbody>
</table>
Informed choice in place of birth for women with obese BMIs

COMMUNITY:

HOME or BIRTH CENTER
- Women with BMIs of 35-40 without co-morbid disease have risks similar to women with lower BMIs (Hollowell)
- Rates of uncomplicated vaginal birth for nulliparas 68% (Rowe, Jevitt)
- Rates of cesarean birth 7-50%

HOSPITAL
- RCOG & NICE guidelines recommend hospital for BMI > 35
- RCOG
  - Notify anesthesia if BMI Class 4
  - Continuous IV access if BMI Class 4
- ACOG
  - Assumes hospital birth & IV
  - Anticoagulation prophylaxis-weight based
Induction of Labor

- Blunted myometrial response to oxytocin (Carlson 2017)
- No superior method of induction: dinoprostone, misoprostol or transcervical catheter + balloon (Sarumi)
- In women with obese BMIs, vaginal compared to oral misoprostol reduced time to 3 cm dilatation and reduced length of labor (Soni)
- Consider “allowing” a longer first stage (ACOG)

Increased cesarean birth (Ellis)
Longer Labor Curves

- 118,978 gravidas, singleton at term, analyzed labor curves by parity & BMI:
  - 48% nulliparas, 22% cesarean birth
  - Inductions of labor and cesarean births increased with increasing BMI
  - Stage 1 was 1.2 hours longer for nulliparas at high BMIs
  - Stage 1 was 1.0 hour longer for multiparas at high BMIs
  - Stage 2 was not prolonged with or without epidural analgesia

Statistically significant but is it clinically significant?
Kominiarek, et al.
US obstetrical units with midwifery care had significantly lower rates of:

- Induction of labor
- Oxytocin augmentation of labor
- Cesarean birth

(Neal JL)
– ADMISSION IN ACTIVE LABOR
– UNDISTURBED LABOR
– MOBILITY
– NUTRITION AND HYDRATION
– WATER IMMERSION
– LONGER LABOR CURVES

LABOR SUPPORT

tried and true
midwifery techniques
# Immediate Postpartum Support

## Mother
- Active management of 3\textsuperscript{rd} stage
- Reduce embolism risks (consider BMI & co-morbidities)
  - Mobility
  - Alternating compression stockings
  - Anticoagulation?

## Newborn
- Skin to skin
- Undisturbed observation to enable breast crawl and initiation of breastfeeding
- Any & all techniques to increase latch & milk production
- Early breastfeeding
  - Uterotonic effect
  - Reduces neonatal hypoglycemia
MICRO
INDIVIDUAL ADVICE & TREATMENTS

MID
EVIDENCE-BASED SYSTEMS SUPPORT

MACRO
GOVERNMENT POLICY & SUPPORT
FOOD SUPPLY

NUTRITIOUS
LIMIT ADVERTISING OF NON-NUTRITIOUS FOODS
REDUCE PESTICIDE & FERTILIZER USE

ENVIRONMENT
REDUCE ENDOCRINE DISRUPTING CHEMICALS

FOOD DISTRIBUTION

AFFORDABILITY
CHOICE
HEALTHY PORTIONS
REDUCE FOOD WASTER

HEALTH CARE REFORM

ADEQUATE ACCESS TO PREVENTATIVE CARE
ADEQUATE REIMBURSEMENT FOR TIME-CONSUMING COUNSELING

POLICY SUPPORT
QUESTIONS?
REFERENCES


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