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### Type II Diabetes

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# Type II Diabetes

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## Introduction

The chosen topic to research is Diabetes Mellitus Type II (DMII). DMII has become a rampant disease in the United States and is becoming more common. The United States has an alarming rate of new diagnosis of DMII (Chen, 2012). One of the most disappointing facts is that it can be prevented. DMII is not a disease that one has to have for a lifetime. An important detail of this disease is that it has so many comorbidities. Many people in the general population think that they can just take a pill or take insulin to control it. However, even this will not be enough to counteract the debilitating, and possibly life ending, symptoms of this disease.

## Signs and Symptoms

There are many signs and symptoms of diabetes. While polyuria, polydipsia, and polyphagia may be common symptoms, they are not always all present with DMII and can sometimes be (Scheen, 2012). DMII symptoms often develop slowly and it's very possible to have the disease and not know it. Other symptoms include fatigue, weight loss, blurred vision, and slow healing wounds (Scheen, 2012). Common signs and symptoms of type 2 diabetes include fatigue, especially after eating as well as feeling hungry even after you have just eaten. Many people also experience polyuria as well as polydipsia. Others experience slower wound healing times as well as frequent infections and other skin issues such as psoriasis. Type 2 diabetics may also experience increased hunger. This is due to the lack of insulin moving sugar into cells which will then cause muscles and organs to be triggered into thinking they are starving (D'Adamio & Capril, 2011).

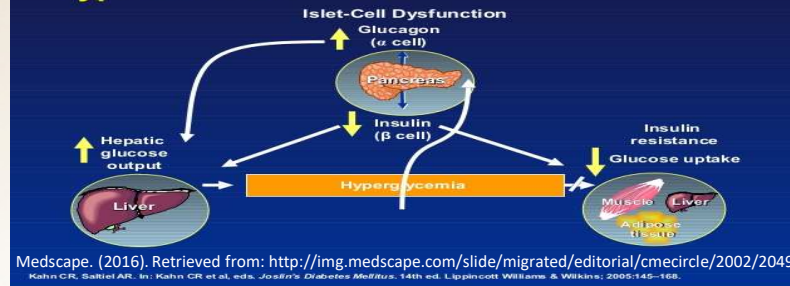
## Underlying and Significance of Pathophysiology

DMII occurs when the body becomes resistant to insulin or when the pancreas stops producing enough insulin. It is not known why this happens, but factors such as obesity and genetics can play a factor in developing this disease (McCance & Huether, 2014). Other factors that may illicit this disease are factors such as poor diet, increased stress, smoking, and history of or current medical conditions.

These factors lead to insufficient amounts of insulin from the beta cells in insulin resistance. Normally, the liver will suppress glucose being released into the body. However, with insulin resistance cells are not able to react to normal levels of insulin. This is also associated with increased breakdown of lipids and higher retention of salt and water in the kidneys. It is important to note that not all people with insulin resistance will develop DMII (D'Adamio & Capril, 2011). Many people will not develop DMII and have classic symptoms while others are genetically inclined to develop the disease.

Insulin resistance is the crux of DMII. Insulin resistance happens when cells fail to normally respond to the hormone insulin. Normally the body will release insulin in the presence of glucose in the blood stream and utilize glucose instead of fat for energy. When insulin resistance is not present, the body will use glucose appropriately and level will remain within normal limits. When insulin resistance is present it prevents the cells from utilizing glucose and leads to hyperglycemia. Hyperglycemia will eventually start to impair beta cells (b cells), in the pancreas, and cause more insulin to be produced therefore creating an even higher level of insulin in the blood and development of DMII (McCance & Huether, 2014).

## Major Pathophysiologic Defects in Type 2 Diabetes



Obesity is one of the largest contributors to the disruptions in several functions in the body. Insulin resistance leads to elevated levels of free fatty acids which can lead to the changes in insulin incretin and glucagon secretion which can lead to inflammation. Inflammation of cytokines from the abdomen can contribute to insulin resistance and can also lead to fatty liver, atherosclerosis, and dyslipidemia (De La Heras, 2017).

The most significant change in DMII is that there is an alteration in the balance between insulin sensitivity and insulin secretion. The change in glucose metabolism is from the fall in B-cell function which damages the pathways of insulin sensitivity and secretion. Studies suggest that diabetes does not develop until B-cells fail completely in regard to compensation for the peripheral insulin resistance state, however, the progressive nature of DMII indicates a progressive decline in B-cell function (Chen, 2012). This along with the other factors including B-cell mass can affect the B cell function loss. This can be due to metabolic changes such as insulin resistance, however, other studies suggest that pre-existing conditions as well as genetics can play a great factor in determining risk for development of the DMII. Early onset of DMII increases risk of mortality and other comorbidities.

## Implications of Nursing Care

One of the key components in nursing care for DMII patients is education. Education on not only the disease and its consequences, but education on lifestyle choices that can help end DMII. Betterment of nutrition and exercise are far more effective than taking medications (D'Adamio & Capril, 2011). This along with management of the comorbidities while they disease is in process will be the main talking points for nurses. While the patient is experiencing the disease it is important to monitor blood glucose and lab levels. Regular follow up with the Primary physician or clinic is key to managing and defeating DMII.

## Conclusion

DMII can be a debilitating and lethal disease if not properly managed (McCance & Huether, 2014). The complications of insulin resistance and sensitivity become the beginning of a slippery slope of a downward health spiral. DMII does not have to be a lifelong disease. Proper diet and exercise can decrease the chances of this disease and help



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