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REVIEW ARTICLE

Understanding the Role of Physical Factors on the Epigenetic Control of Type 2 Diabetes in Tamil Nadu, India

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ABSTRACT: Type 2 Diabetes is an exponentially growing condition affecting the lives of entire populations. The significant prevalence of cases raises issues in the healthcare industry regarding funding and the efficacy of treatments. The magnitude of the problem calls for new insights into epigenetics and whether strategic changes in physical activity can alter the epigenome to become favorable in the prevention and treatment of cases. Epigenetic changes produced by acute exercise or resistance training groups link directly to outcomes connected with the incidence of Type 2 diabetes. Individuals who engage in rigorous and high-intensity exercise lower their chance of acquiring Type 2 diabetes and reduce the condition's symptoms and effects.

KEYWORDS: Biomedical and Health Sciences; Genetics and Molecular Biology of Disease; Epigenetics; Type 2 Diabetes; Physique and Obesity.

Introduction

Diabetes and its significant prevalence have always been a common topic of discussion while growing up in Tamil Nadu, India. From 2014 to 2017, the prevalence of Diabetes Mellitus rose from 6% to 8.3%.[1] This was quite interesting as a student studying abroad as Type 2 Diabetes was prevalent in countries outside India, however, there was a noticeable difference in awareness within South-Indian communities. This paper was written to understand this observation and any links to epigenetics, a relatively new scientific study governing genomes' adaptations and lifestyle changes. This study could aid in developing new concepts on lifestyle changes that can be introduced into the South-Indian communities examined in this study to reduce their risk of Type 2 Diabetes. The study focuses on Type 2 diabetes as it links to dietary and lifestyle causes.

Discussion

Prerequisite Knowledge:

Type 2 Diabetes is a chronic condition where an individual develops insulin resistance. Insulin is an essential hormone produced by the body to signal the storage of glucose. Glucose is the monomer of carbohydrates that are consumed as food and transported around the circulatory system. When blood glucose levels are high, the pancreas produces insulin to store the excess glucose; however, an individual experiencing Type 2 diabetes would be inefficient at this process. As a result, their cells become desensitized to the hormone, and an excess of glucose remains in the circulatory system (hyperglycemia). Thus, the issue is not the lack of insulin; it is the insulin's inefficacy to signal cells to increase glucose uptake from the blood.[2]

Traditionally, Type 1 diabetes links to genetic predisposition and occurrences within family history. Although having no specific causes, identifying such variants of the HLA-DQB, HLA-DRB1, and HLA-DQA1 genes were observed to increase the likelihood of developing the condition known as diabetes. These genes code for the pancreatic beta cells to develop inappropriate immunological responses. The result of the responses leads to the destruction and impairment of pancreatic beta cells. Thus, there is an insulin secretion impairment and can be considered an autoimmune disorder as well.[3]

The genome determines our bodies' physiological and bio-chemical structure; all the genetic information is present in chromosomes. However, looking closer into the structure of a single strand of Deoxyribonucleic Acid (DNA), it can be observed that the double-helical structure consists of repeating units of nucleotides. Nucleotides are composed of a phosphate group, deoxyribose (pentose sugar), and a nitrogenous base. The complementary base pairing between pyrimidines (Thymine and Cytosine) and purines (Adenine and Guanine) leads to coding specific genes. These genetic combinations can remain for life however can be permanently altered in the case of genetic damage caused by carcinogens and ionizing radiation. A relatively young research field on the epigenome has showcased the importance of environmental and lifestyle factors in altering the ability of the body to read the genetic code. Characterized by the ability of specific genes to be expressed, epigenetic factors affect the role of genes in the pathologies of conditions as well.[4]

Specific processes occur on the molecular level to activate or repress specific genes. Several modifications such as methylation and acetylation occur to cause these changes. For example, the addition of a methyl group to a nucleosome represses the genetic expression by interfering with the ability of transcription to bind with a strand of DNA.[5] External stimuli in the environment induce changes in transcription factor levels using chemical responses. As a result, the degree of gene expression varies. A notable example would be Hydrangeas (a flowering plant) that blooms with blue flowers in acidic soils and pink flowers in alkaline soils. In humans, ex-

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posure to solar radiation influences the production of melanin in the epidermal layer.[6]

Issue in Study:

Tamil Nadu is a large state in India with a population of over 72.15 million people.[7] In a 2010 study, it was found that roughly 6% of the studied group were diabetic, and 17.2% of the subset had a Body Mass Index (BMI) of greater than 30 (obese).[8] This observation was commonly noted within the South-Indian population and hence, the researchers of the study proposed Indian Diabetic Risk Score (IDRS) parameter to better understand the subset population's vulnerability to Type 2 Diabetes. The IDRS is a score out of 100 which takes into account age, abdominal obesity, family history of diabetes, and physical activity. Active diabetic history and increasing age and abdominal obesity are linked to a higher IDRS whilst increasing physical activity lowered the IDRS. The study followed up 1936 individuals from February to April, 2018.[8] Although BMI was adopted as a universal measure of physique, ethnic group-based BMI charts were introduced and adopted. BMI is a calculated score that is used to determine body fat levels using the following equation:

$$Body\ Mass\ Index = \frac{Weight\ in\ Kilograms}{Height\ Squared\ \text{in}\ Meters}$$

Example Calculation:

The average Indian man weighs 65 kg and has a height of 177 cm.[9]

$$Body\ Mass\ Index = \frac{65}{1.77^2} = 20.7$$

According to the World Health Organization's Asian BMI categories, the average man would be classified as Ideal Weight (18.5 – 23).[10]

It is commonly known that high obesity rates increase the risk of conditions such as Type 2 diabetes and atherosclerosis. However, less than a fifth of the diabetic set was classified as obese. Although BMI has had a reputation for neglecting the role of lean body mass on the score, the data suggests that the more significant percentage (82.8%) were significantly leaner.[7] This contributes to the notion that BMI alone is not a driving factor in solving the Type 2 diabetes epidemic in Tamil Nadu.[11]

Role of Occupational Activity Levels:

An understanding of the socio-demographic distribution of Tamil Nadu sourced from an external study can be attained using Table 1. This study covered 35,000 individuals from rural health centers in Chunampett and Annechikuppam from February to March, 2008.[7]

Table 1: Occupational Classification of Studied Population

Occupation	Number	Percentage of Total Study (1196)
Homemaker	931	48.14
Labor work	292	15.08
Business	118	6.10
Agriculture	98	5.06
Retired	51	2.63
Student	44	2.27
Service	35	1.81
Other	366	19

Working with the assumption that obesity levels are not the primary cause of Type 2 Diabetes, it is possible to delve into the activity levels of these occupations using Table 2.[12]

Table 2: Occupational Activity Level (OA)

Occupation	Activity Level
Homemaker	Intermediate (110 OA)
Labor work	High (36 OA)
Business	Intermediate (156 OA)
Agriculture	High (31 OA)
Retired	-
Student	Low (183 OA)
Service	High (44 OA)
Other	-

Note: Lower score indicates a more active occupational activity level based on accelerometer data.

Excluding the two null groups (Retired and Other), 33 individuals fall under the 'Low OA' category, 1049 individuals fall under the 'Intermediate OA' category, and 425 individuals fall under the 'High OA' category.

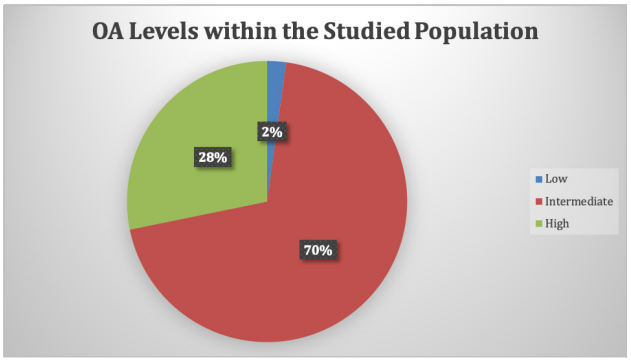

Figure 1: Pie Chart displaying OA Levels within the Studied Population.[12]

Using Figure 1, the vast percentage (70%) of the demographic lies within the Intermediate OA level. The data from Table 2 were gathered using accelerometry, a technique used to measure the mechanical movement of the subject. On paper, this should be pretty accurate for understanding the activity levels. However, there needs to be a clear division between metabolic activity and muscular hypertrophy. Although activities such as walking accumulate to higher metabolic activity levels, they lack sufficient myofibrillar strain, which fails to achieve hypertrophy. Hypertrophy is crucial in developing more leaner mass which acts as a 'glucose sink'. Myofibrils are a basic unit of striated muscle cells responsible for the tensile strength of the cell. Under mechanical tension from activities such as resistance training, these fibers tend to get damaged. Responsive measures from the body repair and build more fibers to resist the strain better. Muscle cells are also multi-nucleated as they are required to generate power for mechanical motion, e.g., flexing an arm. This process requires metabolic respiration to convert glucose into Adenosine Tri-Phosphate (ATP), of which the glucose is absorbed through the bloodstream. This explains the significance of higher lean body mass to reduce hyperglycemic conditions and hence lower the risk of Type 2 Diabetes. An individual's diabetic diagnosis is based on the blood glucose parameters two hours after a meal of 140-199 mg/dL (Pre-Diabetic) and >200 mg/dL (Diabetic).[13] The presence of high lean body mass will not cause the blood glucose level to

plummet due to homeostatic responses where glycogen stored in muscle and liver cells is released into the bloodstream as glucose. This could pose a potential stone wall to individuals at risk; however other epigenetic responses could have been evoked as an effect of resistance training.

Theoretically, the physiological and metabolic strain could cause the skeletal muscles to undergo subjective changes in DNA methylation profiles. Thus, specific genes could govern the metabolism of lipids and glucose associated with Type 2 Diabetes, and the understanding would be that epigenetic control factors would regulate these genes' expression.

Role of Exercise Type and Intensity:

Participants	Type of exercise intervention and comparison group	Duration and intensity intervention	Tissue	Outcome measures		Level of evidence[a]
				epigenetic modification	outcome	
Healthy subjects						
Untrained patients n = 30 (20 F/10 M) Age: 46.1±8.56 y	Rehabilitation training No control group	5 days per wk for 2–3 h during 4 wk	Whole blood	Hypermethylation in one CpG site of *AMPKA2*	Improvement of metabolic responses to an energy imbalance	II-1
Subject with or without FH of T2D n = 28 M FH$^+$ 37.5±4 y FH$^-$ 37.5±5.2 y	Endurance training No control group	1 session of 1-h spinning class and 2 sessions of 1-h aerobic class per wk for 6 months	Skeletal muscle biopsy	Hypomethylation of genes involved in T2D development, respiratory chain, and calcium signaling pathways	Reduction of future risk of T2D	II-1
Healthy trained subjects n = 10 M Age: 30.5±5.5 y	Acute exercise test No control group	60 min of cycle ergometer exercise at 65% of peak watt before and after 12 wk of endurance training	Skeletal muscle biopsy	Upregulation of miR-1 and miR-133a	Improvement of insulin sensitivity by 19%	II-1
Healthy subjects n = 9 M Age: 23±5 y	Endurance training No control group	10 days: 45 min at 75% of VO_2max for 4 days 60 min at 75% of VO_2max one day 90 min at 75% of VO_2max one day 6 × 5 min intervals at 90–100% for 4 days	Skeletal muscle biopsy	Acute endurance exercise: Increase in miR-1, miR-133a, and miR-133b 3h after exercise and reduced miR-9, -23a, -23b and -31	Increase in transcription of genes involved in muscle regeneration and mitochondrial biogenesis	II-1
Healthy physically active subjects n = 12 M Age: 26.2±5.3 y	Acute exercise test No control group	45 min of one-legged knee extensor exercise at 60% of $watt_{max}$ before and after 7 days of bed rest	Skeletal muscle biopsy	Bed rest reduced miR-1 and miR-133a content; in addition, reduced HKII, SIRT1, HAD, and CS protein content	Bed rest induced whole body glucose intolerance and decreased muscle metabolic capacity as well as it abolished the exercise-induced adaptive gene response.	II-1
Healthy subjects n = 14 (9 M/5 F) Age 33±2 y	Acute exercise test No control group	48 min consisting of 4 sets, each composed of 8 min of cycling at 70%, 2 min at 90% of PHR, and 2 min rest	Skeletal muscle biopsy	Upregulation of miR-30, miR-128, and miR-378	Alteration of the skeletal muscle gene expression related to insulin sensitivity	II-1
Healthy subjects n = 10 M Age: 22.6±1.6 y	Acute exercise test No control group	Exercise test at 80% VO_2max for 15 min	Skeletal muscle biopsy	Increase in hydroxymethylation levels followed by hypomethylation of the *NR4A3* promoter	Improvement of metabolic function (normoglycemic levels) and endurance capacity	II-1
Two separate cohorts were used: 1. Healthy sedentary subjects n = 14 Age: 25±1 y 2. Healthy sedentary subjects n = 8 M	First cohorts group: Acute exercise test Second cohorts group: acute exercise test No control group	First cohorts group: Incremental exercise on cycle ergometer until the fatigue Second cohorts group: 2 isocaloric acute exercise trials at 40% and 80% VO_2max on at least 1 week of separate occasions	Skeletal muscle biopsy	First cohort: Intensity-dependent gene hypomethylation Second cohort: Hypomethylation of genes involved in T2D development	Beneficial effect on the prevention and treatment of type 2 diabetes	II-1
Healthy subjects n = 8 M Age: 21.1±2.2 y	Resistance exercise training No control group	Three sets of 8–12 repetitions during 8 wk at 80% of 1RM	Whole blood	Hypermethylation on 28,397 CpG site and hypomethylation on 28,987	Improvement of diabetes-related signaling pathways	II-1
Diseased subjects Obesity and T2D patients n = 17 (13 F/5 M) Age: 49±5 y	Resistance exercise training (n = 9) Endurance training (n = 8)	Resistance training group: 2–3 sets of 6–8 repetitions using machine weights during 16 wk Endurance training group: performed exercise on a cycle ergometer for 40–60 min during 16 wk	Skeletal muscle biopsy	Hypomethylation on inflammatory and glucose homeostasis genes	Better regulation of glycolytic flux	I

Figure 2: Exercise-induced epigenetic modifications related to insulin resistance and diabetes.[14]

Another study set out to understand the exercise-induced epigenetic modifications related to insulin resistance and diabetes and the results are presented in Figure 2. This study followed up on 50 individuals from each test group for 6 months.

Analyzing the data, the broad physical exercise spectrum ameliorates metabolic responses, insulin sensitivity, muscular regeneration, and Type 2 Diabetes risk. However, interesting results emerge based on the different types of training; rehabilitation, endurance, acute exercise, and resistance training. Outcomes directly related to the prevalence of Type 2 diabetes are tied to epigenetic alterations caused by acute exercise or resistance training groups. Furthermore, the study highlights the positive impact of resistance training on obese and Type 2 diabetic patients; improved glycolytic homeostasis induced by genes. Individuals following a strenuous and high-intensity exercise regimen reduce their risk of developing Type 2 diabetes and help them reduce the condition's effects. Studies highlight the high prevalence of sarcopenia in South-Indian populations, characterized by lower lean body mass and higher visceral fat percentages.[15]

Role of Abdominal Obesity:

Body composition synergistically works with physical training in playing a role for Type 2 Diabetes. Understanding that BMI has its limitations with unreliable measures of lean body mass, often waist circumference is the following optimal measurement procedure. Large waist circumference is often a result of excess subcutaneous fat, characterized by consuming a caloric surplus with minimal physical exertion. Visceral fat percentages often rise with this issue, and the scientific basis for the connection between abdominal obesity and diabetes is theoretically viable. Abdominal obesity enhances the risk of diabetes by a variety of released substances such as non-esterified fatty acids and adipocytokines such as tumor necrosis factor and decreased adiponectin.[16]

	CRP (mg/L)	P-value	IL-6 (pg/L)	P-value[b]
	Mean ± SE		Mean ± SE	
Body mass index[c]		0.269		0.666
Normal	3.73±1.22[e]		5.94±2.55[e]	
Overweight	4.54±0.66[e]		2.12±0.25[e]	
Obesity I	10.19±3.68[e]		3.96±1.02[e]	
Obesity II	10.09±1.85[e]		4.32±1.43[e]	
Obesity III	6.56±2.77[e]		6.35±2.55[e]	
Abdominal fat %[d]		0.023		0.002
Low	3.92 ± 0.86[e]		4.90± 2.26[ef]	
Average	5.53 ± 0.54[e]		2.68± 0.28[e]	
High	14.20 ± 1.24[f]		6.40± 2.08[f]	

Figure 3: Relation Between BMI and Abdominal Fat and Inflammatory Biomarkers in Patients with Type-2 Diabetes With Good Glycemic Control.[17]

	CRP (mg/L)		IL-6 (pg/L)	P-value[b]
			Mean ± SEM	
Body mass index[c]		0.02		0.047
Normal	5.42±2.08[e]		4.06±1.82[e]	
Overweight	6.79±1.18[e]		3.38±0.62[e]	
Obesity I	7.80±0.97[e]		3.89±0.34[e]	
Obesity II	11.17±1.16[f]		4.41±0.44[ef]	
Obesity III	16.66±3.78[g]		6.10±0.93[f]	
Abdominal fat %[d]		0.014		0.049
Low	8.82±4.25[e]		3.30±2.14[e]	
Average	8.72±0.71[e]		4.24±0.31[f]	
High	12.47±2.67[f]		4.42±0.60[f]	

Figure 4: Relation Between BMI And Abdominal Fat and Biomarkers of Inflammation in Patients with Type-2 Diabetes with Poor Glycemic Control.[17]

Figures 3 and 4 highlight how Type 2 Diabetes with Poor Glycemic Control has increased inflammation occurrences in pancreatic cells. This study was conducted using 198 individuals with Type 2 Diabetes Mellitus over the course of 3 months. Speculating could explain how epigenetic markers can be activated and repressed by DNA methylation and acetylation. The 'turning on and off' of epigenomic expression could play a role

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in inflaming associated cells where the inflammation disrupts pancreatic beta cells' sensitivity and response signals. Active control of nutritional caloric intake and physical exertion would create a metabolic deficit where the pancreatic alpha cells are primed to produce glucagon, chipping away at glycogen stores and depleting subcutaneous and visceral fat stores. Highlighting the data from Figure 3 shows how even in individuals with Good Glycemic Control, decreasing Abdominal Fat % from High to Low creates a decrease of 10.28 mg/L of C-Reactive Protein (CRP). CRP is a protein synthesized in the liver in response to inflammation and external cross-sectional studies have found a correlation ($P<0.01$) between CRP levels and central obesity. The study indicates that higher levels of adiposity in individuals lead to low-grade chronic inflammation which would hinder functional performance of smooth and skeletal muscles.[18] Poor muscular performance can hinder recovery and effectiveness of training programs which ultimately negate the target of reducing the effects of Type 2 Diabetes Mellitus. Although larger abdominal adiposity is generally explained by a sustained caloric surplus without adequate exercise, there may be a link between Type 2 Diabetes Mellitus and South Asia's consumption of the staple food; white rice. Referring to a study that followed 132, 373 participants for 9.5 years, the researchers set out to find a link between white rice consumption and incident diabetes by analyzing data from 21 countries. Results found a statistical association between higher white rice consumption (>450 g per day) and increased risk of Type 2 Diabetes (Hazard Risk of 1.20) within the whole study. However, whilst comparing different regions, it was observed that South Asia had the highest Hazard Risk of 1.61. This observation may be impacted by uncontrolled factors throughout the study's 9.5 years, however, comparing rice consumption may reveal new insights into the plausibility of rice consumption playing a significant role in South India's Diabetes prevalence. Data from the study show that China (HR of 0.78) had the second-highest rice consumption of 200 g/day whereas South India had the highest rice consumption of 630 g/day. This alarming disparity is supported by the higher risk of incident diabetes within the studied population and is an indicator to reduce white rice consumption to prevent the onset of diabetes.[19] Although external factors could have contributed to this statistical difference, loss of abdominal fat desaturates the circulatory stream from Low-Density Lipoproteins (LDLs) and lowers the occurrences of inflammatory responses.[20] If the buildup of LDLs continues to increase progressively, inflammation will give way to atherosclerotic progression as well. It is understood that inflammation caused by high abdominal fat percentage would be an early indicator of high-risk Type 2 Diabetes, especially amongst the largely sarcopenic South-Indian population.[21]

Comparison of Resistance Training Methods for Improved Glycemic Control:

A randomized controlled trial investigated different resistance training methods to improve glycemic control. The study followed 32 individuals through an 8-week training program. The individuals were randomly assigned to either an 8-week program of Hypertrophy Resistance Training (HRT) where exercises were performed with 2 sets at 10-12 repetitions or an 8-week program of Endurance Resistance Training (ERT) with 2 sets at 25-30 repetitions. The study's conclusions highlight HRT's specific maximal resistance training aids to significantly boost strength in target muscles compared to ERT. However, HRT showed no significant advantage to ERT in improving glycemic control where both groups exhibited comparable improvements.[22] As both resistance training methods provide comparable results, it would be beneficial to leave the option to the patient's discretion and preference.

Conclusion

This study has employed theorized connections between exercise and epigenetic control factors and cross-referenced studies to establish a ground understanding of their influence on Type 2 diabetes within Tamil Nadu, India. It has come to light that occupational activity levels, rehabilitation, and endurance training are insufficient in combatting the epidemic of Type 2 diabetes. Such altercations pose morbid threats to global health systems and require awareness and appropriate action. Evidence suggests the positive effect of regular resistance training and other high-intensity exercise plans on epigenetic alterations in physiological and metabolic systems. It has also been shown that there is no baseline difference between hypertrophy resistance training and endurance resistance training, leaving the choice up to the preference of the patient. Furthermore, the public's use of resistance training could also be woven into their daily lifestyles. Commuting using bicycles and voluntarily opting to use staircases are subtle strategies the average individual could employ to resistance train. The study also highlights a concern with South India's alarming consumption of white rice; a staple food. Higher white rice consumption is linked to a higher risk of developing Type 2 Diabetes Mellitus; hence, it is suggested to reduce rice consumption as a preemptive measure to reduce the risk of Type 2 Diabetes. As such, global awareness would need to be raised, emphasizing resistance training and updating the public's understanding of 'healthy' lifestyles.

An extension to this study can be conducted to quantifiably measure the impact of physical exercise on the epigenetic control of Type 2 diabetes in Tamil Nadu, India. Taking a group of consensual diabetic volunteers and a healthy control volunteer, a 6-month long study could provide different training method regimens at different intensities. In addition, at regular intervals, a glycated hemoglobin (A1C) test could be conducted to measure the average blood sugar level for the past three months.[23] The quantitative analysis could uncover new insights into practical strategies that already affected individuals could employ to alleviate their symptoms and potentially even reverse the effects of Type 2 Diabetes.

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