

NUTRITION DURING  
PREGNANCY: CONDITIONS  
AND INTERVENTIONS  
CHAPTER 5  
NUTD238

# Obesity and pregnancy

**Table 5.1** Comparative prevalence of obesity prior to pregnancy and outcomes related to prepregnancy weight status

Weight status	Underweight	Normal	Overweight	Obese	Very obese	Extremely obese
BMI, kg/m <sup>2</sup>	<18.5	18.5–24.9	25–29.9	30–34.9	35–39.9	≥40
Preterm	11.6%	8.1%	8.4%	10.6%	8.9%	12.4%
Gestational diabetes	3.5%	3.8%	4.7%	7.0%	9.6%	11.0%
Preexisting diabetes	0%	0.8%	1.7%	2.4%	6.9%	9.7%
Hypertensive disorder	5.8%	9.1%	13.3%	20.7%	23.3%	31.7%

SOURCE: Data are from Chu et al.

- Increased risk of these disorders is associated with unfavorable metabolic changes- initiated by the excess amount of fat in the body
  - ▣ Increased blood glucose
  - ▣ Increased C- reactive protein: marker of inflammation
  - ▣ Increased plasma [insulin]
  - ▣ Increased BP
  - ▣ High blood levels of total cholesterol, LDL-C and TG
  - ▣ Low levels of HDL-C

# Obesity and infant outcomes

- Increase in rate of
  - ▣ Still birth
  - ▣ LGA
  - ▣ Cesarean deliveries
  - ▣ Chance that child will be overweight and develop type II diabetes later on → may be due to exposure to high levels of insulin in utero- IR

# Recommendations

- Balanced diet- meet all nutrient needs
- Physical activity
- Appropriate amount of weight gain during pregnancy

# Pregnancy after bariatric surgery

- Side effects of surgery including nausea, vomiting, and dumping syndrome may remain during pregnancy
  - ▣ Dumping syndrome: occurs after eating- due to rapid emptying of the stomach: weakness, abdominal discomfort, dizziness, flushing, nausea, palpitation
- Better to wait to get pregnant for around 1-2 yrs after surgery- stable weight, nutrient intake, and stores
- **Deficiencies** are present- esp: B12, Fe, vitamin D, Ca- must monitor for these and supplement as needed
- Maternal and neonatal health outcomes are good- if intake and nutrient levels are monitored

# Hypertensive disorders of pregnancy

**Table 5.2** Definitions and features of hypertensive disorders of pregnancy\*<sup>2</sup>

## Chronic Hypertension

Hypertension that is present before pregnancy or diagnosed before 20 weeks of pregnancy. Hypertension is defined as blood pressure  $\geq 140$  mm Hg systolic or  $\geq 90$  mm Hg diastolic blood pressure.

Hypertension first diagnosed during pregnancy that does not resolve after pregnancy is also classified as chronic hypertension.

## Gestational Hypertension

This condition exists when elevated blood pressure levels are detected for the first time after mid-pregnancy. It is not accompanied by proteinuria. If blood pressure returns to normal by 12 weeks postpartum, the condition is considered to be transient hypertension of pregnancy. If it remains elevated, then the woman is considered to have chronic hypertension.

Women with gestational hypertension are at lower risk for poor pregnancy outcomes than are women with preeclampsia.

## Preeclampsia–Eclampsia

A pregnancy-specific syndrome that usually occurs after 20 weeks gestation (but that may occur earlier) in previously normotensive women. It is determined by

increased blood pressure during pregnancy to  $\geq 140$  mm Hg systolic or  $\geq 90$  mm Hg diastolic and is accompanied by proteinuria. In the absence of proteinuria, the disease is highly suspected when increased blood pressure is accompanied by headache, blurred vision, abdominal pain, low platelet count, and abnormal liver enzyme values.

- Proteinuria is defined as the urinary excretion of  $\geq 0.3$  grams of protein in a 24-hour urine specimen. This usually correlates well with readings of  $\geq 30$  mg/dL protein, or  $\geq 2$  on dipstick readings taken in samples from women free of urinary tract infection. In the absence of urinary tract infection, proteinuria is a manifestation of kidney damage.
- Eclampsia is defined as the occurrence of seizures that cannot be attributed to other causes in women with preeclampsia.

## Preeclampsia Superimposed on Chronic Hypertension

This disorder is characterized by the development of proteinuria during pregnancy in women with chronic hypertension. In women with hypertension and proteinuria before 20 weeks of pregnancy, it is indicated by a sudden increase in proteinuria, blood pressure, or abnormal platelet or liver enzyme levels.

# Oxidative stress and nutrition

- HTN is related to :
  - ▣ chronic inflammation
  - ▣ oxidative stress
  - ▣ damage to the endothelium of blood vessels →
    - endothelial dysfunction →
      - reduced blood flow
      - increased risk of blood clots
      - plaque formation



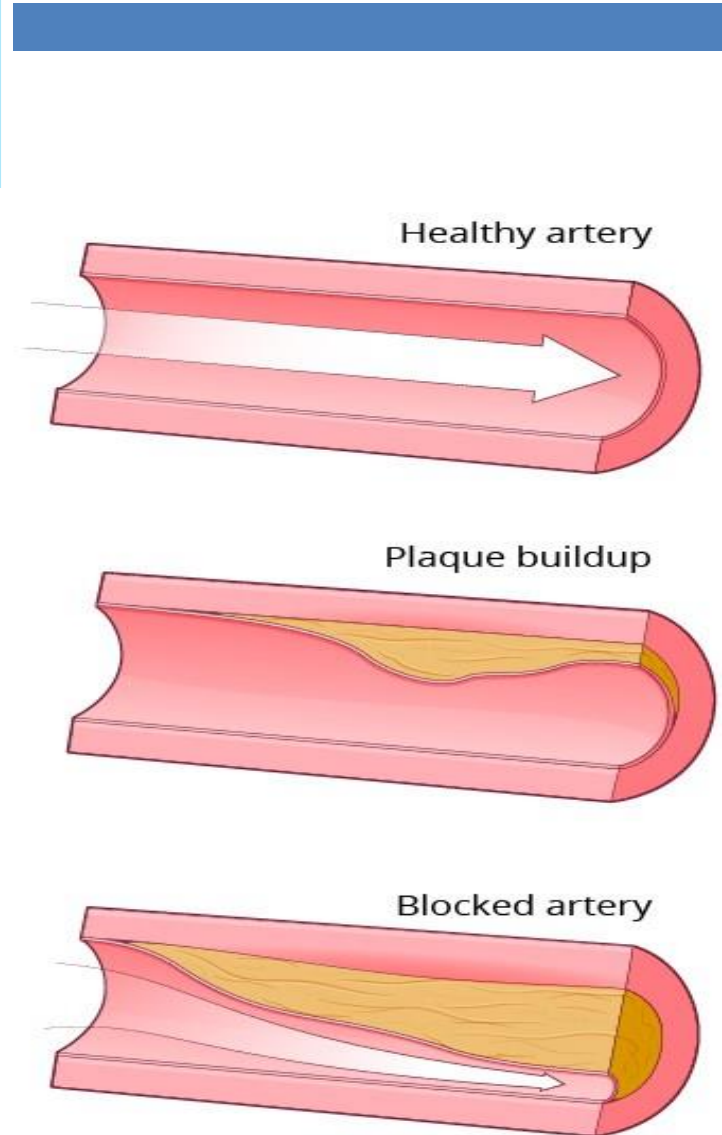
**Table 5.3** Dietary and other environmental exposures that increase or decrease chronic inflammation and oxidative stress<sup>13,20</sup>

1. Decrease

- Regular intake of colorful fruits and vegetables, dried beans, and whole-grain products
- Adequate intake of the omega-3 fatty acids EPA and DHA
- Vitamin D sufficiency
- Physical activity

2. Increase

- Frequent intake of processed and high-fat meats
- Regular intake of baked products and snack foods with *trans* fats
- Frequent consumption of soft drinks, other high-sugar beverages
- Physical inactivity
- High levels of body fat, especially visceral fat
- Smoking



# Chronic HTN

- Mild HTN- patient is taken off antihypertensive meds, *preconception or early in pregnancy*, drugs do not seem to improve the course or outcome of pregnancy
  - ▣ Mild HTN, in healthy women, if it does not become worse during pregnancy → few risks to maternal and newborn health
  
- $BP \geq 160/110$  mmHg- either or both values- increased risk of:
  - ▣ Fetal death
  - ▣ Preterm delivery
  - ▣ Fetal growth retardation→ Selection of proper antihypertensive medication reduces risk to a certain extent

# Nutrition intervention

- Monitor diet preconception and during pregnancy
  - ▣ Balanced; healthy
  
- Same recommendations for weight gain
  
- Salt sensitive HTN-> sodium intake- not too little (effect fetal growth) and not too high
  - ▣ Amount consumed in preconception is recommended if found successful in managing condition

# Preeclampsia-eclampsia

## □ Characterized by:

- Oxidative stress, inadequate antioxidant defenses, inflammation, and endothelial dysfunction
- Platelet aggregation and blood coagulation due to deficits in prostacyclin relative to thromboxane
- Blood vessel constriction, restricted blood flow
- Increased BP
- IR
- Adverse maternal immune system responses to the placenta
- Elevated blood levels of TG, FFA, and cholesterol

# The cause of preeclampsia

- The cause of preeclampsia involves several factors- may originate from **abnormal implantation and vascularization of the placenta**; causes of this may include:
  - Insufficient blood flow to the uterus
  - Damage of blood vessels
  - An immune system problem
  - Genetics

- Abnormal blood flow: ↓ delivery of nutrients to the fetus
- There are no reliable means to identify women who will develop this condition before it is established
- Most affected organs are the placenta, mother's kidneys and liver

- **Cure → delivery**
- S&S of preeclampsia usually disappear rapidly after delivery
- **Eclampsia** may occur within 12 days following delivery
  - ▣ Can be a life-threatening condition- difficult to predict
- Women with preeclampsia are at increased risk for developing-later in life:
  - ▣ Gestational diabetes
  - ▣ Type 2 diabetes
  - ▣ HTN
  - ▣ Heart disease and stroke

**Table 5.4** Signs and symptoms of preeclampsia<sup>33</sup>

- Hypertension
- Increased urinary protein (albumin)
- Decreased plasma volume expansion (hemoglobin levels .13 g/dL)
- Low urine output
- Persistent and severe headaches
- Sensitivity of the eyes to bright light
- Blurred vision
- Abdominal pain
- Nausea

**Table 5.5** Outcomes related to the existence of preeclampsia during pregnancy<sup>23,35</sup>

**Mother**

- Early delivery by cesarean section
- Acute renal (kidney) dysfunction
- Increased risk of gestational diabetes, hypertension, and type 2 diabetes later in life
- Abruptio placenta (rupture of the placenta)

**Newborn**

- Growth restriction
- Respiratory distress syndrome

**Table 5.6** Risk factors for preeclampsia<sup>23,38-40</sup>

- First pregnancy (nulliparous)
- Obesity, especially high levels of central body fat
- Underweight
- Mother's smallness at birth
- African Americans, American Indians
- History of preeclampsia
- Preexisting diabetes mellitus
- Age over 35 years
- Multifetal pregnancy
- Insulin resistance
- Abnormally high blood triglyceride levels
- Chronic hypertension
- Renal disease
- Poor vitamin D status
- Poor calcium status
- Consumption of a pro-inflammatory, pro-oxidative stress diet

Increased rates of preterm delivery and LBW in infants → partly related to clinical decisions to deliver fetuses early in order to treat the disease



# Vitamin and mineral supplements and risk of preeclampsia

- Use of vitamin C and E supplements: antioxidants-no effect
- **Vitamin D**: +ve effect when poor vitamin D status- some study findings/ others do not find this effect
- **Ca supplements**:  $\geq 1$  g/d- reduce risk by half/ esp when Ca intake is initially low
- Diet pattern (1<sup>st</sup> 22wks)- plant/antioxidant/fiber vs. meat, sweet drinks, and salty snacks
- Sodium (salt) intake- restriction does not prevent it

# Nutritional recommendations/ interventions for preeclampsia

- Begin prior to pregnancy; as early as possible in at risk women
- 1-2g/day of dietary or supplemental Ca
- Adequate vitamin D status
- Use of a multivitamin-mineral supplement
- $\geq 5$  servings/d of colorful vegetables and fruits
- Balanced/ varied diet
- Moderate exercise (e.g., walking, swimming)-30mins/d- unless medically contraindicated
- Wt gain- within recommendations
- Avoid high- dose Fe supplements: pro-oxidant/  $\uparrow$ inflammation

# Gestational diabetes

- Increased incidence with increased **obesity** prevalence
- Gestational diabetes in underweight and normal-weight women- related to IR combined with a deficit in insulin production
- Gestational diabetes in obese women → related to IR- not inadequate insulin production

- Development of gestational diabetes- usually indicates a **predisposition** to IR and type 2 diabetes (may not be clinically apparent) which is later **expressed due to physiological changes** that occur during pregnancy
  - Exaggerated metabolic changes favoring oxidative stress and elevated blood glucose levels
  
- Women with gestational diabetes develop elevated levels of glucose, TG, fatty acids, and sometimes BP; thirst, polyuria

# Risk factors for gestational diabetes

## □ High risk:

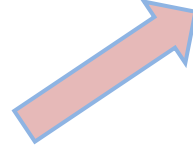
- ▣ History of glucose intolerance
- ▣ Current glucosuria

TABLE 5.8 ▶ Risk factors for gestational diabetes<sup>66,73,78</sup>

- Obesity, especially high levels of central body fat
- Weight gain between pregnancies
- Age over 35 years
- Native American, Hispanic, Asian ancestry
- Genetic traits (GIP variant)
- Strong family history of type 2 diabetes
- History of delivery of a macrosomic newborn (> 4500 g or > 10 lb)
- Chronic hypertension
- Mother was SGA at birth
- History of gestational diabetes in a previous pregnancy
- Physical inactivity
- Polycystic ovary syndrome
- Multifetal pregnancy
- Consumption of Western-type diet (low fiber intake, low vegetable and fruit intake, regular intake of sugars and high-glycemic index foods, red and processed meats)

# Consequences of gestational diabetes

- Elevated **hemoglobin A1c** levels (*normal* <5.7%), a long-term marker of blood glucose [ ] → indicates poor glucose control and higher risk of adverse outcomes
- Hemoglobin A1c level > 8% is associated with higher rates of



**Table 5.7** Adverse outcomes associated with gestational diabetes<sup>62</sup>

## Mother

- Cesarean delivery to prevent shoulder dystocia
- Increased risk for preeclampsia during pregnancy
- Increased risk of type 2 diabetes, hypertension, and obesity later in life
- Increased risk for gestational diabetes in a subsequent pregnancy

## Offspring

- Stillbirth
- Spontaneous abortion
- Congenital anomalies
- Macrosomia (>10 lb or >4500g)
- Neonatal hypoglycemia, death
- Increased risk of insulin resistance, type 2 diabetes, high blood pressure, and obesity later in life

# Consequences of gestational diabetes

- Exposure to high insulin levels *in utero* leads to increased glucose uptake into cells
  - ▣ Conversion of glucose to TG
  - ▣ Increase fetal formation of fat and muscle tissue
- The end of pregnancy initially restores insulin sensitivity in most women- a degree of IR often remains
- Women with weight gain after pregnancy and repeated pregnancies continue to experience insulin insufficiency and resistance → higher risk of developing type 2 diabetes later in life

# Diagnosis of gestational diabetes

- Based on abnormal blood glucose levels
- Glucose screening is recommended for women *at high risk* at the initial visit
- A plasma glucose screening between 24- 28wks of pregnancy is recommended for women at “average risk” and for high-risk women not determined by glucose screen to have elevated glucose levels earlier



# Diagnosis of gestational diabetes

- **Glucose challenge test:** a 50g oral glucose test- blood glucose screening (can be done without fasting)
  - ▣ Blood is collected after 1hr; normal level <130-140mg/dL, if level is higher- oral glucose tolerance test (OGTT) should be done
  
- **OGTT (100g)** is the basis for the diagnosis of most cases of gestational diabetes
  - ▣ Overnight fast- measure blood sugar then,
  - ▣ Drink solution- blood sugar measured every hour for 3 hours
  - ▣ If at least two of the readings are higher than normal → gestational diabetes diagnosis
  
- *The test can be bypassed among women with very high glucose screening results AND TREATMENT STARTED*



M Aqeel & S Muhanna

- A diagnosis of gestational diabetes is made when  **$\geq 2$  values** for plasma glucose [ ] exceed these levels:
  - ▣ Overnight fast 95 mg/dL
  - ▣ 1hr after glucose load 180 mg/dL
  - ▣ 2 hrs after glucose load 155 mg/dL
  - ▣ 3 hrs after glucose load 140 mg/dL

- Glucose screens are not recommended for women at low risk, defined as:
  - ▣ Age <25 years
  - ▣ Member of a low-risk ethnic group
  - ▣ No diabetes in first-degree relatives
  - ▣ Normal pre-pregnancy weight and weight gain during pregnancy
  - ▣ No history of glucose intolerance
  - ▣ No prior poor obstetrical outcomes

# Treatment

- **Initial:** normalize blood glucose levels through diet and PA
  - Considered successful when:
    - Fasting blood glucose values remain  $\leq 95$  mg/dL
    - Or 1-hr PP values  $\leq 140$  mg/dL and 2-hr PP levels  $\leq 120$  mg/dL
- *Insulin is recommended when fasting glucose levels or when 1- and 2hr PP glucose values exceed these cut-points*
- Low caloric intakes- not used for blood glucose control/ ketosis
- Restriction of pregnancy weight gain to below recommended amounts is **not advised- SGA**
- Type 2 diabetes in non-pregnant individuals is often treated with sulfonylurea oral medications
  - These drugs cannot be used in pregnancy because they cross the placenta and stimulate fetal insulin production

# Benefits of exercise

- Decrease IR/ increase sensitivity to insulin
- Enhance blood glucose control
- Levels of exercise ~50–60% of VO<sub>2</sub> max recommended for women with gestational diabetes
- Levels of exercise should make women become **slightly sweaty but not overheated, dehydrated, or exhausted**

# Nutritional management

## □ Goals:

- Normalize blood glucose levels
- Normalize carb metabolism
- Reduce risk of mother/ offspring risk of developing diabetes, HTN, heart disease, and obesity

- Components of the nutritional management:
  - ▣ Assessing dietary habits and exercise habits
  - ▣ Developing an individualized diet and exercise plan for blood glucose control
  - ▣ Monitoring weight gain, dietary intake
  - ▣ Interpreting blood glucose and urinary ketone results
  - ▣ Ensuring follow-up during pregnancy and postpartum
  
- **Same for women who enter pregnancy with type II diabetes**





□ Diet plan:

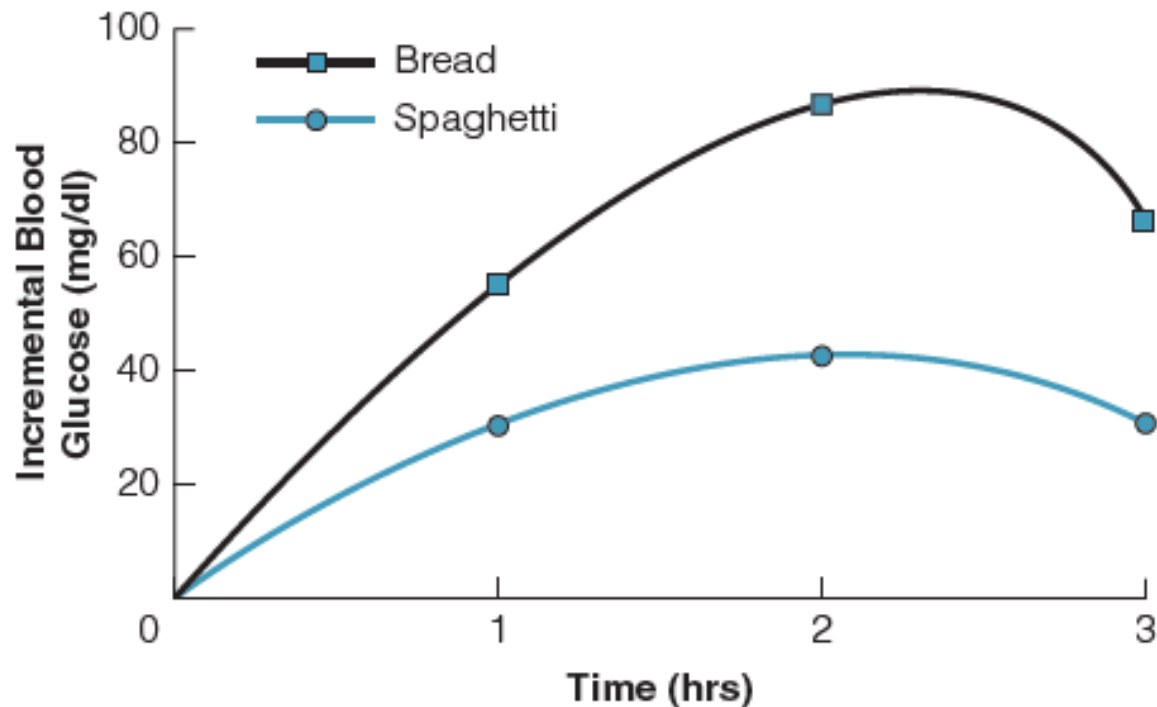
- ▣ Whole-grain breads and cereals, vegetables, fruits, and high-fiber foods
- ▣ Limited intake of simple sugars and foods and beverages that contain them
- ▣ Low-GI foods, or high fiber carbohydrate foods that do not greatly raise glucose levels
- ▣ Unsaturated fats
- ▣ **Three regular meals and snacks daily**

**Table 5.10** Estimating levels of caloric need in women with gestational diabetes<sup>65</sup>

Current Weight Status	BMI, kg/m <sup>2</sup>	Calories per kg Body Weight, kcal/kg
Underweight	<18.5	up to 40
Normal weight	18.5–24.9	30
Overweight, obese	25–34	25
Morbidly obese	≥34	20 or less

- Carbohydrates: 40-50%
  - ▣ Carbohydrate calories should be obtained from **complex carbohydrate** foods that are high in fiber
  - ▣ Low GI?
  
- Fat: 30-40%
  - ▣ Fat calories should be obtained primarily from food sources of **unsaturated fats**
  
- Protein: 20%

# GLYCEMIC INDEX RESPONSE TO FOODS



**ILLUSTRATION 5.3** ► Blood glucose levels after a meal containing white bread (GI = 70) or spaghetti (GI = 48) is consumed by individuals with diabetes.

# Urinary ketone testing

- Monitor urinary ketone levels using dipsticks
- The presence of ketones indicates a –ve calorie balance that is likely related to inadequate calorie intake or skipped meals
- In interpretation of test values- consider that 10-20% of pregnant women spill ketones after an overnight fast

# Post partum follow-up

- About 15% of women with gestational diabetes will remain glucose intolerant postpartum
  - ▣ 10-15% will develop type 2 diabetes within 2-5 years
- Most women who manage their gestational diabetes with diet and exercise will not require monitoring of blood glucose levels after pregnancy
- Women requiring insulin for glucose management should be tested for fasting and 2-hr PP blood glucose values before hospital discharge

# Post partum follow-up

- A 75g OGTT is recommended between 6 to 12 wks postpartum- in women who were diagnosed with gestational diabetes during pregnancy but tested –ve for glucose intolerance postpartum
- Negative results should be followed by repeated glucose testing every 3yrs

# Type I diabetes during pregnancy

- Require insulin injections
- More risky to mother and fetus than gestational diabetes



# Type I diabetes during pregnancy

- Type 1 diabetes places women at risk of kidney disease, HTN, and other complications of pregnancy
- Newborns are at increased risk of:
  - ▣ Mortality
  - ▣ Being SGA or LGA
  - ▣ Experiencing hypoglycemia within 12hrs after birth
- Type I diabetes: increase by 2-9% the risk of:
  - ▣ Congenital malformations of the pelvis, CNS, and heart in offspring
- Good control of blood glucose levels reduces the risk of malformations and macrosomia

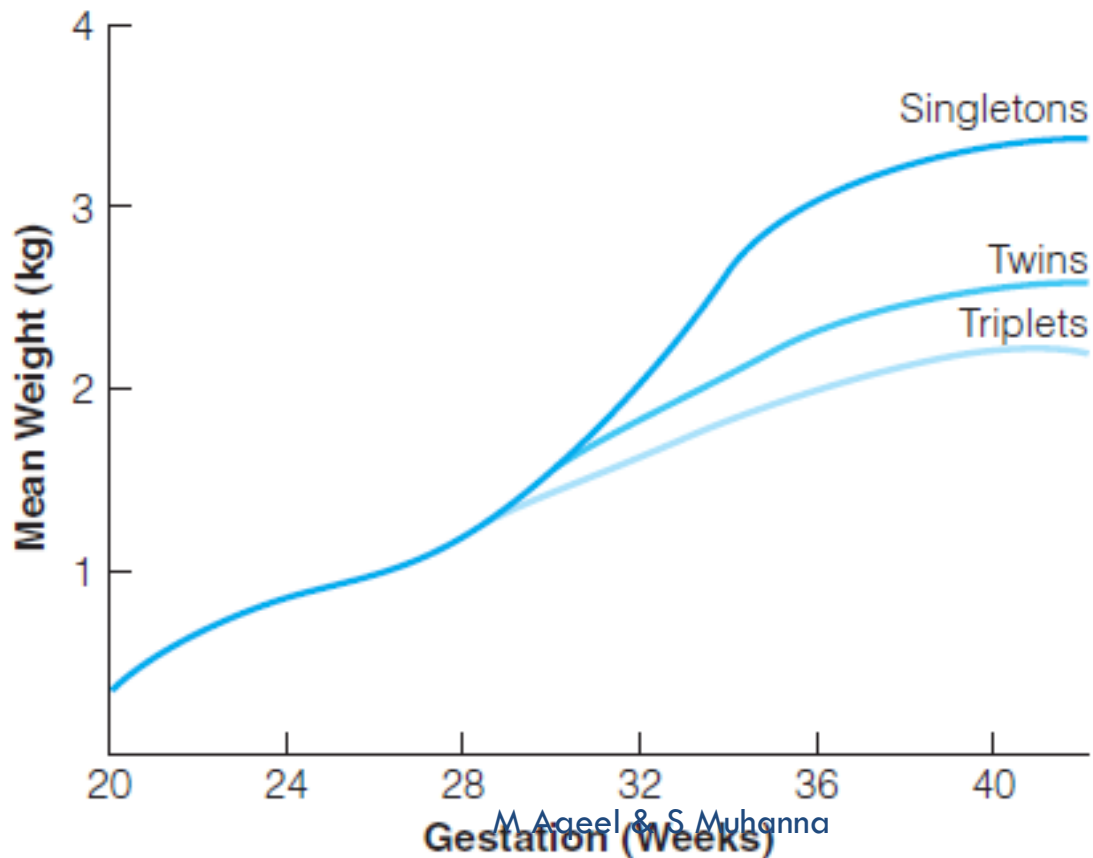
# Nutritional management

- **Goals:**
  - ▣ Control of blood glucose levels
  - ▣ Nutritional adequacy of dietary intake
  - ▣ Achievement of recommended amounts of weight gain
  - ▣ A healthy mother and newborn
  
- *Monitoring urinary ketones is necessary- women susceptible to developing ketosis/ more than in gestational diabetes*

- Dizygotic: 2 fertilized eggs- 70%; same sex 50% of the times
- Monozygotic: one fertilized egg- 30%; always same sex
- Some twins use the same placenta- but it is more common for each fetus to have their own placenta
- May share a common amniotic sac-> higher risk of death, malformations, short gestation; or they can have separate sacs

# In utero growth of twins/ triplets

**Illustration 5.5** Rates of fetal weight gain in singleton, twin, and triplet fetuses.



# Vanishing twin phenomenon

- Disappearance of embryos within 13wks of conception
- 6-12% begin with twins- 3% result in birth of twins
- Absorption into the uterus within first 8 wks of conception

**Table 5.12** Risks to mother and fetuses associated with multifetal pregnancy<sup>92</sup>

**Pregnant Women**

- Preeclampsia
- Iron-deficiency anemia
- Gestational diabetes
- Hyperemesis gravidarum
- Placenta previa
- Kidney disease
- Fetal loss
- Preterm delivery
- Cesarean delivery

**Newborns**

- Neonatal death
- Congenital abnormalities
- Respiratory distress syndrome
- Intraventricular hemorrhage
- Cerebral palsy

**Table 5.13** Average birth weight and gestational age at delivery, and low-birth-weight rates, of singleton, twin, and triplet newborns<sup>92,93</sup>

	Mean Birth Weight	Mean Gestational Age	Low-Birth-Weight Rate
Singletons	3440 g (7.7 lb)	39–40 weeks	6%
Twins	2400 g (5.4 lb)	37 weeks	54%
Triplets	1800 g (4.0 lb)	33–34 weeks	90%

# Interventions for risk reduction

- Consistent care
- Increased attention to nutritional needs
- Intensive follow-up
  
- **Outcomes:**
  - ▣ Rate of very-LBW decreased
  - ▣ Perinatal mortality is lower

# Nutrition and outcome of multifetal pregnancies

- Weight gain in multifetal pregnancies:
  - ▣ Linear relationship with birth weight
  - ▣ Depend on pre-pregnancy weight status

**Table 5.15** Prepregnancy weight status and weight-gain relationships in twin pregnancy<sup>84</sup>

Prepregnancy Weight Status	Weight Gain Related to Birth Weights of >2500 g (5.5 lb)
Underweight	44.2 lb (20.1 kg)
Normal weight	40.9 lb (18.6 kg)
Overweight	37.8 lb (17.2 kg)
Obese	37.2 lb (16.9 kg)
Very obese	29.2 lb (13.3 kg)



- Weight gain in twin pregnancies:
  - ▣ +ve rate of weight gain in the first half of pregnancy- strongly associated with increased BW
  - ▣ Weight loss after 28wks- increases risk of preterm delivery by threefold
  
- 0.2 kg/wk in the 1<sup>st</sup> trimester
- 0.7 kg/wk in the 2<sup>nd</sup> and 3<sup>rd</sup> trimesters
  
- Weight gain in triplet pregnancies:
  - ▣ ~23kg- healthy sized triplets → ≥ 0.7kg/week

# Dietary intake in twin pregnancies

- Adequate nutrition- *main component of prenatal care*, energy and nutrient needs increase
- Higher caloric need for tissue maintenance and growth in these pregnancies
  - Evidence for this- increased weight gain and quicker onset of starvation metabolism
  - Higher incidence of twins in overweight and obese women- weight status is an important factor

- Research finding: women with multifetal pregnancies consume an avg of 265kcal more/day than singleton pregnancies (when she enter pregnancy!)
- Need for essential fatty acids increase- poor intake associated with neurological abnormalities and vision impairment in twins
- Increased requirements of Ca and Fe

# Nutritional recommendations for women with multifetal pregnancy

- *Theoretically*, to achieve a 18.2kg weight gain (4.5 kg more than in singleton pregnancy)
  - ▣ Women with twins would need to consume **more by 150kcal/d**, average of 450cal more per day than pre-pregnancy
- Adequacy of caloric intake- estimated by weight gain progress

**Table 5.16** “Best practice” recommendations for nutrition during multifetal pregnancy<sup>92</sup>

### Weight Gain

Twin pregnancy: Overall gain of 35–45 lb (15.9–20.5 kg). Underweight women should gain at the upper end of this range, and overweight and obese women at the lower end.

- First trimester: 4–6 lb (1.8–2.7 kg)
- Second and third trimesters: 1.5 lb (0.7 kg) per week

Triplet pregnancy: Overall gain of approximately 50 lb (22.7 kg)

- Gain of 1.5 lb (0.7 kg) per week through pregnancy

### Daily Food Intake

Twin pregnancy (2400–2800 calories a day)

- Grains: 8–10 oz
- Vegetables: 3–3.5 c
- Fruits: 2–2.5 c
- Meat and beans: 6.5–7 oz
- Milk: 3 c
- Oil: 7–8 tsp
- Discretionary calorie allowance: 362–426

Triplet pregnancy

- Food intake from the MyPyramid groups should be consumed at a level that promotes targeted weight gain.

### Caloric Intake

Twin pregnancy

- 450 calories above prepregnancy intake, or the amount consistent with targeted weight-gain progress

Triplet pregnancy

- Caloric intake levels should promote targeted weight-gain progress.

### Nutrient Intake

Twin and triplet pregnancy

- RDA or AI levels or somewhat more than these levels
- Intakes should be lower than ULs.

### Vitamin and Mineral Supplements

Twin pregnancy

- Use a prenatal vitamin and mineral supplement.

Triplet pregnancy

- Provide prenatal vitamin and mineral supplements; avoid excessively high amounts of nutrients.

# Fetal alcohol spectrum

- The term *fetal alcohol spectrum* is used to describe the range of effects of fetal alcohol exposure on mental development and physical growth
  
- This spectrum includes:
  - ▣ Behavioral problems
  - ▣ Mental retardation
  - ▣ Aggressiveness
  - ▣ Nervousness
  - ▣ Short attention span
  - ▣ Growth-stunting
  - ▣ Birth defects

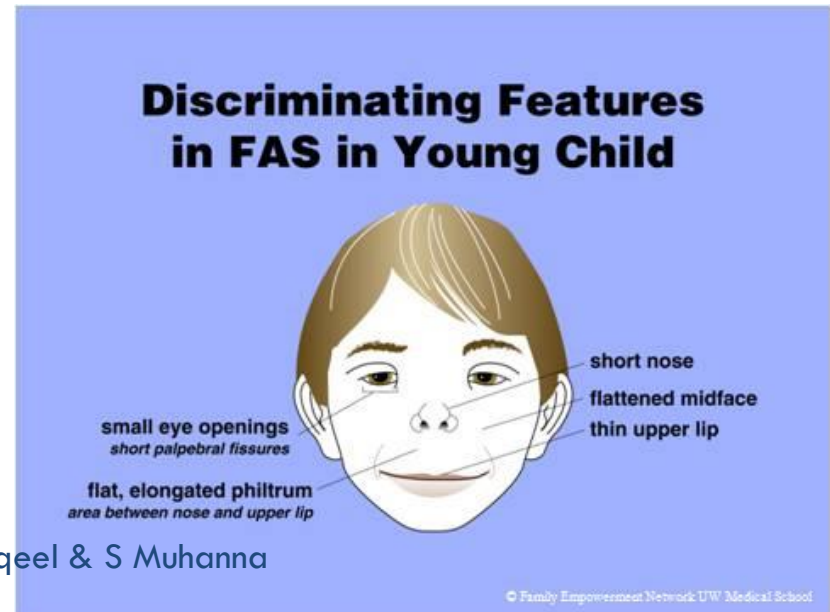
# Effects of alcohol on pregnancy outcome

- Alcohol crosses placenta to fetus- linger in fetal circulation
  - ▣ **Exposure during CPs** can permanently impair organs and tissue formation, growth, health , and mental development
  - ▣ **The fetus has no enzymes to break it down**
- Women who drink alcohol may have poor dietary intake plus alcohol has a –ve effect on availability of certain nutrients → can contribute to harmful effects of exposure during pregnancy
- Heavy drinking during pregnancy  $\geq 4$  drinks/d
  - ▣ Increases risk of miscarriage, stillbirth, and infant death within the 1<sup>st</sup> month after delivery
  - ▣ 40% develop fetal alcohol syndrome

# Fetal alcohol syndrome

## □ **Diagnosis of FAS:**

- Smooth philtrum
- Thin vermilion border: thin upper lip
- Small palpebral fissures: space between top and bottom eyelid when the eye is open- is small
- Neurological disorder (small head circumference, cognitive delay, seizures)
- Reduced growth
- Hyperactivity
- Short attention span
- Behavioral problems
- Small for age





# Nutrition and adolescent pregnancy

## Table 5.18 Risks associated with adolescent pregnancy<sup>122-124</sup>

- Low birth weight
- Perinatal death
- Cesarean delivery
- Cephalopelvic disproportion (head too large for birth canal)
- Preeclampsia
- Iron-deficiency anemia
- Delayed, reduced educational achievement
- Low income

# Growth during adolescent pregnancy

- Very young adolescents who become pregnant within a few years after the onset of menstruation may be at risk due to biological immaturity
  - ▣ They tend to have **shorter gestations** and a higher likelihood of cephalopelvic disproportion
- **Poorly nourished, growing adolescent mothers may compete with the fetus for calories and nutrients- and win**
- Continued gain of ht and wt during pregnancy at the expense of fetal growth
  - ▣ Give birth, on avg, to infants that weigh 155g less than infants of adult women, even if they gain more wt than adults do

- Rates of spontaneous abortion, preterm birth, and LBW are higher in growing than non-growing adolescents
  
- Growing teens experience a surge in blood leptin levels during the last trimester- may decrease maternal use of fat stores and increase utilization of glucose by the mother
  - ▣ ↑ use of glucose by the mother- ↓ E availability to the fetus

# Obesity, excess weight gain, and adolescent pregnancy

- Increasing rates of OW status and obesity among adolescents- places teens at additional risk of poor pregnancy outcomes
  
- Increased risk for:
  - Cesarean delivery
  - Hypertensive disorders of pregnancy
  - Gestational diabetes
  - Delivery of excessively large newborns

# Dietary and other recommendations for pregnant adolescents

- **They may need more calories** to support their own growth as well as that of the fetus
  - ▣ **Nutrient-dense diet:** lead to rates of weight gain within recommendation
- Recommendations for weight gain and protein intake are the same as older pregnant women
- **Higher requirement for Ca**
  - ▣ AI for pregnant teens 1300mg/d (300mg higher than for adult pregnant women)- consumption of 4 daily servings of milk and milk products

- **Nutrition services to improve pregnancy outcome include:**
  - ▣ Nutrition counseling
  - ▣ Nutrition assessment
  - ▣ Intervention
  - ▣ Education
  - ▣ Guidance on weight gain
  - ▣ Follow-up
  - ▣ Psychosocial needs- support/ discussion groups
  - ▣ Home visits
  - ▣ Low income- referral to appropriate food and nutrition programs and other assistance related to health care, housing, and education

# Read HIV/AIDS during pregnancy

