

Pathophysiology and Role of Environmental Factors in Type 2 Diabetes Mellitus

Jones Sam*

Department of Biochemistry and Clinical Analysis, University of Sarajevo, Herzegovina

Introduction

Peripheral insulin resistance, poor hepatic glucose production control, and decreasing β -cell activity define the pathophysiology of type 2 diabetes mellitus, finally leading to β -cell failure. Initial insulin secretion deficits and, in many individuals, relative insulin insufficiency in conjunction with peripheral insulin resistance are thought to be the main occurrences. Environmental variables such as dietary factors, endocrine disruptors and other environmental pollutants, and gut microbiome composition have all been linked to type 1 and type 2 diabetes. Obesity and insulin resistance, in addition to their well-known involvement in type 2 diabetes, may act as type 1 diabetes accelerators. In contrast, in a subgroup of patients diagnosed with type 2 diabetes, islet autoimmunity linked to putative environmental factors (e.g., food, infection) may play a role [1].

Pathophysiology in type 2 diabetes mellitus

The transmembranous transport of glucose and the coupling of glucose to the glucose sensor are required for the insulin response to begin. By maintaining the protein and preventing its breakdown, the glucose/glucose sensor complex causes an increase in glucokinase. The initial step in connecting intermediate metabolism to the insulin secretory system is to induce glucokinase. Glucose transfer appears to be substantially decreased in type 2 diabetes patients' β -cells, moving the control point for insulin production from glucokinase to the glucose transport system. Sulfonylureas help to correct this problem [2].

The second phase release of freshly produced insulin is hindered later in the disease's course, an impact that can be partially restored, at least in some patients, by reinstating tight glycemia control. Desensitization, also known as β -cell glucotoxicity, is the result of glucose's paradoxical inhibitory impact on insulin release, and may be due to the buildup of glycogen within the β -cell as a result of prolonged hyperglycemia. Sorbital buildup in the β -cell or nonenzymatic glycation of β -cell proteins have both been postulated as possibilities [3].

Defective glucose potentiation in response to nonglucose insulin secretagogues, asynchronous insulin release, and a reduced conversion of proinsulin to insulin are all abnormalities in β -cell activity in type 2 diabetes mellitus. In patients with previous gestational diabetes, an impairment among first phase insulin secretion may serve as a marker of risk for type 2 diabetes mellitus in family members of persons with type 2 diabetes mellitus. Impaired first-phase insulin secretion, on the other hand, will not result in impaired glucose tolerance.

The precise relationship between glucose and insulin level as a surrogate measure of insulin resistance has been questioned because chronic hyperinsulinemia inhibits both insulin secretion and action, and hyperglycemia can impair both the insulin secretory response to glucose as well as cellular insulin sensitivity. The insulin sensitivity of lean type 2 diabetes patients over 65 years of age was shown to be comparable to that of age-matched nondiabetic controls. Furthermore, obesity is nearly always prevalent in type 2 diabetes individuals who are insulin resistant. Some believe that insulin resistance in type 2 diabetes is solely attributable to the presence of increased adiposity, because

obesity or an increase in intraabdominal adipose tissue is related with insulin resistance in the absence of diabetes. Insulin resistance is also present in hypertension, hyperlipidemia, and ischemic heart disease, all of which are typical complications of diabetes.

In first degree relatives of type 2 diabetes patients, insulin's capacity to inhibit hepatic glucose production both fasting and postprandially is normal. As type 2 diabetes develops, both fasting and postprandial glucose production rise. Despite the presence of insulin, hepatic insulin resistance is characterised by a significant reduction in glucokinase activity and a catalytic enhanced conversion of substrates to glucose. As a result, the liver in people with type 2 diabetes is set up to both overproduce and underuse glucose. Increased hepatic glucose production may be linked to higher free fatty acid levels observed in type 2 diabetes [4].

Role of environment in type 2 diabetes mellitus

When β -cells fail to produce enough insulin to meet demand, type 2 diabetes develops, generally in the setting of increasing insulin resistance. Islet autoimmunity is seen in a small percentage of patients with type 2 diabetes. Type 2 diabetes has a complicated genetic and environmental aetiology, and obesity is a key risk factor.

With ectopic fat accumulation in the liver and muscle, insulin resistance occurs. Fat accumulation in the pancreas may also lead to B-cell function decrease, islet inflammation, and final B-cell mortality. Type 2 diabetes develops at various levels of BMI/body fat composition in different people, with Asians and Asian Americans having a lower BMI. There may be a personal "fat threshold" at which ectopic fat buildup develops, increasing insulin resistance and causing B-cell decompensation in those who are sensitive.

Weight reduction enhances insulin sensitivity in the liver and skeletal muscle, as well as the formation of pancreatic fat. In prediabetes and type 2 diabetes with recent start, insulin secretion defects are at least partly reversible with calorie restriction and weight loss. Even with the substantial weight reduction associated with bariatric surgery, it is difficult to cure long-standing diabetes [5].

Obesity and diabetes are linked to both reduced sleep duration and increased sleep time. Obstructive sleep apnea is linked to type 2 diabetes and metabolic syndrome, and it lowers sleep length and quality. The current "24-hour culture" may result in less sleep and, as a result, a higher risk of type 2 diabetes. While there are correlations

*Corresponding author: Jones Sam, Department of Biochemistry & Clinical Analysis, University of Sarajevo, Herzegovina; E-mail: jonesam@ius.edu.ba

Received August 30, 2021; Accepted September 13, 2021; Published September 20, 2021

Citation: Sam J (2021) Pathophysiology and Role of Environmental Factors in Type 2 Diabetes Mellitus. J Diabetes Clin Prac 4: 133.

Copyright: © 2021 Sam J. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

with other environmental variables, no direct causal links have been shown to far.

References

1. Porte D (1991) B cells in type 2 diabetes mellitus. *Diabetes* 40:166–180.
2. O'Rahilly S, Turner RC, Matthews DR (1988) Impaired pulsatile secretion of insulin in relatives of patients with non-insulin dependent diabetes mellitus. *N Engl J Med* 318:1225–1230.
3. Kissebah A, Freedman D, Peiris A (1989) Health risk of obesity. *Med Clin North Am* 73:111–138.
4. Charles MA, Eschwege E, Thibault N, et al. (1997) The role of non-esterified fatty acids in the deterioration of glucose tolerance in Caucasian subjects: results of the Paris prospective study. *Diabetologia* 40:1101–1106.
5. Colditz GA, Willett WC, Rotnitzky A, Manson JE (1995) Weight gain as a risk factor for clinical diabetes mellitus in women. *Ann Intern Med* 122:481–486.