How Sweet It Is! Management of Diabetes: Introduction to Diabetes Mellitus
Mary Lynn McPherson, PharmD, MA, BCPS, CPE

Live Activity Handout
2 slides per page
How Sweet It Is! Management of Diabetes:

Introduction to Diabetes Mellitus

ACTIVITY DESCRIPTION
Pharmacy practitioners need to be able to recognize patients at risk for diabetes mellitus, and how to differentiate between types 1 and 2, because this drives drug therapy decision making.

TARGET AUDIENCE
The target audience for this activity is pharmacists, pharmacy technicians, and nurses in hospital, community, and retail pharmacy settings.

LEARNING OBJECTIVES
After completing this activity, the pharmacist and pharmacy technician will be able to:

- Describe the prevalence of type 1 and type 2 diabetes mellitus in the United States and worldwide
- Compare and contrast three characteristics that differentiate type 1 from type 2 diabetes mellitus
- Describe the pathogenesis of type 1, type 2, and other types of diabetes mellitus

ACCREDITATION
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Knowledge-Based Live Webinar

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ABOUT THE AUTHOR
Mary Lynn McPherson, Pharm.D., BCPS, CPE, is Professor and Vice Chair in the Department of Pharmacy Practice and Science at the University of Maryland School of Pharmacy in Baltimore. She has maintained a practice in both hospice (local and national) and ambulatory care her entire career. At present, Dr. McPherson is the Director of Pharmacotherapy Services at UniversityCare Heritage Crossing in Baltimore where she works primarily with chronic pain patients and patients with diabetes mellitus. Dr. McPherson teaches extensively in the Doctor of Pharmacy curriculum on pain management and end of life care, including didactic and experiential content. She also developed one of the first and few palliative care pharmacy residencies in the U.S.

Dr. McPherson serves on the Board of the Hospice Network of Maryland and is also the President of the American Society of Pain Educators. McPherson is a Fellow in the American Society of Health-Systems Pharmacists, the American Pharmacists Association, the American Society of Consultant Pharmacists and the American Society of Pain Educators. She is Board Certified in Pharmacotherapy, a Certified Diabetes Educator and a Certified Pain Educator. She has received many honors for her work, including the American Pharmacists Association Distinguished Achievement Award in Specialized Practice, the Maryland Pharmacists Association Innovative Practice Award, and the Maryland Society of Health-Systems Pharmacists W. Purdum Lifetime Achievement Award. Dr. McPherson has received many awards for teaching including the Presidential Citation from the Hospice and Palliative Nurses Association, Professor of the Year many times from the School of Pharmacy, University of Maryland Baltimore Founder’s Week Teacher of the Year and the Robert Chalmers Distinguished Educator Award from the American Association of Colleges of Pharmacy. She has written four books, including "Demystifying Opioid Conversion Calculations: A Guide for Effective Dosing," and many book chapters and peer-reviewed articles on pain management, palliative care, and other topics.

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Objectives

1. Describe the prevalence of type 1 and type 2 diabetes mellitus in the United States and worldwide.
2. Compare and contrast three characteristics that differentiate type 1 from type 2 diabetes mellitus.
3. Describe the pathogenesis of type 1, type 2, and other types of diabetes mellitus.

1. Introduction to diabetes mellitus.
2. Lifestyle modification and diabetes mellitus
3. Monitoring diabetes mellitus
4. Medication management Part 1
5. Medication management Part 2
6. Problem solving with diabetes mellitus
7. Coping with diabetes mellitus
8. Risk reduction and management of complications with diabetes mellitus Part 1
9. Risk reduction and management of complications with diabetes mellitus Part 2
10. Diabetes disease state management
Fed State Metabolism

**Carbohydrates**
- 100% breakdown to glucose
- Glycolysis – use of glucose for energy by cells
- Glycogenesis – conversion of glucose to glycogen; stored in liver and muscle
- Lipogenesis – conversion of remaining glucose to fat

**Protein**
- Broken down to amino acids
- In liver, 50% of amino acids converted to glucose for energy or stored as glycogen
- Gluconeogenesis – conversion of protein to glucose

**Fat**
- Only 5% of fat consumed is converted to glucose
- Remaining fatty acids stored in adipose tissue
Glucose Regulation

- Blood glucose (BG) levels fluctuate over a 24-hour period
- Absorptive state (after meals)
  - Absorbing nutrients
  - Blood glucose levels rise, glucose converted to storage form
- Post-absorptive state
  - Digestion complete
  - Absorption of nutrients decreases
  - Blood glucose levels fall
- BG levels need to stay within limits
Effects of Primary Glucoregulatory Hormones

### Pancreas

<table>
<thead>
<tr>
<th>Cells</th>
<th>Hormone</th>
<th>Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>α-cells:</strong></td>
<td>Glucagon</td>
<td>• Stimulates the breakdown of stored liver glycogen</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Promotes hepatic gluconeogenesis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Promotes hepatic ketogenesis</td>
</tr>
<tr>
<td><strong>β-Cells:</strong></td>
<td>Insulin</td>
<td>• Affects glucose metabolism and storage of ingested nutrients</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Promotes glucose uptake by cells</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Suppresses postprandial glucagon secretion</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Promotes protein and fat synthesis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Promotes use of glucose as an energy course</td>
</tr>
<tr>
<td><strong>β-Cells:</strong></td>
<td>Amylin</td>
<td>• Suppresses postprandial glucagon secretion</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Slows gastric emptying</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Reduces food intake and body weight</td>
</tr>
</tbody>
</table>

### Intestine

<table>
<thead>
<tr>
<th>GLP-1</th>
<th>Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• Enhances glucose-dependent insulin secretion</td>
</tr>
<tr>
<td></td>
<td>• Suppresses postprandial glucagon secretion</td>
</tr>
<tr>
<td></td>
<td>• Slows gastric emptying</td>
</tr>
<tr>
<td></td>
<td>• Reduces food intake and body weight</td>
</tr>
<tr>
<td></td>
<td>• Promotes β-cell health</td>
</tr>
</tbody>
</table>


A closer look at insulin

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**IMPACTANCE OF INSULIN**

[Diagram showing the role of insulin in cell function]

**HOW DOES INSULIN WORK?**

[Diagram illustrating the mechanism of insulin action]

For more information - https://www.youtube.com/watch?v=OLHez8gwMgw
So what is diabetes mellitus?

• “A condition characterized by hyperglycemia resulting from the body’s inability to use blood glucose for energy. In Type 1 diabetes, the pancreas no longer makes insulin and therefore blood glucose cannot enter the cells to be used for energy. In Type 2 diabetes, either the pancreas does not make enough insulin or the body is unable to use insulin correctly.”

• See more at: http://www.diabetes.org/diabetes-basics/common-terms/#sthash.OIpR7p0L.dpuf

What’s your type?

DIABETES MELLITUS

TYPES OF DIABETES

Type I diabetes

Type II diabetes
Diabetes Incidence and Prevalence

• 2012 – 29.1 million Americans (9.3% population) had diabetes
  • ~ 1.25 million American children and adults had type 1 diabetes
  • Of the 29.1 million, 8.1 million were undiagnosed
  • 25.9% of Americans > 65 years old had diabetes
• In 2012, 86 million Americans ≥ 20 years old had PREDIABETES (90% don’t know it)
• In 2010, diabetes 7th leading cause of death in US

<table>
<thead>
<tr>
<th>Ethnic Group</th>
<th>Rate of Diagnosed DM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-Hispanic Whites</td>
<td>7.6%</td>
</tr>
<tr>
<td>Asian Americans</td>
<td>9.0%</td>
</tr>
<tr>
<td>Hispanics</td>
<td>12.8%</td>
</tr>
<tr>
<td>Non-Hispanic Blacks</td>
<td>13.2%</td>
</tr>
<tr>
<td>American Indians/ Alaskan Natives</td>
<td>15.9%</td>
</tr>
</tbody>
</table>

• Total costs of diagnosed diabetes in US in 2012 - $245 billion
• More than 20% health care spending is on patients with DM
• Leading cause of kidney failure, lower-limb amputations, adult-onset blindness
• Without major changes, as many as 1 in 3 US adults could have diabetes by 2050
• Lifestyle change in prediabetes can reduce risk of DM by 58%


Signs and Symptoms of Hyperglycemia
Type 1 Diabetes Mellitus

- Previously known as “IDDM” or “juvenile-onset DM”; 5-10% DM
- Due to cellular-mediated autoimmune destruction of the pancreatic β-cells
  - Autoimmune markers include islet cell autoantibodies and autoantibodies to GAD, insulin and other markers
  - Rate of β-cell destruction is variable
  - Rapid in infants and children
  - Slow in adults
- Presentation in ketoacidosis
- Idiopathic Type 1 DM

### Staging of type 1 diabetes

<table>
<thead>
<tr>
<th>Stage 1</th>
<th>Stage 2</th>
<th>Stage 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage</td>
<td>Autoimmunity</td>
<td>Autoimmunity</td>
</tr>
<tr>
<td></td>
<td>Normoglycemia</td>
<td>Dysglycemia</td>
</tr>
<tr>
<td></td>
<td>Presymptomatic</td>
<td>Presymptomatic</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Diagnostic Criteria</th>
<th>Multiple autoantibodies</th>
<th>Multiple autoantibodies</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No IGT or IFG</td>
<td>Dyglycemia: IFG and/or IGT</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FPG 100-125 mg/dl</td>
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<tr>
<td></td>
<td></td>
<td>2-h PG 140-199 mg/dl</td>
</tr>
<tr>
<td></td>
<td></td>
<td>A1c 5.7-6.4% or ≥10% increase in A1c</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Clinical symptoms</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Diabetes by standard criteria</td>
</tr>
</tbody>
</table>

**Testing for Type 1 Diabetes Risk:**
- Measuring islet autoantibodies in relatives of those with type 1 DM may identify individual at risk for type 1 diabetes (particularly in the context of a clinical trial).
- This may allow earlier identification and management of type 1 diabetes.
Type 2 Diabetes Mellitus

- Previously known as “NIDDM” or “adult-onset DM”; 90-95% DM
- Patients with relative (rather than absolute) insulin deficiency, and have peripheral insulin resistance
- Autoimmune destruction of β-cells does not occur
- Most patients with T2DM are overweight or obese (XS weight → insulin resistance; especially apple build)
- May take years to diagnose
- Risk increases with age, obesity, inactivity, prior GDM, HTN, dyslipidemia, certain ethnic groups

Type 2 Diabetes Risk Assessment/Testing

- Testing should be considered in overweight or obese (BMI > 25 kg/m² or > 23 kg/m² in Asian American adults with one or more of the following risk factors:
  - A1c > 5.7%, IGT, or IFG on previous testing
  - First-degree relative with diabetes
  - High-risk race/ethnicity (e.g., African American, Latino, Native American, Asian American, Pacific Islander)
  - Women who were diagnosed with DGM
  - History of CVD
  - Hypertension (> 140/90 mmHg) or on therapy for hypertension
  - HDL cholesterol < 35 mg/dl and/or a TG level > 250 mg/dl
  - Women with polycystic ovary syndrome
  - Physical inactivity
  - Other clinical conditions associated with insulin resistance (e.g., severe obesity, acanthosis nigricans)
- For all patients, testing should begin at age 45 years
- If testing normal, repeat at minimum of 3 year intervals
Criteria for the Diagnosis of Diabetes

<table>
<thead>
<tr>
<th>FPG &gt; 126 mg/dl. Fasting is defined as no caloric intake for at least 8 hours*</th>
</tr>
</thead>
<tbody>
<tr>
<td>OR</td>
</tr>
<tr>
<td>2-h PG &gt; 200 mg/dl during an OGTT. The test should be performed as described by the WHO, using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water*</td>
</tr>
<tr>
<td>OR</td>
</tr>
<tr>
<td>A1c &gt; 6.5%. The test should be performed in a laboratory using a method that is NGSP certified and standardized to the DCCT assay</td>
</tr>
<tr>
<td>OR</td>
</tr>
<tr>
<td>In a patient with classic symptoms of hyperglycemia or hyperglycemic crisis, a random plasma glucose &gt; 200 mg/dl</td>
</tr>
</tbody>
</table>

*In the absence of unequivocal hyperglycemia, results should be confirmed by repeat testing

Categories of increased risk for diabetes (prediabetes)
- FPG 100 mg/dl – 125 mg/dl (IFG), OR
- 2-h PG in the 75-g OGTT 140 mg/dl-199 mg/dl (IGT), OR
- A1c 5.7-6.4%

Signs and Symptoms of Hyperglycemia
Gestational Diabetes Mellitus (GDM)

- Diabetes that is first diagnosed in the second or third trimester of pregnancy that is not clearly either pre-existing type 1 or type 2 diabetes.
- Test for undiagnosed diabetes at the first prenatal visit in those with risk factors, using standard diagnostic criteria
- Test for gestational diabetes mellitus at 24-28 weeks of gestation in pregnant women not previously known to have diabetes
- Test women with gestational diabetes mellitus for persistent diabetes at 4-12 weeks' post-partum, using the oral glucose tolerance test and clinically appropriate non-pregnancy diagnostic criteria
- Women with GDM should have lifelong screening for the development of diabetes or prediabetes at least every 3 years
- Women with GDM found to have prediabetes should receive intensive lifestyle interventions or metformin to prevent diabetes

Testing for T2DM or prediabetes in asymptomatic children*

- Criteria – Overweight
  - BMI > 85th percentile for age and sex
  - Weight for height > 85th percentile
  - Weight 120% of ideal for height
- Plus any two of the following risk factors:
  - Family history of type 2 diabetes in first- or second-degree relative
  - Race/ethnicity (Native American, African American, Latino, Asian American, Pacific Islander)
  - Signs of insulin resistance or conditions associated with insulin resistance (acanthosis nigricans, hypertension, dyslipidemia, polycystic ovary syndrome, or small-for-gestational-age birth weight)
  - Maternal history of diabetes or GDM during the child’s gestation
- Age of initiation: Age 10 years or at onset of puberty, if puberty occurs at a younger age
- Frequency: every 3 years

* Persons aged ≥ 18 years
Type 1 - Type 2 – GMD

<table>
<thead>
<tr>
<th>Factor</th>
<th>Type 1</th>
<th>Type 2</th>
<th>Gestational Diabetes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at detection</td>
<td>• Usually young</td>
<td>• Usually older</td>
<td>• First detected in pregnancy</td>
</tr>
<tr>
<td>Speed of onset</td>
<td>• Usually more acute</td>
<td>• Insidious onset</td>
<td>• Routine testing</td>
</tr>
<tr>
<td>BMI</td>
<td>• Not overweight</td>
<td>• Usually overweight</td>
<td>• Often overweight</td>
</tr>
<tr>
<td>Insulin status</td>
<td>• Insulin deficiency</td>
<td>• Insulin resistance and/or insulin deficiency</td>
<td>• Usually insulin resistance – placental hormones</td>
</tr>
<tr>
<td>Interventions</td>
<td>• Requires insulin from diagnosis</td>
<td>• Diet and lifestyle modification</td>
<td>• Diet and lifestyle modification</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Oral medications</td>
<td>• Insulin</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Injectable medications</td>
<td></td>
</tr>
<tr>
<td>Familial association</td>
<td>• Often random</td>
<td>• Strong family history</td>
<td>• Family history of T2DM</td>
</tr>
</tbody>
</table>

Let’s meet Trent Jamison

• Trent Jamison is 11 years old
• He is 4’7”, 78 pounds (usually)
• His mother brings him in to see the pediatrician with complaints of bed-wetting, general lethargy, and a recent 5 pound weight loss.
• On questioning Trent says he’s been drinking a lot of Gatorade because he’s been thirsty.
• No family history of diabetes.
How about Hector Hernandez?

- Hector is also 11 years old
- Presents to pediatrician for annual physical
- Hector LOVES to play computer games
- 4’10”, 130 pounds
  - BMI 27.2; 98th percentile
- Mom (Sofía) had GDM during her pregnancy with Hector
- What risk factors does Hector have for diabetes? What “type” would Hector likely have?

Acute Complications

<table>
<thead>
<tr>
<th>Level</th>
<th>Glycemic Criteria</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose alert value</td>
<td>≤ 70 mg/dl</td>
<td>Sufficiently low for treatment with fast-acting carbohydrate and dose adjustment of glucose-lowering therapy</td>
</tr>
<tr>
<td>Clinically significant hypoglycemia</td>
<td>&lt; 54 mg/dl</td>
<td>Sufficient low to indicate serious, clinically important hypoglycemia</td>
</tr>
<tr>
<td>Severe hypoglycemia</td>
<td>No specific glucose threshold</td>
<td>Hypoglycemia associated with severe cognitive impairment requiring external assistance for recovery</td>
</tr>
</tbody>
</table>

Diabetic ketoacidosis
Hyperglycemia hyperosmolar non-ketotic syndrome
Chronic Complications

• Vascular disease
  • Macrovascular
    • Coronary artery disease
    • Cerebral vascular disease
    • Peripheral vascular disease
  • Microvascular
    • Retinopathy
    • Nephropathy

• Neuropathic conditions
  • Sensorimotor neuropathy
  • Autonomic neuropathy

• Mixed Vascular and Neuropathic Disease

Glycemic Recommendations
(Non-pregnant adults)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Target</th>
</tr>
</thead>
<tbody>
<tr>
<td>A1c</td>
<td>&lt; 7.0%*</td>
</tr>
<tr>
<td>Preprandial capillary plasma glucose</td>
<td>80-130 mg/dl*</td>
</tr>
<tr>
<td>Peak postprandial capillary plasma glucose</td>
<td>&lt; 180 mg/dl*</td>
</tr>
</tbody>
</table>

Goal adjustment:
• More or less stringent glycemic goals may be appropriate for individual patients.
• Goals should be individualized based on duration of diabetes, age/life expectancy, comorbid conditions, known CVD or advanced microvascular complications, hypoglycemia unawareness, and individual patient considerations.
• Postprandial glucose may be targeted if A1c goals are not met despite reaching preprandial glucose goals
• Postprandial glucose measurements should be made 1-2 h after the beginning of the meal, generally peak levels in patients with diabetes.
Other metabolic goals

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Goal</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood pressure</td>
<td>SBP &lt; 140 mmHg and DBP &lt; 90 mmHg</td>
<td>• &lt; 130/80 mmHg may be appropriate for individuals with CVD</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• 120-160/80-105 mmHg suggested for pregnant patients with DM and chronic HTN</td>
</tr>
<tr>
<td>Lipids</td>
<td>Risk reduction</td>
<td>• TG &gt; 150 mg/dl, and/or HDL &lt; 40 mg/dl for men, &lt; 50 mg/dl for women → intensive lifestyle therapy and optimize BG control</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Patient of any age with ASCVD → High-intensity statin therapy and lifestyle therapy</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Age</th>
<th>+/- ASCVD Risk Factors</th>
<th>Level Statin Therapy + Lifestyle Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 40 years old</td>
<td>+</td>
<td>Moderate or high</td>
</tr>
<tr>
<td>40-75</td>
<td>-</td>
<td>Moderate</td>
</tr>
<tr>
<td>75-79</td>
<td>+</td>
<td>High</td>
</tr>
<tr>
<td>&gt; 75</td>
<td>-</td>
<td>Moderate</td>
</tr>
<tr>
<td>&gt; 75</td>
<td>+</td>
<td>Moderate or high</td>
</tr>
</tbody>
</table>

Let’s meet Sofía Hernandez

- Sofía is 42 years old; she was born in Puerto Rico but has lived in Maryland for over 20 years
- Sofía is 5’4” and weighs 375 pounds
- LOVES regular soda and apple juice
- Wondering if there’s a problem – feels fatigued, not sleeping well, nocturia x 3
- BP 148/94 mmHg (145/92 on repeat)
- Lipid panel: TC 220 mg/dl, TG 260 mg/dl, HDL 28 mg/dl, LDL 140 mg/dl
- Fasting plasma glucose 185 mg/dl

Can Sofía be diagnosed with diabetes based on this data?
Let’s meet Fred Flintstone

• Fred is a 72 year old man, whose wife passed away about a year ago.
• He lives alone and admits “I’m a terrible cook,” consequently he hits the fast food restaurants frequently, and eats a lot of frozen dinner entrees.
• His BG profile is as follows:
  • FPG 118 mg/dl
  • A1c 6.2%
• Does Fred have diabetes?

So what should we do with Fred?

Preview of Coming Attractions

1. Introduction to diabetes mellitus.
2. Lifestyle modification and diabetes mellitus
3. Monitoring diabetes mellitus
4. Medication management Part 1
5. Medication management Part 2
6. Problem solving with diabetes mellitus
7. Coping with diabetes mellitus
8. Risk reduction and management of complications with diabetes mellitus Part 1
9. Risk reduction and management of complications with diabetes mellitus Part 2
10. Diabetes disease state management

What does this mean for Fred?
Can Fred turn things around?
Can Fred prevent progressing to T2DM?
Tune in next time to find out!