Pathophysiology and Role of Environmental Factors in Type 2 Diabetes Mellitus

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Introduction

Peripheral insulin resistance, poor hepatic glucose production control, and decreasing -cell activity de ne the pathophysiology of type 2 diabetes mellitus, nally leading to -cell failure. Initial insulin secretion de cits and, in many individuals, relative insulin insu ciency 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

e 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. e 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].

e 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 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

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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, Thibult N, et al. (1997) The role of non-esterifed 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.