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# Pathophysiology of Insulin Resistance and Type II Diabetes Mellitus

Drew Linardi  
linardi@otterbein.edu

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# Type II Diabetes Mellitus

Drew Linardi RN BSN

Otterbein University, Westerville, Ohio

## Introduction

- The topic I have chosen for my poster presentation is type II diabetes mellitus: a case of beta cell underproduction and insulin resistance (Kahn, Cooper, & Del Prato, 2014).
- The rationale for selecting this topic was to clearly illustrate the pathophysiological process of type II diabetes mellitus as well as the negative impact hyperglycemia has on clinical outcomes (Kahn et al., 2014).
- I was personally motivated in selecting this topic as I am a type II diabetic patient who contracted my condition via a viral infection from a bee sting. I have been personally blessed with a fantastic endocrinologist whom together keeps my A1C level in the mid to upper five range.
- Working in a cardiac recovery unit, patients are frequently observed having poor or worsening cardiovascular and vascular outcomes due to uncontrolled type II diabetes.
- Type II diabetes presents itself in one of two ways (Kahn et al., 2014). First, beta cells within the pancreas are unable to produce adequate amounts of insulin to keep blood glucose levels regulated (Kahn et al., 2014). Second, insulin resistance occurs where individuals who are typically overweight or obese have been producing too much insulin due to lessened sensitivity in appropriate cells (Kahn et al., 2014). Eventually the beta cells cannot keep up the excess insulin production and production declines despite there being an increased demand for it (Kahn et al., 2014).
- As of 2014, it was estimated that 29 million individuals in the United States were diabetic with an additional 86 million afflicted with prediabetes ("CDC," 2014).
- A diagnosis of type II diabetes is made when a patient has two separate tests indicating an A1C of 6.5 or higher ("MayoClinic," 2018).
- Treatment of type II diabetes includes weight loss, proper diet, exercise, possible medication and insulin therapy, and frequent blood glucose monitoring ("MayoClinic," 2018).

## Signs & Symptoms

- Signs and symptoms of type II diabetes include increased thirst, increased hunger, dry mouth, frequent urination, unexplained weight loss, fatigue, blurred vision, numbness or tingling in the hands or feet, dry and itchy skin, and slow healing of cuts and sores ("Cleveland Clinic," 2018).
- Type II diabetics can go into diabetic ketoacidosis exhibiting signs such as fruity breath however this is less likely to occur compared to type I diabetics ("Medline Plus," 2018). Diabetic ketoacidosis is typically less severe in type II diabetics ("Medline Plus," 2018). Diabetic ketoacidosis is brought on in type II diabetics by medication noncompliance, or onset of infection, resulting in sustained uncontrolled hyperglycemia ("Medline Plus," 2018).
- Long term effects of uncontrolled type II diabetes include retinopathy, neuropathy, nephropathy, elevated coronary artery disease risk, elevated cerebrovascular disease risk, and elevated peripheral arterial disease risk ("Johns Hopkins Medicine," 2018).

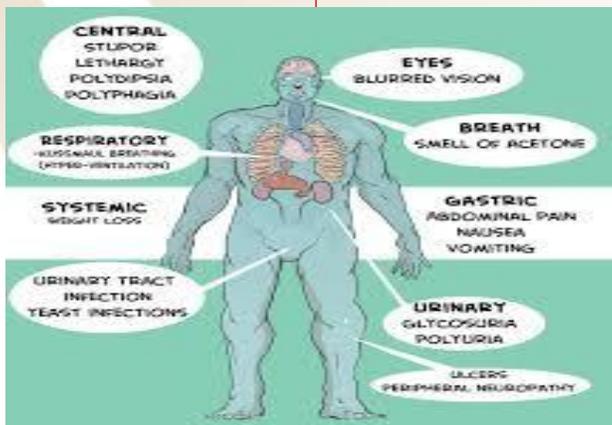


Figure 1 Type II Diabetes Symptoms & Warning Signs (Lupo D.C., 2018).

## Patient Presentation

- Mr. R is a 59 year old Hispanic-American male presenting to the health clinic with complaints of fatigue, thirst, and increased appetite times three weeks despite eating, drinking, and sleeping more than usual.
- Upon a complete history and physical examination Mr. R is diagnosed with type II diabetes. Mr. R is obese with a BMI of 32, hypertensive with a BP of 172/104, has a sedentary lifestyle, accucheck of 262, and a HGA1c of 10.3 ("NIDDK," 2018).

## Risk Factors

- Mr. R has several risk factors including obesity, hypertension, age, Hispanic ethnicity, and a sedentary lifestyle ("NIDDK," 2018).
- A definitive diagnosis of type II diabetes can be made after Mr. R's HGA1c registers at 10.3 and his fingerstick shows a blood glucose of 253 (Accu-Check," 2018).

## Pathophysiology of Type II Diabetes Mellitus and Insulin Resistance.

Type II diabetes mellitus and insulin resistance are complex intertwined conditions that involve some form of eight different pathophysiological abnormalities (Cersosimo, Triplitt, Solis-Herrera, Mandarino, and DeFronzo, 2018). These eight mechanisms include decreased peripheral glucose uptake in mainly muscle combined with altered endogenous glucose production, increased free fatty acid lipolysis contributing to elevated glucose output, a reduction in peripheral glucose utilization combined with beta cell dysfunction (Cersosimo et al., 2018).

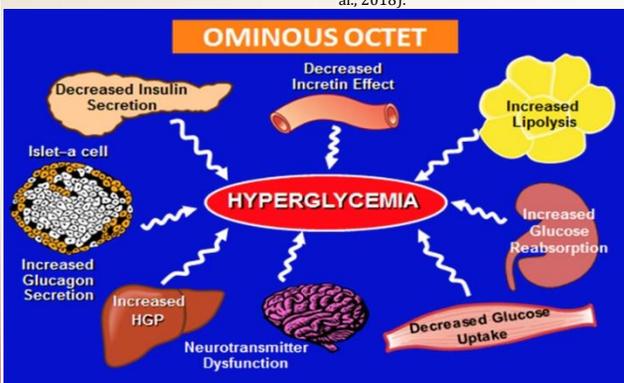


Figure 2 Ominous Octet and Hyperglycemia (Cersosimo et al., 2018).

## Pathophysiology continued.

These pathological mechanisms are more commonly known as the ominous octet leading to hyperglycemia in type II diabetes depicted above in figure 2 (Cersosimo et al., 2018).

In insulin resistance the kidneys increase rates of glucose reabsorption at times to levels of 220 to 250 mg/dl before reaching a maximum reabsorptive capacity (Cersosimo et al., 2018).

Once reabsorptive capacity is reached elevated blood glucose occurs in peripheral circulation (Cersosimo et al., 2018).

Beta cells at first are able to increase insulin production but eventually are unable to keep up with the elevated demand (Cersosimo et al., 2018). At the same time, an inappropriate release of glucagon from the alpha cells of the pancreas occurs post-prandially (Cersosimo et al., 2018). The combination of impaired insulin production and elevated glucagon secretion is known as the decreased incretin effect occurring in the gastrointestinal tract (Cersosimo et al., 2018). The central nervous system also plays a major role in type II diabetes as the hypothalamus contributes to insulin resistance by impairing insulin's capacity to suppress glucose production (Cersosimo et al., 2018).

## Significance of Pathophysiology.

With an increasingly overweight and obese population insulin resistance I becoming more commonplace (Kahn et al., 2014).

Insulin resistance and obesity often times go hand in hand when it comes to type II diabetes (Kahn et al., 2014). There is a feedback loop that exists between beta cells and insulin sensitive tissues (Kahn et al., 2014).

Insulin released by the beta cells facilitates the uptake of glucose, fatty acids, and amino acids (Kahn et al., 2014).

The tissue of overweight and obese individuals becomes less sensitive to insulin increasing the demand on the beta cells to produce greater amounts of insulin to maintain normal blood glucose levels (Kahn et al., 2014).

Over time the beta cells fatigue and can no longer keep up with the increased demand for insulin production resulting in hyperglycemia (Kahn et al., 2014). This is a frequent occurrence in obese and overweight individuals which can be rectified with weight loss (Kahn et al., 2014).

As of 2018, nearly 70% of Americans are either overweight or obese ("AHA," 2018).

Obesity is defined as being at or greater than 20% of an individual's ideal weight ("AHA," 2018).

Currently there are nearly 13 million children ages 2-19 who are obese with an additional 11 million classified as overweight ("AHA," 2018).

The figures yield a total of 23.9 million children qualifying as either overweight or obese ("AHA," 2018).

Roughly 35% of adults in the United States qualify as obese totaling 78 million individuals ("AHA," 2018).

Individuals qualifying as overweight or obese are at increased risk of developing diabetes, heart disease, hypertension, and stroke ("AHA," 2018).

As healthcare evolves to a primary prevention based focus individuals will need counseling regarding ideal body weight, portion control, prevention of obesity, as well as the risks of concomitant conditions such as diabetes and heart disease (Macha & McDonough, 2012).

## Implications for Care.

A study was conducted evaluating patients undergoing noncardiac surgery from the years 2004 to 2013 encompassing roughly 10.5 million individuals (Newman, Wilcox, Smilowitz, and Berger, 2018).

The study evaluated MACCE outcomes in diabetic patients versus nondiabetic patients (Newman et al., 2018). MACCE outcomes were defined as major adverse cardiovascular and cerebrovascular events (Newman et al., 2018).

The study found that diabetic patients exhibited a statistically significant increased likelihood of experiencing a MACCE event including all diabetes: mortality, myocardial infarction, and ischemic stroke versus nondiabetic patients (Newman et al., 2018).

The increased risk of MACCE events in diabetic patients was supported with a p value of <0.001 (Newman et al., 2018). The study found that from 2004 to 2013 the amount of diabetic patients presenting for surgery had increased significantly from the studies inception (Newman et al., 2018).

Rehman and others found that patients presenting with ischemic stroke were more often found to have hyperglycemia and raised hemoglobin A1C than not (Rehman, Alam, & Rehman, 2017).

## Conclusion

Hayward and others conducted a study evaluating 1,791 military veterans with type II diabetes and randomly assigned half the subjects to intensive glucose control and the other half to standard therapy (Hayward et al., 2015).

The results revealed that individuals in the intensive glucose therapy group experienced significantly fewer major cardiovascular events than those assigned to the standard therapy group (Hayward et al., 2015).

Current projections have shown that by the year 2030 roughly 552 million individuals will have diabetes (Laakso & Kuusisto, 2014).

As healthcare providers, it is imperative to reverse this trend and limit the micro and macrovascular complications of type II diabetes and insulin resistance (Laakso & Kuusisto, 2014).

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