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THE RELATIONSHIP BETWEEN FATTY ACIDS AND TYPE II DIABETES MELLITUS

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ABSTRACT

We can summarize the main risk factors for type 2 diabetes mellitus (T2DM) by looking at our nutrition, age, and lifestyle. β-cell dysfunction and insulin resistance (IR) are outcomes of the pathophysiology of type 2 diabetes. As an indirect result of IR on important metabolic enzymes, lipid and lipoprotein abnormalities are also a factor in T2DM patients. Recent research has indicated that lipid fluctuation may be the cause of poor glucose metabolism as well as one of its effects. Fatty acids (FAs) affect cell membrane fluidity and permeability, insulin receptor binding and signaling, and the translocation of glucose transporters. Therefore, it is suggested that FAs might play a crucial part in the emergence of IR and T2DM. The current review's objective was to

INTRODUCTION

The obesity epidemic is intimately associated with the growing worldwide health issue of type 2 diabetes mellitus (T2DM) [(1)]. Globally, 415 million people worldwide have diabetes, and an estimated 193 million more are suspected of having the condition but are not yet recognized as patients. Over 90% of individuals with diabetes have type 2 diabetes, which can result in microvascular and macrovascular problems that highly tax health care systems and cause patients and caregivers significant psychological and physical anguish.

Despite increased knowledge of risk factors and evidence for successful preventive strategies, type 2 diabetes incidence and prevalence are rising globally. Early detection made possible by screening programs and the accessibility of safe and efficient medications reduce morbidity and death by averting or delaying issues. A deeper comprehension of certain diabetes phenotypes and genotypes may result in more individualized and customized care for people with type 2 diabetes, as has been shown in patients with maturity onset diabetes of the young [(2)].

Saturated, monounsaturated, polyunsaturated, and Trans fats are the four broad types into which fatty acids can be separated. There is a link between Trans fats and saturated fatty acids and a higher risk of coronary heart disease. Consuming a lot of fat may make obesity more likely. One of the main risk factors for insulin resistance and non-insulin-dependent diabetes mellitus is obesity, particularly abdominal obesity. It is proposed that a high percentage of fat in the diet, regardless of obesity and body fat distribution, is linked to decreased insulin sensitivity and a higher chance of acquiring diabetes, with the type of fatty acids in the diet potentially having an impact on this risk. Significant correlations have

been found in cross-sectional investigations between insulin sensitivity and the composition of serum lipid fatty acids.

People who have type 2 diabetes have an increased risk of several short- and long-term complications, many of which end in premature death. Because type 2 diabetes is so common, it can have a hidden beginning and delayed identification, which increases the risk of morbidity and death. This is especially true in developing countries with inadequate resources, such those in Africa [4,5].

Literature Review

Epidemiology:

Due to lifestyle and environmental risk factors, the incidence of type 2 diabetes varies significantly between geographical regions.

In 2011, an estimated 366 million individuals suffered with diabetes. Type 2 diabetes is becoming more prevalent in every nation.DM, with 80% of the world's population living in low- and middle-income countries. 4.6 million lives were lost to DM in 2011. By 2030, 439 million individuals are expected to develop type 2 diabetes (DM).) [6, 7].

Lifestyle:

Type 2 diabetes is mostly caused by lifestyle and genetic factors. [10]. numerous lifestyle factors are known to have an impact on type 2 diabetes. These include smoking cigarettes, drinking a lot of alcohol, not exercising, and living a sedentary lifestyle [9,11]. Obesity has been found to have an impact on more than 55% of type 2 diabetes cases [12]. The surge in juvenile obesity between the 1960s and 2000s is assumed to be the cause of type 2 diabetes in children and adolescents. The current increase in type 2 diabetes cases may be related to environmental toxins [13].

Genetics:

Having relatives with type 2 diabetes, especially first-degree relatives, is strongly associated with inheritable genetic risk for type 2 DM.DM significantly raises the likelihood of type 2 DM development. Nearly 100% of monozygotic twins have concordance, and 25% of patients have a family history of DM (15). TCF7L2 is one of the genes that have recently been found to be strongly linked to the development of type 2.DM. (similar transcription factor 7). controls the expression of the proglucagon gene, which in turn controls glucagon-like peptide-1 synthesis [16].

Furthermore, obesity is highly hereditary and is an independent risk factor for type 2 diabetes [14, 17]. Types that are monogenic, such as young people's maturity-onset diabetes (MODY). makes up 5% or more of cases [18].

Medical conditions

Type 2 diabetes can be exacerbated by or caused by a variety of medical conditions. These include the metabolic syndrome, sometimes known as Syndrome X or Reaven's syndrome, which is commonly linked to these disorders, obesity, hypertension, high cholesterol (combined hyperlipidemia), and hypertension [19].Aceromegaly is another explanation. Medications, thyrotoxicosis, pheochromocytoma, cancer, chronic pancreatitis, and Cushing's syndrome [20]. An elevated risk of type 2 diabetes has also been associated with advanced age, high-fat diets, and a sedentary lifestyle [21].

Pathophysiology

Free fatty acids (FFA) in plasma have physiologically significant functions in the heart, pancreas, liver, and skeletal muscle. Nevertheless, persistently high plasma. There seem to be pathophysiological implications for FFA. Height ended. FFA concentrations have been associated with the formation of both hepatic and peripheral insulin resistance. Although the exact mechanism in the liver is still unknown, a model has recently been proposed to explain how elevated FFA contributes to the development of insulin resistance in skeletal muscle [22].

Randle postulated more than thirty years ago that FFA and glucose compete with one another for the role of primary energy substrate in heart muscle, and that when FFA levels are raised, glucose oxidation is reduced. According to recent evidence, elevated plasma FFA significantly contributes to insulin resistance. A decrease in insulin-stimulated muscle glucose transport may be mediated by elevated FFA and intracellular lipids, which appear to impede insulin signalling. translocation of GLUT-4.

There is less muscle glycogen synthesis and glycolysis as a result of the reduction of muscle glucose transport. Elevated FFA in the liver can counteract the effects of insulin on endogenous glucose synthesis, which can lead to hyperglycaemia. FFA also have an impact on insulin secretion, while it's still unclear how they relate to one another. Lastly, data is presented indicating that FFA play a critical role in the relationship between β-cell malfunction and insulin resistance; hence, lowering increased plasma FFA levels should be a major treatment goal in type 2 diabetes and obesity [23].

Screening and Diagnosis:

Numerous readily available assays are available for the diagnosis and screening of DM. The suggested test for screening is the same as the test for diagnosis, meaning that a positive screen corresponds to a diagnosis of diabetes mellitus (DM) or pre-diabetes [27]. Approximately 25% of individuals diagnosed with type 2 diabetes had microvascular problems at the time of diagnosis, indicating that they had been afflicted with the illness for over five years [24].

As for a single elevated glucose reading with, it continues to be based on the World Health Organization's (WHO) 2006 National Diabetic Group Criteria or the American Diabetic Association's (ADA) 1997 recommendations. The patient experienced symptoms such as polyuria, polydipsia, polyphagia, and weight loss. Furthermore, two hours following the oral dose, two instances of elevated (FPG) readings of 7.0 mmol/L (126 mg/dL) or 11.1 mmol/L (200 mg/dL) in the (OGTT) were noted. [25].

The 1997 ADA criteria for diagnosing diabetes mellitus give greater weight on the FPG than does the WHO, which favours the OGTT. (Fructosamine and HbA1c) are still helpful in assessing blood sugar management over time. Nevertheless, practical physicians frequently go above and beyond the recommended safety measures. In July 2009, the International Expert Committee (IEC) recommended that patients with diabetes mellitus (DM) fulfill an additional diagnostic need, namely, a HbA1c result of 6.5%. This committee acknowledged that those at high risk of developing diabetes mellitus can be identified using a HbA1c range of 6.0% and <6.5%; however, they also suggested that the term "pre-diabetes" could be phased out [26]. Comparable to the glucose-based assays. There is uncertainty over the HbA1c level that separates DM from normality [27]. The IEC made the decision to recommend a cut-off point that gives priority to specificity in the diagnosis of diabetes mellitus. They observed that approach achieved a compromise between the minor clinical ramifications of postponing the diagnosis in a patient whose HbA1c level is less than 6.5% and the embarrassment and cost associated with making an inaccurate diabetes diagnosis. [29]

Diabetes and dietary fat: cross-sectional studies:

Dietary fat intake of diabetic patients and healthy participants has been studied in numerous cross-sectional or case-control studies [28]. For the Mediterranean Group, in the international, multicenter trial. Dietary surveys and studies of diabetes were carried out in 0.6 nations. The findings demonstrated that, in comparison to healthy controls, Recent diabetes diagnoses were associated with higher relative intakes of total fat from sources derived from animals.

Additionally, compared to controls, those with undiagnosed type 2 diabetes consumed substantially more saturated fat. The latter is significant because, as a result of their type 2 diabetes diagnosis or dietary interventions, these participants were unable to alter their diet [30]. The perplexing issue is still present.

However, these results are consistent with a Dutch trial that included participants who consumed more total fat and were recently diagnosed with diabetes. Saturated fat in particular (40 percent energy, E). (15 percent E) [31]. It should be emphasized that sectional observational research evaluating the association between fat in the diet quality and diabetes or diabetic are subject to a variety of causes of bias, thus cautious interpretation is required. For instance, altering one's diet because of obesity or prediabetes, or neglecting to make precise modifications for physical activity or adiposity [29].

Insulin resistance, FA biomarkers, and the risk of diabetes.

Insulin sensitivity and FA biomarkers:

Studies that are cross-sectional There is a clear correlation between the amount of long-chain and insulin sensitivity in skeletal muscle, according to cross-sectional research that evaluate FA composition using the clamp technique. Insulin activity and PUFA [32]. Conversely, an individual becomes more insulin resistant the more saturated their muscular membrane is. Similar results were found in a cross-sectional research conducted among Pima Indians. [33]. i.e., a negative correlation between insulin resistance and the quantity of n-3 FAs.

FA biomarkers and diabetes:

prospective research According to a number of prospective studies, type 2 diabetes is predicted by an FA pattern that exhibits a high intake of saturated fat and a low relative intake of linoleic acid. A prospective investigation of the ULSAM cohort, which included males aged 50 who were followed for ten years, revealed a relationship between blood cholesterol esters' FA content at baseline and and the incidence of diabetes during follow-up [34].

Trans fatty acids (TFA) and diabetes:

Although trans fatty acids (TFA) are known to have a negative impact on cardiovascular disease, little research has been done on how TFA contributes to the development of type 2 diabetes. Because the Nurses' Health Study assessed diet multiple times, it is the largest and most comprehensive epidemiologic study. [35]. It demonstrated a dose-response relationship and a favorable correlation between TFA intake and diabetes risk. There was no evidence of a positive correlation in small epidemiologic studies or studies without repeated food measures (36). TFA raises inflammatory cytokines that are linked to diabetes risk, despite the fact that it has not been demonstrated that inflammation is a causal component in the development of diabetes. [37].

Dairy fat, insulin resistance, and diabetes:

Dairy fat consumption was found to be inversely correlated with a number of metabolic abnormalities, including higher fasting plasma glucose concentrations, in a cross-sectional investigation of elderly Swedish males [38]. Additionally, it showed a substantial correlation with blood lipids (.15:0 and.17:0), which are regarded as indicators of dairy fat intake.

On the other hand, in a case-cohort investigation, phospholipid concentrations in plasma of.15:0 were negatively linked to the occurrence of type 2. Four years of monitoring [39]. Pereira et al. discovered an adverse correlation between the prevalence of metabolic traits linked to insulin resistance and dairy consumption in young people who had been overweight at baseline (but not in leaner individuals). (40). In the Health Professionals Follow-up Study, a dietary pattern that included higher intake of dairy goods, especially low-fat varieties, was associated with a decreased prevalence of type 2 diabetes in males. A particular dietary pattern heavy in low-fat milk and cheese has also been connected to a lower risk of type 2 diabetes in older or middle-aged women. diabetes [41]. The specific dairy product ingredients that contribute to the advantage that has been seen are unclear. There is a greater correlation between low-fat or fat-free dairy products and the likelihood that dairy fat does not independently protect against diabetes.

T2DM and prediabetes in youth:

Previously thought to be a metabolic condition that mainly affected adults, type 2 diabetes (T2DM) is now more common in young people, adolescents, and occasionally even children. [42]. For instance, it was estimated that in 2001, there were 42 cases of T2DM for every 100,000 kids in North America, according to the crude prevalence of the disease [43]. Less than 4% of instances were documented twenty years ago, nonetheless, among certain racialized groups, like Asian, Pacific Islander and American Indian

, up to 80% of new cases among adolescents occur, A growing proportion of occurrence cases of paediatric diabetes mellitus are caused by type 2 diabetes (T2DM). [44, 45]

The incidence and prevalence of type 2 diabetes in young people differ significantly according on ethnicity, with greater rates seen in high-risk communities such Native Americans, Australian aboriginal populations, Hispanic, African American, Pacific Islander, and Asian populations [46, 49]. For instance, compared to the general population, Australian Indigenous people had a six-fold greater incidence of type 2 diabetes in their youth (50). A Teenagers in minority populations among the ages of fifteen and nineteen showed a higher incidence rate. (from 17.0 to 49.4 per 100,000-person years) in the USA's SEARCH for Diabetes in Youth study, as opposed to 5.6 per 100,000-person years in non-Hispanic whites [47].

Additional analysis from the same research shows that different ethnic groups may have different contributions from genetic and/or environmental variables to early-onset T2DM [48].

Not only is type 2 diabetes becoming more common in young people, but teens are also increasingly showing signs of prediabetes. Based on the most recent data from the US National Health and Nutrition Examination Survey (NHANES), the prevalence of IFG among US adolescents aged 12-19 years increased by 87.1% between 1999–2000 (.7%) and 2005–2006 (13.1%). [51]. Furthermore, in 2005–2006, it was projected that 16.1% of teenagers in the US had IFG and/or IGT. In pediatric populations with additional risk factors including hyperinsulinemia, obesity, and relatives with a history of diabetes mellitus, prediabetes is far more common than diabetes mellitus [52, 53].

The persistent rise in childhood obesity rates in Asian nations like China and India indicates that more people will likely get type 2 diabetes earlier in life, if not already. Efficacious intervention tactics mitigate the obesity pandemic. [54]. The decline in T2DM start age and the poor metabolic management

in young patients will have a significant impact on the disease's long-term burden. Youth with type 2 diabetes are a demographic that is more likely to have early issues and permanent chronic repercussions because of the long-term nature of their diseases. [55,56].

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