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RELATIONSHIP OF DIABETES MELLITUS AND PHYSIOLOGIC FACTORS – A MINI REVIEW

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ABSTRACT

Diabetes is presently the leading cause of end-stage renal disease (ESRD) in both the US and Europe. Type 2 diabetes is growing more common, people with diabetes live longer, and patients with diabetic ESRD are increasingly being accepted for treatment in ESRD programs that had previously turned them away. Twenty to thirty percent of diabetics with type 1 or type 2 have nephropathy; however, only a far lower percentage of those with type 2 diabetes go on to develop end-stage renal disease. These individuals make up over half of all diabetic patients starting dialysis due to the much greater incidence of type 2 diabetes. Several treatments work, according to new research.

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1. INTRODUCTION

Diabetes Mellitus (DM) is a set of common metabolic illnesses that share the hyperglycemia phenotype. Diabetes Mellitus has several subtypes that are caused by a complex interplay of genetics, environmental factors, and lifestyle decisions [1–3]. Factors leading to hyperglycemia may include decreased insulin secretion, decreased glucose utilisation, and increased glucose generation, depending on the aetiology of Diabetes Mellitus. People with TIIDM are more prone to short and chronic physiological problems, which frequently leads to an increase in mortality [4,5]. The bulk of the 382 million individuals with type 2 Diabetes Mellitus is between the ages of 40 and 59, with four fifth of existing population in low- and middle-income countries. Diabetes of all sorts is on the rise, particularly type 2 Diabetes Mellitus: the number of persons with Diabetes Mellitus will rise by 55% by 2035 [6–8]. An extra 21 million instances of elevated blood glucose during pregnancy are anticipated to contribute to the worldwide Diabetes Mellitus burden. That equates to 17% of live births in 2013 to women who had some sort of elevated blood glucose in pregnancy [9–11].

2. PREVALENCE OF DIABETICS IN POPULATION

The International Diabetes Federation calculated the regional burden of type 2 diabetes mellitus prevalence across South East Asian nations, including India. The prevalence of type 2 Diabetes Mellitus among adults aged 20 to 79 years in South East Asian nations was predicted to be 883 million in 2013 [12–14]. TIIDM is mostly caused by changes in life-style and physiological factors. They are lesser physical activity, sitting in a place for prolonged-time, smoking habits, and alcoholism etc.,[15–17]

3. ROLE OF PHYSIOLOGICAL FACTORS AND GENETIC CONTRIBUTION

TCF7L2 (transcription factor 7-like 2) influences proglucagon gene expression10n and, as a result, glucagon-like peptide-125 production. Obesity (another independent risk factor for diabetes) is also inherited substantially [18,19]. MODY, or maturity-onset diabetes of the young, accounting for up to 5% of all cases. 27 Several medical disorders can cause or aggravate type 2 diabetes (Philipson, n.d.).
These include obesity, hypertension, high cholesterol, and metabolic syndrome (also known as Syndrome X, Reaven's syndrome). Sedentism, high-fat diets, and being above the age of 30 have all been linked to an increased prevalence of type 2 diabetes mellitus. This dysfunction means that meals do not regulate the rises in glucagon and hepatic glucose that occur during fasting [22–24]. Hyperglycemia begins with low insulin levels and progresses to insulin resistance. The gastrointestinal tract has a crucial role in the release of insulin and, in the case of glucagon, in the suppression of glucagon (peptide 1). Incretins [25]. Although the insulinotropic effects of glucagon-like peptide-1 are preserved in type 2 diabetes patients, gastric inhibitory peptide activity is reduced. Dipeptidyl peptidase IV swiftly inactivates glucagon-like peptide-1, as it does gastric inhibitory peptide [26,27].
Researchers are trying to figure out how mitochondrial malfunction contributes to insulin resistance and the onset of type 2 diabetes [28–30]. The idea also states that adipose tissue is necessary as an endocrine organ; this is because insulin resistance and perhaps beta-cell dysfunction have been associated with the release of many adipocytokines, such as leptin, TNF-alpha, resistin, and adiponectin [31,32]. The bulk of people with type 2 diabetes mellitus are overweight, and many also have central visceral adiposity. So, fat cells contribute to the development of type 2 diabetes. Although the portal/visceral concept has been used [33–35].

4. PLASMA LIPOPROTEINS AND DIABETICS

Plasma lipoproteins (pseudomicellar particles) are water-soluble macromolecules composed of lipid complexes (triglycerides, cholesterol, and phospholipids) and one or more apolipoproteins [36]. Lipoproteins are composed of a hydrophobic lipid core (triglycerides and cholesteryl esters) surrounded by hydrophilic lipids (phospholipids and unsaturated cholesterol) and proteins that interact with physiological fluids [37,38]. Based on their relative densities, size, mobility during electrophoresis, and protein content, plasma lipoproteins are classified into five fundamental types: Chylomicrons, very low-density lipoproteins (VLDL), intermediate density "1-poproteins (IDL), low-density lipoproteins (LDL), and high-density lipoproteins (HDL) are all lipoproteins (HDL). The quantity of lipid and protein per particle determines the density of a lipoprotein. Lipoproteins are necessary for dietary cholesterol absorption, long-chain fatty acid absorption, and fat-soluble vitamin absorption, as well as triglyceride, cholesterol, and fat-soluble vitamin transport from the liver to peripheral tissues and cholesterol transport from peripheral tissues to the liver [39,40].
Exogenous lipoprotein metabolism promotes dietary lipid transport. In the intestinal lumen, pancreatic lipases hydrolyze dietary triglycerides, which are then emulsified with bile acids to create micelles [17,41]. In the enterocyte, dietary cholesterol and retinal are esterified (by the addition of a fatty acid) to create cholesteryl esters and retinyl esters, respectively. Nacent chylomicrons are produced in the intestinal lymph and quickly transferred to the systemic circulation, where they are thoroughly processed by peripheral organs before reaching the liver [42,43]. Lipoprotein lipase (LPL) is a protein that interacts with proteoglycans on the capillary endothelial surfaces of adipose tissue, the heart, and skeletal muscle[44]. Lipoprotein lipase hydrolyzes chylomicron triglycerides, releasing free fatty acids; in this process, ApoC-2 functions as a cofactor for Lipoprotein lipase, which is supplied to circulating chylomicrons [45]. Triglycerides are formed when nearby myocytes or adipocytes take up the free fatty acids, oxidize or reesterify them, and then store them. Albumin binds certain free fatty acids, which then go to other organs, most notably the liver. The chylomicron particle shrinks when its hydrophobic core is digested and its surface hydrophilic lipids (cholesterol and phospholipids) are transferred to HDL [46,47]. Cholesterol ester-rich chylomicron remnants are the tiny particles that result. ApoE quickly filters out any leftover particles in the blood [48,49].

5. CONCLUSION

All lipoprotein categories differ in size and composition. Because lipids are largely concentrated in the core, the lipid-to-denser-protein ratio shifts, affecting both buoyancy and particle size. VLDL and HDL particles differ in their apoprotein composition, notably the amounts of ApoCs and ApoE on the particle. Lipoproteins are all made up of a triglyceride core and a hydrophobic cholesteryl ester. These lipids' contents are governed by CETP-mediated lipid exchange and lipase activities, which convert triglycerides to monoglycerides, glycerol, and free fatty acids. When these enzyme activities are disrupted in type 2 Diabetes Mellitus, triglyceride-rich lipoproteins are transformed to smaller, denser forms. This applies to both HDL and LDL.

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COMPETING INTEREST

The authors declare no conflict of interest.

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