Nurse Practitioner Perspective

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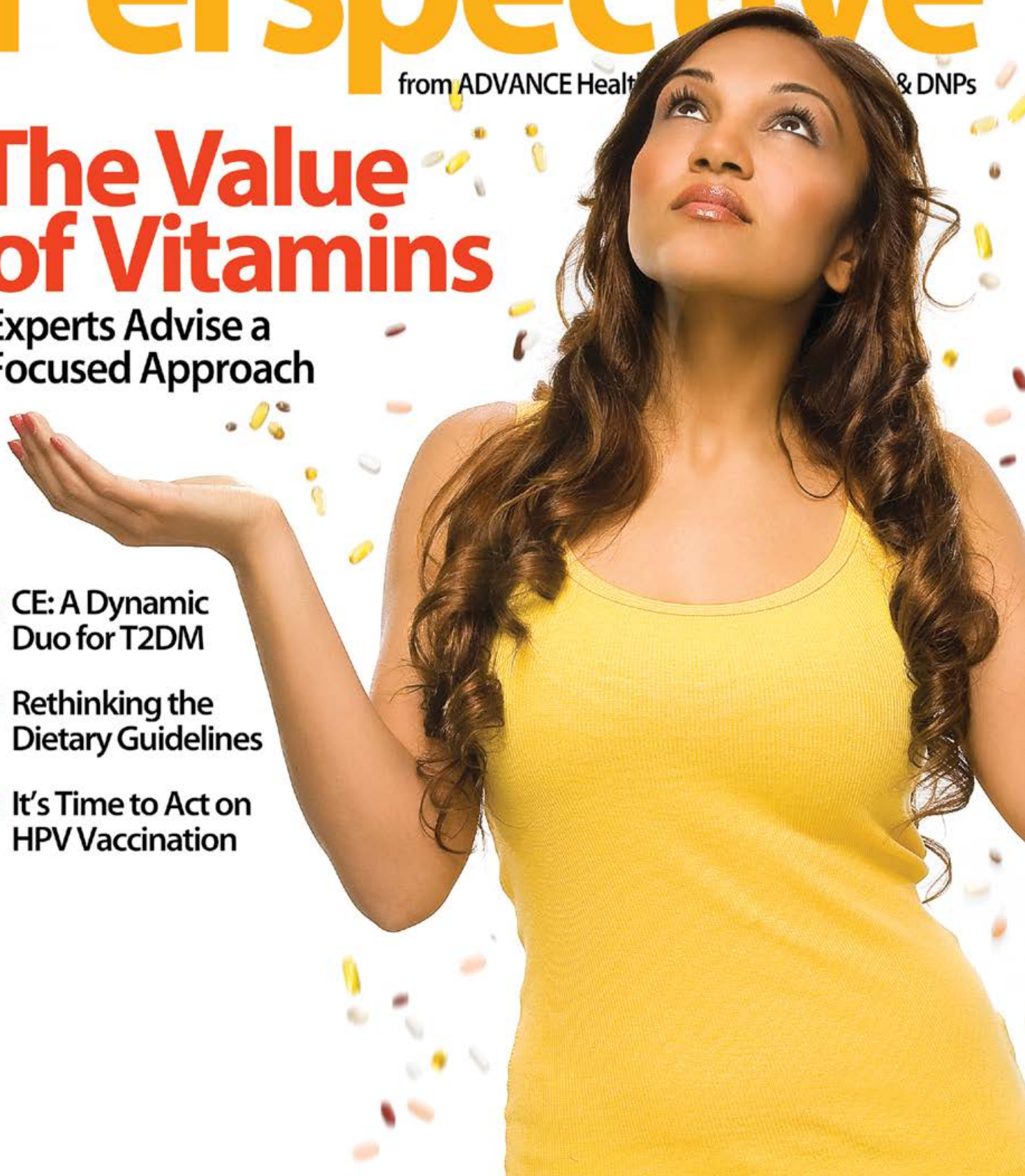
The Value of Vitamins

Experts Advise a Focused Approach



- CE: A Dynamic Duo for T2DM
- Rethinking the Dietary Guidelines
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A Dynamic Duo for T2DM

Aggressive use of lifestyle change and pharmacotherapy

By Cheryl Haas Winter, MS RD, MS APRN, CDE, BC-ADM, FNP-BC

ONE OF THE MOST challenging chronic health conditions to manage is type 2 diabetes mellitus (T2DM). Complex and time-consuming decision-making by providers and significant lifestyle adjustments by patients are key contributors to the dynamics that make management of this condition so difficult. A continuum of risk for poor health outcomes exists in the progression from normal glucose tolerance to overt T2DM.[1] As providers, it is our responsibility to educate patients about the risk factors for prediabetes and T2DM, and to perform screenings every 3 years in patients at risk or annually in patients with two or more risk factors. Guidelines issued earlier this year by the American Association of Clinical Endocrinologists and the American College of Endocrinology identify the following risks for prediabetes and T2DM:[1]

- Age older than 45 without other risk factors
- Cardiovascular disease (CVD) or family history of T2DM
- Overweight or obese (based on body mass index for appropriate ethnic group)
- Sedentary lifestyle
- Member of an at-risk racial or ethnic group: Asian, black, Hispanic, Native American or Pacific Islander
- High-density lipoprotein (HDL) cholesterol <35 mg/dL (0.90 mmol/L) and/or a triglyceride level >250 mg/dL (2.82 mmol/L)
- Impaired glucose tolerance (IGT), impaired fasting glucose (IFG) or metabolic syndrome
- Polycystic ovarian syndrome, acanthosis nigricans or non-alcoholic fatty liver disease
- Hypertension (>140/90 mm Hg) or taking medication for hypertension
- History of gestational diabetes or delivery of a baby weighing more than 9 pounds
- Antipsychotic therapy for schizophrenia and/or severe bipolar disease
- Chronic glucocorticoid exposure
- Sleep disorders in the presence of glucose intolerance (A1C >5.7%, IGT or IFG on previous testing), including obstructive sleep apnea, chronic sleep deprivation and night shift work.

Although as clinicians we understand the significant lifestyle adjustments that are necessary by patients, we must also take responsibility for our frequent clinical inertia. This has become even more crucial since a "legacy effect" or "metabolic memory" may exist when early intensive control of T2DM is achieved.[2] Metabolic memory is now recognized as a phenomenon related to the prolonged harm produced by hyperglycemia. It is believed that in response to hyperglycemia, excess superoxide anion is produced by the mitochondria and leads to disturbances at the nuclear level of the accumulation of potentially harmful substances, such

Learning Objectives

The goal of this continuing education article is to educate nurse practitioners about the management of type 2 diabetes using aggressive pharmacotherapy and lifestyle change. Successful completion of the article and quiz qualifies the reader for 2 continuing education credits. After reading this article, the learner will be able to:

1. Discuss the pathophysiologic defects inherent in type 2 diabetes that should guide the nurse practitioner when prescribing lifestyle treatments.
2. State the pathophysiologic defects inherent in type 2 diabetes that should guide the nurse practitioner when prescribing pharmacologic treatments.
3. Summarize the armamentarium of antidiabetes medications available in the United States and when and how they are prescribed.
4. Explain why aggressive and early treatment of type 2 diabetes is necessary.

This activity is approved for **2 CE contact hours**. The issuer of CE contact hours is Merion Matters, which is approved as a provider of nursing continuing education by ANCC. For details on CE provider numbers, visit the CE test center at www.advanceweb.com/NPPA.

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Table 1
Target Glycemic Levels

Guidelines	HbA1C	Fasting Plasma Glucose	2-Hour Postprandial Glucose Concentration
The American Association of Clinical Endocrinologists[11]	≤ 6.5%	< 110 mg/dL	< 140 mg/dL
American Diabetes Association[8]	< 7%	70-130 mg/dL	< 180 mg/dL

as advanced glycated end products. These adverse effects are not reversed when high blood glucose is corrected and thus may be permanent because of epigenetic changes.[3] Prevention or early and aggressive treatment is vital. Diabetes management is not just about lowering blood glucose. It is also about delaying disease progression and eventual treatment failure.[4]

Longstanding, poorly managed and poorly controlled T2DM can lead to serious microvascular and macrovascular sequelae, including nephropathy, neuropathy, retinopathy, heart disease, stroke, and other potentially devastating complications that greatly compromise quality of life and/or needlessly shorten it. Despite a plethora of antidiabetes medications and an abundance of scientific organizations and professionals promoting ideal eating and exercise plans, T2DM continues to be a worldwide epidemic resulting in significant pain and suffering for patient and their families as well as major financial implications for the healthcare system.

One goal of this article is to briefly review the numerous pharmacotherapies available and to simplify how and when they are prescribed. This requires an understanding of the multiple pathophysiologic defects inherit in this disease. It is beyond the scope of this article to list all potential side effects, precautions and contraindications for these medications, so the discussion focuses on key facts and concepts.

Another goal of this article is to explain why prescribers should forgo longstanding but outdated treatment regimens and open themselves to newer, more aggressive therapies. Traditionally, algorithmic approaches have been utilized for treatment of T2DM. But these often are too rigid, suboptimal and fail to consider important variables for individualized care.[5]

A final goal is to prompt readers to consider that more aggressive target goals for A1C, fasting and postprandial blood glucose may be indicated, especially for patients recently diagnosed. This is based on scientific evidence about the effects of what might be considered normal blood glucose on the brain and other body systems; it can lead to conditions including dementia and cancer.[6-8]

In the limited time we have with our patients, it is our responsibility to communicate to them the urgency of treating T2DM. We must help them understand the need for aggressive lifestyle adjustments, especially in the areas of stress reduction, diet and activity. We have no time to waste. By 2050, 1 in 3 people will have T2DM.[9] Inertia is no longer an option.

Targeting Glycemic Goals

A1C testing, which measures the amount of glycated hemoglobin in the previous 2 to 3 months, has a strong predictive value for diabetes complications.[10] When proteins are glycated, the production of damaging chemicals such as free radicals dramatically increases. Free radicals damage cell protein, lipids and nucleic acids and increase the chemical mediators of inflammation.[11]

With every 1% decrease in A1C, the risk of diabetes-related death is reduced by 21%.[12] The risk of myocardial infarction is reduced by 14%, and the risk of microvascular complications is reduced by 37%.[12] Table 1 lists the glycemic targets set by two major diabetes organizations, the American Association of Clinical Endocrinologists (AACE) and the American Diabetes Association (ADA).[1,10]

Are these values low enough? Research and practice show that certain parameters may prevent the achievement of a more normal A1C: age (life expectancy); duration of disease; presence or absence of microvascular and/or macrovascular complications; and risk for severe hypoglycemia.[13] But for the newly diagnosed patient who has remaining pancreatic beta cells to produce insulin and minimal complications, more aggressive targets may be appropriate. That is because even prediabetic A1C levels (5.7 and 6.4) and normal A1C values have been linked to increased risk for dementia and cancer.[6-8]

A1C is also directly related to the rate at which the brain shrinks each year. An A1C in the range of 5.6 to 5.8 is often considered normal, but one study found it to be associated with the second highest category for brain shrinkage.[14] DeFronzo describes a more rational goal of therapy as an A1C of less than 6%, since the Diabetes Prevention Program found that 12% of patients with impaired glucose tolerance (IGT) and an A1C of 6% already had background diabetic retinopathy.[15]

Targeting Pathophysiologic Defects

In his 2008 lecture upon receiving the Banting Medal for Scientific Achievement, Ralph DeFronzo, MD, described the "Ominous Octet" describing eight core defects of T2DM.[15] From 1987 until this presentation, our understanding of the pathophysiology of T2DM consisted of only three core defects: impaired insulin secretion as a result of pancreatic beta-cell failure; increased hepatic glucose production; and decreased peripheral glucose utilization (insulin resistance in muscle). These three defects are known as the triumvirate of core defects in T2DM. Although pancreatic beta-cell failure is a given at diagnosis, we now know that it occurs much earlier and is more severe than previously thought. By the time the average patient is diagnosed with T2DM, 80% of his or her beta cells have been destroyed.[15]

The Ominous Octet consists of the triumvirate plus accelerated lipolysis in the fat cell; incretin deficiency/resistance in the gastrointestinal tract; hypergluca-

Table 2

Antidiabetes Medications & Their Medical Nutrition Therapy Significance

Drugs, Class/ Site of Action/ System(s) Targeted	Glycemic Elevations Most Affected & Expected A1C Reduction	Recommended SMBG Testing for Effectiveness	Greatest Risk for Hypoglycemia	Medical Nutrition Therapy (MNT) Implications
Secretagogues: Sulfonylureas/Pancreas				
Glipizide *Glyburide* *Glimeperide*	Fasting & postprandial & A1C 1-2%	2-3 times per day, especially fasting	• 4-6 hours after meals & fasting • with missed meal or snacks	• Emphasize weight management techniques • Appropriate snacks and timing • Take before meal; skip drug if not eating
Secretagogues: Non-Sulfonylureas/Pancreas				
Repaglinide *Nateglinide*	Postprandial & A1C 0.5-2%	2 hours after meal	• 1 hour after meal • with missed meal or snacks	• Emphasize weight management techniques • Appropriate snacks and timing • Take before meal; skip drug if not eating
Sensitizers: Biguanides/Liver/Muscle/Adipose Tissue				
Metformin	Fasting & postprandial & A1C,1-2%	Fasting	• None	• May cause weight loss • Limit foods that can cause GI side effects • Take with food to reduce GI side effects
Sensitizers: Alpha-Glucosidase Inhibitors/Small Intestines				
Acarbose *Miglitol*	Postprandial & A1C 0.5-0.8%	2 hours after meal	• None • Treat with glucose tablets (pre-digested carbohydrates)	• Must be taken before carbohydrate-containing meals, with first bite of food • Caution for GI side effects; minimize by reducing foods that cause abdominal bloating & flatulence • May cause low serum iron
Sensitizers: Thiazolidinediones/Muscle/Liver/Adipose Tissue				
Rosiglitazone *Pioglitazone*	Fasting & postprandial & A1C 1-2%	2-3 times per day, especially fasting	• None	• Reduce caloric consumption to avoid weight gain • Reduce sodium to reduce fluid retention • Can be taken with or without food • Adequate osteoporosis protection
Incretin System: DPP-4 Inhibitors/Small Intestines/Pancreas/Liver/Muscle				
Sitagliptin *Saxaliptin* *Linagliptin* *Alogliptin*	Fasting, postprandial & A1C 0.7-1.4%	2-3 times per day	• None	• Weight-neutral • Concentrate on healthy food choices • Can be taken with or without food
Incretin-Mimetic: GLP-1 Receptor Agonists/Small Intestines/Pancreas/Liver/Musce/Brain/Adipose Tissue				
Exenatide & *Exenatide XR* *Liraglutide* *Albiglutide*	Postprandial & A1C 0.5-1.5%	2 hours after meals and fasting	• A reactive hypoglycemia if significant hyperglycemia	• Slows gastric emptying & causes feeling of fullness halfway through meals • Can cause some nausea or feelings of satiety early in meals (avoid greasy or acidic foods; counteract with carbonated beverage or ginger) • Increased water & fiber since potential side effect of constipation • Give 30-60 minutes before eating & do not take after or during eating • Consume at least 30 grams of complex carbohydrate
Amylin Mimetic: Pancreas				
Pramlintide	Postprandial & A1C 0.3-0.6%	Before meals & 2 hours after	• 2-3 hours after meals	• Slows gastric emptying • Causes feeling of fullness halfway through meals • Can cause some nausea or feelings of satiety early in meals • Consume at least 30 grams carbohydrates
Dopamine Agonist: Brain				
Bromocriptine mesylate	Postprandial & A1C 0.6-0.9%	Postprandial	• None	• Take with food within 2 hours of awakening • May cause nausea • Weight neutral
Sodium Glucose Co-transporter 2 Inhibitors: Kidney				
Canogliflozin *Dapagliflozin*	Fasting, postprandial & A1C 0.91-1.16%	Fasting, premeals & postprandial	• None	• May increase LDL cholesterol • May increase risk of hypotension • May promote weight loss
Insulins: Basal Analogs (long-acting)				
Glargine *Detemir*	Fasting & A1C	Fasting & Premeals	• None	• Timing of meals not an issue if receiving proper dose, but carry snacks in case meal is delayed
Insulins: Mealtime Analogs (rapid-acting)				
Lispro *Aspart* *Glulisine*	Postprandial & A1C	Postprandial	• 1-1 ½ hours post-injection	• Insulin-to-carbohydrate ratio education • Hypoglycemic precautions
Insulins: Intermediate-Acting (NPH)				
Humulin N *Novolin N*	Fasting & HbA1C	Fasting & Premeal & Between meal	• 6-12 hours post-injection	• Eat 3 meals daily with between meal snacks • Keep carbohydrate content of meals as consistent as possible
Insulins: Short-Acting (Regular)				
Humulin R *Novolin R*	Postpranidal & HbA1c	Postprandial & Between meal	• 2-4 hours post-injection	• Insulin-to-carbohydrate ratio education • Keep snacks available due to unpredictability
Insulins: Pre-mixed Analogs (combination basal & mealtime)				
Lispro Protamine/Lispro *Aspart Protamine/Aspart*	Fasting, Postprandial & A1C	Fasting, Premeals & Postprandial	• 1-4 hours post-injection	• Eat 3 meals daily • Keep carbohydrate content of meals as consistent as possible

Source: Reference 18. Reproduced with permission

gonemia (pancreatic alpha cell failure); increased glucose reabsorption by the kidney; and insulin resistance in the brain (reducing the feeling of satiety).

The therapy implications as a result of the discovery of the Ominous Octet are as follows:[15]

- Effective treatment of T2DM requires multiple drugs used in combination to correct multiple pathophysiologic defects.
- Treatment should be based on reversal of known pathogenic abnormalities and not simply on reduction of A1C.
- Therapy must be started early to prevent or slow the progressive beta-cell failure that is already well established in patients with impaired glucose tolerance.

Pharmacotherapy

Insulin is the most potent and efficacious treatment for diabetes. When initiated early in the disease course, it can lower insulin resistance, reverse glucotoxicity (chronically elevated plasma glucose levels) and prolong pancreatic beta cell function.[16] Innovative technology can help increase insulin adherence,[17] and insulin can be used alone or in conjunction with most antidiabetes medications.

Table 2 outlines insulin types and 10 other antidiabetes medication classes, along with the core defects they target and their expected glycemic reductions.[18] Hypoglycemia and weight gain are often dreaded side effects of diabetes pharmacotherapy, therefore understanding medical nutrition therapy (MNT) implications that may require timely self-monitoring of blood glucose (SMBG) and other precautions is crucial to minimize these risks. Table 2 also provides guidance in this area. Many of these medications are also marketed in combination forms not listed in the table.

Hypoglycemia is viewed as a contributor to major macrovascular and microvascular events, death from CVD, and all-cause mortality.[19] For example, in the ACCORD trial, mortality was higher among patients who had hypoglycemia than those without hypoglycemia.[20] Hypoglycemia is heavily influenced by the use of sulfonylureas and insulin. Sulfonylureas not only contribute to hypoglycemia and weight gain, they also lead to rapid beta-cell burnout, which then leads to loss of glycemic control and a need for additional antidiabetes agents.[15]

Clinicians likely continue to prescribe sulfonylureas due to their upfront low expense and easy third-party reimbursement. But they are quite expensive for the healthcare system overall, due to hospital visits for hypoglycemia, additional office visits and metabolic consequences associated with weight gain. Clinicians should avoid this class of medication as part of their quest to forgo outdated diabetes pharmacotherapy.

The ADA's traditional algorithm for treating T2DM advocates a stepwise therapeutic approach based on reduction in plasma glucose concentration and not on the known pathophysiologic defects of T2DM.[5] An alternate therapeutic algorithm (see figure) based on the Ominous Octet is believed to result in greater glycemic control durability (less treatment failure), greater preservation of beta-cells, and minimal hypoglycemia and weight gain compared to the ADA algorithm.[15]

At the level of the liver, metformin and thiazolidinediones (TZDs) are potent insulin sensitizers and decrease the rate of hepatic gluconeogenesis (core defect number 2). In muscle, metformin is a weak insulin sensitizer, but TZDs are more potent in this area (core defect number 3). Combining these two together will actually provide an additive effect to reduce the A1C, without the risk of hypoglycemia. In addition, TZDs are excellent sensitizers in adipose tissue and greatly inhibit lipolysis (core defect number 4). They mobilize fat out of muscle, liver and beta-cells, thereby reducing lipotoxicity, which further contributes to insulin resistance and beta-cell burnout. The TZDs have been conclusively shown to improve and preserve beta-cell function (core defect number 1), and they are well-recognized as having positive effects on lipids (increasing HDL and decreasing triglycerides). The glucagon-like peptide 1 agonists (GLP-1 agonists) also appear to preserve beta cell function over the long term (core defect number 1).

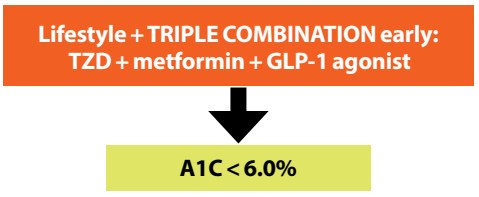
Unfortunately, the most commonly prescribed diabetes drugs in the U.S., metformin and the sulfonylureas, do not exert significant protection on the beta-cell.[15] Therefore, after initial diagnosis when only 20% of beta cell function is thought to be remaining, pharmacotherapy with metformin and sulfonylurea only will eventually lead to total beta-cell destruction. Eight years is the average length of time that 80% of patients will require insulin.

In addition to the GLP-1 agonists protecting beta-cell function, they positively impact four other core defects of the Ominous Octet: defects 2, 5, 6 and 8. Therefore, the triple combination of diabetes drugs in the alternate algorithm targets seven of the eight core defects of T2DM.

At the ADA 2013 Scientific Sessions, Muhammad A. Abdul-Ghani, MD, from the University of Texas Health Science Center at San Antonio, presented interim results of an ongoing open-label, randomized, controlled trial involving 155 drug-naïve patients newly diagnosed (less than 2 years) with T2DM started on triple therapy (metformin + TZD + GLP-1 agonist) or conventional therapy.[21] The conventional therapy group was started on metformin, then glyburide, then glargine insulin added as needed to get to goal A1C.[21] Treatment failure was defined as an A1C of > 6.5% on two consecutive visits, 3 months apart. The starting mean A1C was 8.6%. At the

Pathophysiology-Based Algorithm

(Alternate to ADA's traditional algorithm)



TZD, thiazolidinediones GLP-1 agonist, glucagon-like peptide 1 agonist

2-year interim analysis, patients in the triple therapy group had a mean A1C of 6.0% compared to 6.6% in patients on conventional treatment. Patients in the triple therapy group lost 1.2 kg on average, compared with a weight gain in patients on conventional therapy. Hypoglycemia occurred in 46% of patients on conventional treatment compared to 15% on triple therapy. This study is ongoing.

Triple therapy is simple and effective. It also is potentially affordable, since both metformin and pioglitazone are available in generic form and can be combined in a single pill dosed once per day. Although not yet generic, a once-per-week GLP-1 agonist makes triple therapy simplistic, and as new GLP-1 agonists begin to appear on the market, prices may decline.

Lifestyle Modification

Diet and exercise can reduce A1C by 1% to 2%,[10] thus lifestyle changes have the potential to reduce or eliminate the need for pharmacotherapy. However, lifestyle interventions should not delay pharmaceutical intervention; these two approaches should be used in combination. One study found that the initiation of metformin within 3 months of diagnosis and while A1C was <7% decreased failure rates by 50% compared to patients with diabetes of longer duration and greater A1C values.[22] The mean failure rate for patients in whom metformin was not initiated early was 17% per year, compared to 12.2% per year in the metformin group.

An ADA position paper on managing adults with T2DM, published in 2013, calls for avoidance of beverages sweetened with sugar or any caloric sweetener (including sucrose and high-fructose corn syrup) to reduce the risk of weight gain and worsening of cardiometabolic risk profile.[23,24] The ADA does not endorse or advocate a specific diet or macronutrient distribution. With respect to carbohydrates, the ADA advises that people with diabetes choose nutrient-dense, high-fiber foods as opposed to processed foods. Although a specific recommendation for total fat intake was not established, the ADA guidelines state that the quality of the fat, rather than the quantity of the fat, appears to be a key component. With regard to diet, a paradigm shift for more aggressive outcomes may also be indicated.

Research shows that all patients, including those with diabetes, should spend no more than 90 minutes at a time sitting.[10] Strong evidence exists for the A1C-lowering value of resistance training and for an additive benefit of combined aerobic and resistance exercises in T2DM.[10] Resistance training at least two times per week (with no contraindications) is recommended.[10] Moderate-intensity aerobic activity performed at least 150 minutes/week spread over at least 3 days is also recommended for adults with diabetes.[10] Higher levels of exercise intensity are associated with greater improvements in A1C and fitness.[10]

Early, Aggressive Treatment

Generally, when A1C is > 9.0, the patient is considered to be in glucotoxicity and experiences the classic symptoms of diabetes: polyuria, polydipsia, polyphagia and weight loss. Without relieving the pancreas from this toxicity or exposure to excess blood glucose, pancreatic beta-cell destruction continues to be a risk, and the diabetes condition continues to progress.

The ability to achieve goal A1C is greatly improved when treatment is based on reversing the known pathophysiologic defects. Early and aggressive treatment of patients with T2DM can lead to maintenance of normal plasma glucose concentrations and reduce potential complications, as well as enhance quality of life. DeFronzo made the analogy that "we don't treat breast cancer with one drug and wait for it to fail and hope we have a good outcome."[21] So why do we do this with T2DM? A paradigm shift in the treatment of T2DM is warranted. ■

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Questions

1. Because there is a continuum of risk for poor health outcomes in the progression from normal glucose tolerance to overt T2DM, what do providers have a responsibility to do?
a. Educate patients about the risk factors for prediabetes and T2DM
b. Perform screening every 5 years in patients at risk
c. Perform screening annually in patients with three or more risk factors
d. Place all patients at risk on metformin

2. Which answer choice describes metabolic memory?
a. It is reversed when high blood glucose is corrected.
b. It is a response occurring with hypoglycemia, as a result of a fast metabolism.
c. It is a phenomenon related to the prolonged harm produced by hyperglycemia.
d. It is another name for metabolic syndrome.

3. Which one of the following is a correct statement about A1C values?
a. An A1C level of 4.7 to 5.4 has been linked to increased risk of dementia and cancer.
b. An A1c in the range of 5.8 to 6.0 is often considered normal.
c. DeFronzo describes a more rational goal of therapy as an A1C of less than 7%.
d. The Diabetes Prevention Program found that 12% of patients with impaired glucose tolerance (IGT) and an A1C of 6% already had background diabetic retinopathy.

4. Which of the following is considered part of the "triumvirate" of core defects in T2DM?
a. impaired insulin secretion as a result of pancreatic beta-cell failure
b. accelerated lipolysis in the fat cell
c. increased glucose reabsorption by the kidney
d. decreased low-density lipoprotein levels

5. Which answer choice states side effects of pharmacotherapy in T2DM?
a. hyperglycemia and weight gain
b. weight loss and dizziness
c. hypoglycemia and weight gain
d. nausea and weight loss

6. Which class of diabetes pharmacotherapy should clinicians avoid due to their risk for hypoglycemia and weight gain?
a. thiazolidinediones c. GLP-1 agonists
b. sulfonylureas d. biguanides

7. At the level of the liver, which two diabetes medications together are potent insulin sensitizers and decrease the rate of hepatic gluconeogenesis (core defect number 2)?
a. metformin and thiazolidinediones
b. metformin and DPP4 inhibitors
c. TZDs and GLP-1 agonists
d. Sulfonylureas and metformin

8. Interim results of an ongoing study from the University of Texas Health Science Center at San Antonio show improved A1C of 6.0% in the triple therapy group, compared with 6.6% in the conventional treatment group and weight loss compared to weight gain and minimal hypoglycemia in the triple therapy group compared to the conventional therapy group. Which three diabetes pharmacotherapies are included in the triple therapy group?
a. metformin, a thiazolidinedione and sitagliptin
b. metformin, a GLP-1 agonist and glyburide
c. metformin, a thiazolidinedione and glargine
d. metformin, a thiazolidinedione and a GLP-1 agonist

9. Which of the following is true about American Diabetes Association (ADA) nutrition therapy recommendations for the management of adults with diabetes?
a. The ADA calls for the avoidance of beverages sweetened with sugar or any caloric sweetener.
b. The ADA advocates for a specific diet and macronutrient distribution for diabetes of 35% carbohydrate, 10% protein and 55% fat.
c. With respect to carbohydrates, the ADA advises that people with diabetes choose nutrient-dense, low-fiber foods.
d. Diet should be used for at least one year before adding pharmacotherapy.

10. On the subject of exercise recommendations, which of the following is NOT an accepted piece of advice for patients with diabetes?
a. Patients with diabetes should perform resistance training at least two times per week (in the absence of contraindications).
b. Patients with diabetes should engage in moderate-intensity aerobic physical activity at least 150 minutes/week spread over at least 3 days/week.
c. Patients with diabetes should reduce their sedentary times, specifically by breaking up extended amounts of time (>90 minutes) spent sitting.
d. Higher levels of exercise intensity above moderate intensity are strongly prohibited.

Evaluation

On a scale of 1-5, with 1 meaning that you strongly disagree and 5 that you strongly agree, please respond to the following statements. You must complete this evaluation to receive your certificate.

1 = poor **2** = fair **3** = good **4** = very good **5** = excellent

1. I can discuss the pathophysiologic defects inherent in type 2 diabetes that should guide the nurse practitioner when prescribing lifestyle treatments.
2. I can state the pathophysiologic defects inherent in type 2 diabetes that should guide the nurse practitioner when prescribing pharmacologic treatments.
3. I can summarize the armamentarium of antidiabetes medications available in the United States and when and how they are prescribed.
4. The objectives relate to the overall goal of the article.
5. The article is well-written and logically organized, and defines terms adequately.

A Dynamic Duo for T2DM

NPP13 (2 credits)

Registration & Answer Form

Three options are available to earn CE contact hours: website, fax and postal mail. This activity is eligible for CE credit for 2 calendar years after publication **(July 13, 2017)**. Visit the CE Test Center at **www.advancewebcom/NPPA** to receive instant test results and a printable CE certificate upon passage. Or fax the completed form and credit card information to (610) 278-1426. Or send the completed answer form and registrant information to Merion Matters CE Program for NPs, 2900 Horizon Dr., King of Prussia PA 19406. Include a check for $10 payable to Merion Matters.

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Questions

	A	B	C	D		A	B	C	D
1.	○	○	○	○	6.	○	○	○	○
2.	○	○	○	○	7.	○	○	○	○
3.	○	○	○	○	8.	○	○	○	○
4.	○	○	○	○	9.	○	○	○	○
5.	○	○	○	○	10.	○	○	○	○

Evaluation

	1	2	3	4	5
1.	○	○	○	○	○
2.	○	○	○	○	○
3.	○	○	○	○	○
4.	○	○	○	○	○
5.	○	○	○	○	○

How many minutes did you need to complete this CE offering (reading the article and taking the test)? _____

Please provide suggestions for future CE topics. _____

_____

Statement of Completion

I attest to having completed the CE activity.

Signature _____ Date _____

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