Student's Journal of Health Research Africa
Vol. 4 No. 12 (2023): December 2023 Issue
https://doi.org/10.51168/sjhrafrica.v4i12.764
Original article

HAS THE EVOLUTION OF HOMO SAPIENS DOMESTICATED METABOLIC DISEASES LIKE DIABETES? A NARRATIVE REVIEW.

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ABSTRACT.

In this narrative review article, we attempt to review the information available to support the possible link between the evolution of Homo sapiens and the increase in metabolic diseases like diabetes. The Ardipithecus group, which lived around 6 million years ago, is considered the earliest humans closely linked to primates. The taxonomic assemblage encompasses Orrorin tugenensis, Sahelanthropus tchadensis, Ardipithecus ramidus, and Ardipithecus kadabba. Australopithecus anamensis, the initial reported member of the Australopithecines, inhabited the Earth approximately 4.2 to 3.8 million years in the past, demonstrating a combination of characteristics similar to both apes and human beings. They were bipedal and had an ankle joint that allowed them to walk upright. Australopithecus afarensis, another member of the group, had apelike traits such as long arms with curved fingers but was also bipedal and had a small brain case. Their capability to walk standing and climb trees assisted them in surviving for nearly 900,000 years.

Various studies have shown that food, BMI, habits, early puberty, use of pesticides, and environmental changes can all impact the incidence of diabetes. Domestication has allowed us (Homo Sapiens) to survive more efficiently, civilize more effectively and socialize more adequately. This natural course of evolution has taken and will continue to take millions of years. Future research and policy development may benefit from interdisciplinary approaches and a deeper exploration of the evolutionary aspects of human health.

Keywords: *Evolution; Metabolic diseases, Human evolution, Diabetes*
Submitted: 2023-10-21 Accepted: 2023-10-27

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BACKGROUND.

It is widely acknowledged in the scientific community that Homo sapiens and apes, possess a shared lineage originating from a common ancestor that existed approximately 6 to 8 million years in the past. Despite the compelling presence of fossil and DNA proof substantiating our intimate phylogenetic connection, the precise characterization of this elusive progenitor continues to elude scientific comprehension. The aforementioned progenitor defined the divergence that ultimately ended in the genesis of diverse human taxa. Homo habilis, the most ancient Homo species, is estimated to have emerged around 1.4-2.3 million years ago, exerting a significant impact on the evolutionary trajectory of Homo sapiens in terms of genetic inheritance, anatomical characteristics, and behavioral patterns. [1].

In this review article, we have attempted to review the available information to support the possible link between the evolution of Homo sapiens and the increase in metabolic disorders such as diabetes.

The History of Human Evolution.

Approximately 6 million years ago, the Ardipithecus group was extant. Ardipithecus is classified within the taxonomic category of genus Hominidae, encompassing the entirety of Homo sapiens while excluding the great apes. The aforementioned group encompasses Orrorin tugenensis, Sahelanthropus tchadensis, Ardipithecus

ramidus, and Ardipithecus kadabba. The ancient Homo sapiens emerged from the African continent, as supported by scientific evidence, indicating that a majority of these individuals exhibited walking on two legs, with a subset demonstrating arboreal capabilities. Despite the lack of available data about these initial human ancestors, it was the Ardipithecus grp. that precipitated the emergence of the Australopithecines approximately 4 million years in the past [1,2].

Australopithecus anamensis, the first reported member of the Australopithecines, inhabited the Earth approximately 4.2 to 3.8 million years in the past, embodying the evolutionary changes that shaped the human lineage. This adaptation toward bipedalism marked a pivotal moment in the gradual divergence of humans from their ape ancestors [1,2]. While exhibiting a blend of ape and human traits, these early species paved the way for the anatomical transformations that would follow.

Echoing the theme of evolutionary change, a fascinating shift has been observed in a different context: the occurrence of the median artery in the context of human anatomy. The median artery, an embryonic construction that normally degenerates around the 8th week of pregnancy, has demonstrated a remarkable trend of persistence and even resurgence over time. However, previous literature has indicated a likely secular trend in the occurrence of this anatomical variant. A study conducted before 1960 reported the existence of the median artery in adult cadavers to be between 4.4 percent to 8.3 percent. In contrast, Henneberg M, et al. found a significantly higher frequency of 27.2%. Furthermore, a recent retrospective analysis of a cohort of individuals in Australia, ranging from 51-101 years of age, who passed away between the years 2015 and 2016, has unveiled a noteworthy incidence rate of 33.3%. These findings suggest that the existence of the median artery has been experiencing a notable increase over time. The present study delivers supplementary evidence to substantiate this observation, suggesting that the incidence of the median artery has exhibited an upward trend, increasing from around 10 percent in individuals born during the middle of the 1880s to around thirty percent by the conclusion of the twentieth century. The reasons behind this microevolutionary change in the occurrence of the median artery remain unclear.

Introduction.

Fire about 700,000 years ago allowed early humans to occupy colder regions and was the first sign of modernization. The emergence of anatomically modern humans occurred approximately 150 thousand to 200 thousand years ago within the African continent, followed by subsequent migration to various geographical regions approximately fifty thousand to seventy thousand years ago. The evidence of recent human conduct becomes apparent within the archaeological documentation comprising a temporal range of three million to fifty million years in the past. During this temporal period, Homo sapiens in their nascent stage predominantly engaged in the activities of hunting and gathering, while theories suggest that certain genetic material inherited from Neanderthals might have conferred adaptive advantages in the face of nutritional deprivation. The moral and behavioral patterns exhibited by contemporary Homo sapiens are subject to the influence of various factors, encompassing the realms of psychology, religion, and sociology. The capacity of today's Homo sapiens to partake in religious practices and partake in intricate cognitive processes and cultural activities can be attributed to the evolutionary trajectory of our species. The examination of evolution yields valuable insights into the extensive narrative of the evolutionary journey that has shaped contemporary Homo sapiens, elucidating the development and manifestation of their present-day individual characteristics [1,2].

Although it is indeed verifiable that certain individuals may derive advantages from the incorporation of Neanderthal DNA, it is crucial to acknowledge that this outcome is not universally applicable. Indeed, in the context of today's Homo sapiens inhabiting Western civilizations, there exists a heightened probability that the presence of Neanderthal genetic material makes them susceptible to various pathological conditions, including but not limited to T2DM, urinary tract disorders, and Crohn's ailment [3]. T2DM is a heterogeneous disorder that poses a universal health threat, with varying pathophysiology and phenotype by ethnicity. Asian Indians, for example, have a phenotype that increases their susceptibility to T2DM compared to white Caucasians. Recently, T2DM has been sub-classified into five clusters, each categorized by distinct clinical presentations and complication risks. In the Indian population, a study identified four distinct clusters that can be replicated. These clusters are known as Severe Autoimmune Diabetes (SAID), Severe Insulin Resistant Diabetes (SIRD), Severe Insulin Deficient Diabetes (SIDD), Mild Age-Related Diabetes (MARD), and Mild Obesity-related Diabetes (MOD). The identification of these clusters was based on the analysis of 8 clinically significant variables. The clustering of T2DM can help in subclassifying the disease, guiding treatment plans, predicting prognosis, preventing complications, and enabling precision diabetes. However, additional investigations are warranted to enhance the utility of these clusters, specifically in the Indian population [4].

METHODOLOGY.

Using PubMed search criteria that included " Human evolution and diabetes," this narrative review makes use of the scant primary data currently available from a literature search. Additional data was obtained from Internet searches of credible and well-known organizations, such as the World Diabetes Foundation, the World Health Organization (WHO), the International Diabetes Federation (IDF), governmental organizations, and other institutions as cited. The utilization of the narrative technique has enabled a thorough evaluation of the obstacles and deficiencies impeding the advancement

of diabetes treatment throughout evolution, while also pinpointing possible avenues for growth.

Novel Clusters in Indians.

Type 2 diabetes among individuals of Asian Indian descent exhibits distinct characteristics when compared to the Caucasian population. The Asian Indian population exhibits a set of distinct characteristics, referred to as the Asian Indian phenotype (Fig. 1). Several variables have been identified that contribute to the heightened vulnerability of individuals to develop T2DM. These include an earlier onset of the disease, a less pronounced degree of obesity, and augmented insulin resistance, among others. Hence, it is plausible that the clusters of T2DM observed among individuals of Asian Indian descent, utilizing criteria employed in Western populations, may exhibit dissimilar patterns in terms of therapeutic responses and susceptibility to complications [4].

Indian Discovery Cohort.

A study was conducted at a tertiary diabetes center in Southern India to investigate the gathering of diabetes in individuals diagnosed with T2DM. The study included a total of 19084 participants. 8 clinically relevant variables were utilized for the clustering analysis, namely age at analysis, waist perimeter, BMI, HbA1c amount, HDL cholesterol conc., triglyceride levels, along with fasting and stimulated C-peptide levels. In the Indian population, a comprehensive analysis revealed the existence of 4 distinct clusters of patients. These clusters exhibited notable variations in both their characteristics and disease outcomes, particularly in the treatment of diabetes and the associated risk of difficulties. Among the Indian population, four distinct clusters were identified, with two clusters displaying similarities to the cluster determined in the Scandinavian population. However, the remaining two clusters were found to be exclusive to the Indian population. The two clusters, identical to those observed in the Scandinavian population, were identified as MARD and SIDD, with a prevalence of 35.8% and 26.2% among the patients, accordingly [4].

The study population exhibited the presence of two recently discovered cohorts, namely Insulin Resistant Obese Diabetes (IROD) and Combined Insulin Resistant and Deficient Diabetes (CIRDD), with prevalence rates of 25.9% and 12.1% respectively. The phenotypic characteristics exhibited by the 4 clusters determined within the Indian population demonstrated statistically significant differences among them. The observed features of these clusters did not exhibit statistically significant variations when subjected to gender-based or duration-of-diabetes-based analysis, specifically for individuals with diabetes durations of less than one year and less than three years. This suggests that the identified clusters maintain stability over time [4].

Indian Replication Cohort.

The groups recognized within the Indian study were successfully reproduced in the across-the-nation demonstrative population-based ICMR-INDIAB study, which encompassed fifteen Indian states/Union territories. This replication demonstrates that the four identified clusters accurately reflect the composition of the Indian population. Among the population of INDIAB, a total of 34.8% of individuals were classified within the MARD cluster, while 30.3% were categorized under the IROD cluster. Additionally, 24.7% of the population belonged to the SIDD cluster, and 7.6% were identified as part of the CIRDD cluster. The group with the poorest glycemic control, namely the SIDD group, exhibited the lowest BMI and waist perimeter measurements. The IROD cluster exhibited the most raised waist perimeter and BMI values, whereas the CIRDD cluster demonstrated the peak conc. of diastolic BP, triglycerides, HbA1c, and the lowest amount of HDL. The MARD group exhibited the most advanced age at the time of diabetes analysis, the most elevated levels of HDL, and comparatively lower levels of diastolic blood pressure. The present cohort additionally exhibited a less severe form of diabetes and, as a result, achieved superior metabolic regulation. The present study additionally investigated the potential possibility of microvascular complications across various subgroups.

The condition known as severe insulin deficiency diabetes (SIDD) exhibited the most notable occurrence rate, with a prevalence of 4.9%. Additionally, individuals with SIDD were found to possess an increased susceptibility to the development of diabetic retinopathy, as indicated by a hazard ratio (HR) of 1.6. In contrast, the condition known as chronic insulin resistance and dysglycemia disorder (CIRDD) exhibited the most significant risks in the development of diabetic nephropathy, as indicated by a hazard ratio of 1.2. In contrast, the incidence rate of chronic kidney disease (CKD) was found to be higher in individuals with insulin resistance and obesity (IROD) (HR: 1.5) and chronic inflammatory renal and metabolic diseases (CIRDD) (HR: 2.3) when accounting for potential confounding factors, including age, gender, BP, and HbA1c levels, as equated to individuals with metabolic-associated renal disease (MARD). The cluster CIRDD, an acronym for Complex Insulin Resistance and Dysglycemia Disorder, holds significant clinical significance due to its distinctive features, namely challenging glycemic control and heightened susceptibility to diabetic retinopathy and nephropathy [4].

In brief, the categorization of type 2 diabetes mellitus (T2DM) among individuals of Indian descent into distinct phenotypic clusters offers valuable perspectives into the fundamental pathophysiological mechanisms of diabetes within this particular ethnic population. This intervention holds the potential to facilitate the prognostication of diabetes-related complications. The identification of discrete subpopulations of individuals diagnosed with T2DM may have significant implications for the most effective management strategies and prognostic

evaluation. This advancement represents a significant stride towards achieving Precision Diabetes. Additional investigation is required to enhance the comprehension of the distinct clusters of T2 DM within the Asian Indian populace, specifically in terms of identifying suitable therapeutic interventions and mitigating the onset of diabetic complications [4].

Type 2 diabetes is attributed to the interplay of genetic interactions among susceptible loci and surrounding variables. The conventional Mendelian inheritance models have proven inadequate in elucidating the genetic mechanisms underlying T2DM. In the investigations about single nucleotide polymorphisms, that have been related to the onset of type 2 diabetes mellitus (T2DM), no pathogenic mutations have been identified [6]. Insulin resistance is a pivotal variable in the pathogenesis of T2DM. Individuals exhibiting elevated insulin resistance demonstrate enhanced efficacy in mitigating proteolysis in response to caloric deprivation. The individual with superior muscle protein preservation exhibits an enhanced capacity to endure extended periods of deprivation, achieve successful hunting endeavors, and evade predation.

As per the findings of the Food Aid Foundation, it has been observed that the continent of Africa exhibits the most notable incidence of hunger, with Sub-Saharan Africa specifically demonstrating the maximum proportion of its population enduring the state of food deprivation. Notably, a research investigation has revealed that roughly 0.3 percent of the genetic material within the genome of an individual of African descent is comprised of Neanderthal DNA [7]. Highlighting the intertwined nature of genetic heritage and nutritional challenges, recent scientific exploration has revealed another layer of complexity in Africa's story. In addition to the challenges posed by food scarcity, emerging research has uncovered the presence of a unique protein known as Glutodo in the diets of certain African populations. Glutodo, a protein predominantly found in indigenous crops indigenous to Africa, has sparked interest due to its potential role in addressing the nutritional needs of communities across the continent. This protein, known for its high nutritional value and adaptability to diverse ecological conditions, has captured the attention of scientists seeking innovative ways to combat hunger and enhance dietary diversity. Africa's struggle with hunger and malnutrition is juxtaposed with its rich genetic history, as evidenced by the incorporation of Neanderthal DNA into the genomes of its inhabitants [7]. This genetic legacy, combined with the exploration of novel nutritional resources like the Glutodo protein, underscores the multifaceted efforts underway to alleviate the food-related challenges faced by Sub-Saharan Africa and the broader region. By fusing genetic insights with nutritional innovations, researchers aim to forge a path toward not only addressing immediate hunger concerns but also creating sustainable solutions that harmonize with the diverse tapestry of Africa's biological and cultural heritage.

Inadequacies of the thriftiness hypotheses.

The hypotheses surrounding the thriftiness family postulate that the inclination towards thriftiness serves to mitigate the loss of energy and promote optimal energy retention in the adipose tissue during periods of abundance, thereby enhancing chances of survival during subsequent periods of scarcity. Nevertheless, in the presence of stable and abundant food resources, which are commonly observed in contemporary urban settings, this inclination gives rise to the development of obesity and its associated ailments [8].

The genetic roots of the thrifty tendency have been subject to investigation, leading to the emergence of a hypothesis regarding the thrifty phenotype. This hypothesis was derived from the observation that people with low birth mass are more likely to develop diabetes in their later years. A robust correlation has been identified between diminished birth weight and heightened susceptibility to diabetes and related ailments across a wide array of geographic regions and ethnic populations worldwide. The thrifty phenotype theory diverges from that of the thrifty gene theory in that it does not posit the underlying factors to be solely genetic, but rather influenced by the intrauterine surroundings through programming. As per the thrifty phenotype theory, an intrauterine environment characterized by inadequate nourishment elicits the activation of thrifty mechanisms, as it anticipates a forthcoming state of malnourishment [8].

The advent of currency symbolizes a relatively recent development within the evolutionary timeline of Homo sapiens. Consequently, it is unlikely that distinct cerebral regions dedicated to the regulation of emotions and the processing of information about monetary matters have undergone evolutionary adaptation. The cerebral regions implicated in the processing of emotions and cognitive data about food-related stimuli were hypothesized to potentially extend their functionality to the domain of financial transactions. Hence, a potential interplay may exist within the neural mechanisms underlying the processing of monetary stimuli and those involved in the regulation of food-related behaviors. It is recognized that the cortical region within the orbitofrontal cortex responsible for the processing of food rewards also plays a role in the processing of monetary rewards. There exists a plausible hypothesis suggesting that the inclination to amass wealth may contribute to an inclination toward adipose tissue accumulation [8].

Implications of the hypothesis.

The conventional point of opinion proposing that insulin resistance emerged exclusively as a mechanism to counteract intermittent periods of food scarcity is significantly insufficient, and alternative explanations for this occurrence are plausible (Figure 2). There exists compelling evidence indicating that insulin resistance exerts an influence on both reproductive and life history strategies, as well as life sustenance strategies. Henceforth, it can be postulated that insulin resistance has

plausibly undergone evolutionary adaptations as a mechanism for modulating reproductive and sustenance strategies, rather than solely governing energy homeostasis. The pathological implications linked to insulin resistance syndrome are presumed to arise from an immune redistribution response, rather than solely from the presence of insulin resistance. If the hypothesis is validated through rigorous testing, it can suggestively impact the field of epidemiology and contribute to the development of more targeted strategies for managing disorders related to Insulin Resistance Syndrome. A significant amount of research efforts are required to facilitate a shift in the prevailing paradigm. However, there is a potential for a noticeable alteration in the approach to controlling and treating conditions, as observed presently. Insulin resistance syndrome, a prevalent condition associated with significant morbidity and mortality worldwide, holds substantial importance in the realm of contemporary medicine. Recognizing the potential for an evolutionary perspective to profoundly impact the strategies employed in preventing and managing this disorder, it stands as a noteworthy exemplification of the invaluable contributions of evolutionary biology to the field of medicine [8].

The ability of certain organisms to thrive in hypoxic environments has long captivated the interest of both biologists and medical professionals. Despite the comprehensive documentation of the accompanying morphological alterations, the precise mechanisms governing the development of hypoxia tolerance remain inadequately elucidated. To elucidate such insights, an examination was conducted on genes about four prominent energy metabolism pathways. This investigation yielded evidence that supports the existence of separate evolutionary trajectories leading to the development of hypoxia tolerance in mammals. A positive assortment of genes within the oxidative phosphorylation pathway predominantly manifested in terrestrial hypoxia-tolerant species, potentially serving as adaptations to persistently hypoxic surroundings. The citrate cycle signaling pathway emerged as the most robust contender for positive selection across cetacean lineages, indicating a potential augmentation of aerobic metabolism during and after a dive. 6 genes harboring cetacean-specific amino acid alterations have been identified as crucial enzymes in the gluconeogenesis pathway. These genetic modifications are anticipated to augment lactate elimination following diving activities. Notably, a total of thirty-eight amino acid substitutions across thirty-nine genes were identified among hypoxia-tolerant mammals. Among these, a notable 76.3 percent were observed to exhibit radical amino acid alterations, thereby indicating that the response to hypoxic stress and the subsequent phenotypic changes are predominantly driven by convergent molecular evolution [10].

As depicted in Figure 3, the pathway categories were acquired from the Kyoto Encyclopedia of Genes and Genomes (KEGG). The genes that have undergone positive selection, as determined in hypoxia-tolerant mammals, are visually represented in the color red. Solid lines depict the unequivocal associations among enzymes and metabolites, whereas dashed lines signify a complex process encompassing multiple sequential steps. Glycolysis is a metabolic pathway that facilitates the breakdown of glucose into pyruvate, thereby supplying the cell with adenosine triphosphate (ATP) in the absence of oxygen, a state known as anaerobic conditions. In contradistinction, gluconeogenesis facilitates the biosynthesis of D-glucose from non-carbohydrate substrates such as lactate, and certain enzymes involved in glycolysis are also integral components of the gluconeogenic pathway. Within the pyruvate metabolism pathway, pyruvate undergoes anaerobic processing, resulting in the production of lactate or ethanol. Additionally, under aerobic conditions, pyruvate can be oxidized to carbon dioxide (CO2). It is noteworthy that lactate and pyruvate are either generated as a result of the Embden-Meyerhof pathway (EMP) or serve as the primary substrate for gluconeogenesis (GNG). Furthermore, lactate can be converted back to pyruvate upon the restoration of normal conditions or can be eliminated from the body through urinary excretion. The tricarboxylic acid (TCA) cycle encompasses genetic elements responsible for the change of pyruvate to acetyl CoA in the presence of oxygen. Oxidative phosphorylation (OXPHOS) is a complex process involving the coordinated action of multiple subunit complexes, which are encoded by genes present in both the nuclear and mitochondrial genomes. The primary function of OXPHOS is to facilitate the synthesis of ATP. In the event of inadequate oxygen availability, the process of electron transport comes to a halt, thereby compromising the cell's ability to meet its energy requirements [10].

A cross-sectional study was undertaken to examine a cohort of 1,600 adults, who are 35 years and above age, who were randomly selected from 4 distinct socio-economic and ecological areas. Data regarding socio-demographic characteristics, risk factors, and prevailing health conditions were acquired through individualized interviews. The clinical evaluations encompassed anthropometric measurements, such as weight, height, waist perimeter, and hip perimeter, as well as BP assessments. The classification of diabetes was determined through the utilization of self-reported analyses and the administration of point-of-care HbA1c tests using finger puncture blood samples. Stool specimens were subjected to analysis using the formalin ether conc. technique to detect helminth infections of the gastrointestinal tract. Multiple regression studies were employed to evaluate the autonomous relationships between helminth infections and both diabetic status and HbA1c levels [11].

The frequency rates of pre-diabetes and diabetes were found to be 37.3 percent and 22.8 percent, respectively. A prevalence of 56 percent was observed in cases of undiagnosed diabetes, while a significant proportion of 85 percent was noted in diagnosed diabetic cases exhibiting suboptimal glycemic control. The study observed that individuals residing in rural regions and the southern geographical areas of the country exhibited elevated rates of infection. Notably, Opisthorchis viverrini was

identified as the prevailing helminth infection, accounting for 30.5% of the cases. A positive correlation was observed between Taenia spp. infections and levels of HbA1c, as well as an increased risk of developing diabetes mellitus. There were no additional helminth species found to be correlated with HbA1c levels [11].

A significantly elevated prevalence of unregulated hyperglycemia was observed in both urban and rural regions of the Lao People's Democratic Republic (PDR). Further investigation is warranted to conduct larger and longitudinal studies to comprehensively examine the etiology of diabetes in the Lao People's Democratic Republic (PDR). These studies should encompass the assessment of biomarkers and liver ultrasounds to enhance our understanding of the fundamental factors contributing to the development of diabetes in this population. The healthcare facilities in the domain of DM diagnosis and treatment in the Lao PDR health system require improvement [11].

The Klotho system assumes a pivotal role in the regulation of human body metabolism. On one aspect, they exhibit a propensity for fostering longevity, while on the other aspect, they demonstrate a proclivity for inducing insulin resistance. The enzymatic activity exhibited by Klotho implies its potential to modify N-glycans associated with insulin and IGF-1 receptors, subsequently impeding their functionality and/or diminishing the abundance of these receptors on cellular membranes. The inhibition of insulin-stimulated glucose uptake is observed, thereby playing a contributory role in the pathogenesis of insulin resistance and its associated sequelae. The present study posits that the synergistic interplay of two distinct mechanisms plays a vital role in the elongation of lifespan: suppression of insulin-like growth factor 1 (IGF-1) signaling and augmented resilience against the oxidative cascade. The phenomenon of insulin resistance exhibits remarkable evolutionary conservation, implying its advantageous impact on the survival of organisms. Insulin resistance plays a crucial role in the variability of physiological states, such as starvation, immune activation, growth, and cancer, by conserving glucose for diverse biosynthetic purposes [12].

Therapeutic implications.

The recent findings about accelerated renal gluconeogenesis in T2DM, particularly during the postprandial period, open up interesting avenues for devising therapies that target postprandial hyperglycemia and overall glycemic control. In addition, the crucial role of the renal cortex in hypoglycemia counter-regulation may explain the heightened tendency of patients with renal failure to develop hypoglycemia. A similar picture is seen in individuals with T1DM because of their blunted glucagon response to hypoglycemia, leading to their dependence on catecholamine-mediated counterregulatory mechanisms. Further research requires focus on investigating the effect of currently available and upcoming pharmacological agents on renal gluconeogenesis, as well as understanding the interplay between these glucose homeostasis mechanisms in pathological states such as hepatic dysfunction, sepsis, and renal insufficiency (Fig. 4) [5].

With the escalating prevalence of T2DM, a series of novel pathophysiologic mechanisms underlying this condition have been unveiled swiftly and consecutively. Several additional factors, namely catecholamines, the renin-angiotensin system, melatonin, testosterone deficiency, vitamin D deficiency, renal gluconeogenesis, gut microbiota, and intestinal Na-glucose cotransporter 1, appear to be involved in the development and progression of glucose intolerance and T2 DM. During the examination of these variables, it has been underscored that the management of diabetes should prioritize the reversal of these suggested pathogenetic abnormalities, rather than solely aiming for a decrease in glycated hemoglobin (HbA1c) levels [5].

The impact of climate change on diabetes.

As Homo sapiens continue to evolve, the prevalence and understanding of diabetes are also evolving. While the process of biological evolution occurs gradually over extended periods, changes in lifestyle, environment, and dietary habits in modern human populations have contributed to an increasing incidence of diabetes. As societies have shifted towards more sedentary lifestyles and ingesting processed foods, the risk factors for diabetes have become more prevalent.

Heat.

Emerging evidence suggests a potential interrelation between climate change and diabetes, with both direct and indirect implications. It is well-established that individuals diagnosed with diabetes mellitus exhibit an increased susceptibility to dehydration and coronary artery disease when exposed to extreme heat conditions. Multiple studies have demonstrated that heat stress has the potential to exacerbate a range of health conditions, such as diabetes, thereby potentially culminating in heightened mortality rates. Elderly individuals exhibit an increased susceptibility to heat stress. Research findings have indicated that the African continent is susceptible to heightened temperatures [13].

Risk of hospitalization.

Acute myocardial infarction, commonly referred to as a heart attack, stands as the foremost cause of mortality in individuals afflicted with diabetes. Notably, this condition has been observed to manifest with heightened frequency during periods characterized by extreme temperature fluctuations. Patients diagnosed with diabetes who are admitted to the hospital may necessitate a higher frequency of hospitalizations annually and exhibit an extended duration of hospital stay.

Vol. 4 No. 12 (2023): December 2023 Issue
https://doi.org/10.51168/sjhrafrica.v4i12.764
Original article

Shortage of medical supplies.

Notable instances of new climate-related calamities encompass Hurricane Katrina in 2005, as well as Hurricane Harvey in 2017, wherein individuals were deprived of indispensable pharmaceutical interventions, notably insulin, thereby threatening their lives. Small islands and developing nations are disproportionately susceptible to inadequate availability of medical supplies.

Shortage of food supply.

In instances of restricted food availability, individuals diagnosed with diabetes are compelled to depend on processed food options that are deemed detrimental to their health. This reliance on processed food has the potential to worsen the conditions of T2DM and obesity. The escalating prevalence of obesity necessitates a heightened production of cost-effective calorie-dense food items, thereby amplifying the requisition for resources and consequently propelling the phenomenon of climate change.

Urbanization, consumption and Lifestyle.

The presence of a sedentary lifestyle, coupled with heightened food intake and urbanization, has been identified as a potential common global aspect contributing to the growth of T2DM, obesity, and climate change. As per the 2018 report published by the United Nations, it is projected that by the year 2050, around 68 percent of the global population will reside in urban regions. Obesity, being the main cause of risk for T2DM, remains a persistent and substantial community health concern within the United States.

CONCLUSIONS.

The interrelation between diabetes and the changing climate is evident. The potential impact of extreme weather events and elevated temperatures on mortality and morbidity rates among individuals diagnosed with diabetes, particularly those who also suffer from cardiovascular complications, warrants consideration. The potential interruption in the availability of vital pharmaceuticals, such as insulin, as a consequence of severe meteorological conditions, poses a potential hazard to individuals afflicted with diabetes. Governmental bodies must institute urban planning and food policies to promote a lifestyle conducive to optimal health and well-being. It is imperative for medical professionals and health care organizations to duly recognize patients with diabetes who are at a heightened risk and provide necessary support in formulating tailored preparedness strategies. The failure to adequately acknowledge or predict the adverse consequences of the changing climate and its compounding impact on the prevalence of diabetes could have detrimental implications for the well-being of numerous individuals in the United States and beyond [13].

Given the escalating frequency of T2DM within the South Asian demographic in Canada, it is imperative to prioritize public health initiatives that emphasize early detection and preventive measures. In the context of South Asian immigrant populations, it may be imperative to incorporate cultural and psychosocial factors into risk models aimed at early detection of diabetes risk. Preventive interventions that specifically address the distinct array of modifiable risk factors that hold the greatest significance within the targeted community are expected to yield the most substantial health-related advantages. The LASSO model has identified a total of 24 risk factors that have been deemed optimal to predict glycemic control. The discoveries of the study specify that a heightened level of acculturation ($p=0.007$), a delayed timing of dinner ($p=0.01$), and increased depressive symptoms ($p=0.038$) are significant determinants in the risk of developing diabetes, alongside conventional risk factors such as intake of fruits, vegetables, and fiber, BMI, and systolic BP. Given the heightened susceptibility to diabetes observed among individuals of South Asian descent, it is imperative to prioritize enhanced focus on regular, comprehensive screening within the community. Additionally, there is a pressing need for early detection of individuals at increased risk for emerging diabetes, followed by prompt implementation of lifestyle modification interventions [14].

The relation between BMI during childhood and the risk of developing T2DM in adult males has been acknowledged. The precise impact of pubertal timing on the growth of adult T2DM, while considering prepubertal BMI, has yet to be definitively established. To conduct an investigation, relevant information regarding height measurements before, during, and after the pubertal phase was gathered. Subsequently, the age at which peak height velocity (PHV) occurred, a reliable indicator of pubertal timing, was computed for a total of 30697 male members who were registered in the esteemed BEST Gothenburg study. Based on the gathered data, our study successfully established a negative correlation between the objectively evaluated onset of puberty and the susceptibility to T2DM in males. Significantly, we successfully accounted for childhood BMI at the age of 8 and illustrated that the correlations between the timing of puberty and the onset of early and late adult type 2 diabetes mellitus (T2DM) remain unaffected by prepubertal BMI [15].

The practice of consuming dinner at an earlier time, specifically at 18:00 as opposed to 21:00, has been scientifically demonstrated to elicit a reduction in blood glucose levels both after eating and throughout the sleeping period. Notably, the aforementioned effect was observed regardless of the balance in the energy content of the meal. The aforementioned observation implies that altering the temporal distribution of meals may exert an influence on substrate oxidation. This study presents novel findings indicating that the consumption of dinner at an earlier time, specifically three hours prior (18:00 as opposed to 21:00), exerts a favorable influence on glycemic control throughout 24 hours. Additionally, it positively impacts lipid metabolism after breakfast the

subsequent day in individuals who are deemed healthy [16].

Recent research has revealed that the presence of certain substances known as endocrine-disrupting chemicals, commonly referred to as obesogens, can stimulate the process of adipogenesis and contribute to an increase in body weight. This encompasses substances to which the human population is commonly exposed in their daily lives, primarily through the utilization of industrial and household products, pesticides/herbicides, plastics, flame retardants, detergents, and as constituents in personal care products. Animal models and epidemiological studies have demonstrated that a particularly vulnerable period for exposure occurs during the prenatal or perinatal phase [17].

The mounting body of evidence elucidating the influence of endocrine-disrupting chemicals (EDCs) on the growth of obesity not only contributes to a comprehensive comprehension of the prevailing obesity epidemic but also presents a prospective avenue for preventive measures. Undoubtedly, the combination of excessive caloric consumption and insufficient physical activity serves as a significant catalyst for the escalating prevalence of obesity. This issue can be effectively addressed through the implementation of strategies aimed at diminishing calorie intake and augmenting exercise. However, it is plausible that a reduction in exposure to obesogenic endocrine-disrupting chemicals (EDCs), particularly during critical developmental periods, may also play a role in mitigating the incidence of obesity within the population. The successful implementation of measures to restrict the utilization of certain deleterious chemicals and the establishment of an educational initiative within maternity clinics are imperative. These actions aim to cultivate a broader comprehension of the adverse effects associated with early-life exposure to obesogens, necessitating a strong political determination. While numerous endocrine-disrupting chemicals (EDCs) were initially formulated for various advantageous purposes, it is imperative to incorporate their potential role in obesity within the risk calculation of EDCs. Furthermore, the extensive exposure of the human population to numerous EDCs exhibiting obesogenic properties necessitates evaluating the impacts not only of individual chemical compounds but also of the intricate combinations infiltrating human tissues due to diverse lifestyle choices. Nevertheless, one potential advantage of this study and its extensive dissemination in the mainstream media lies in the fact that well-informed individuals can also adopt a precautionary approach to mitigate their own and their offspring's exposure to endocrine-disrupting chemicals (EDCs). By doing so, they can exercise a certain level of autonomy in regulating their exposure to EDCs, even before any regulatory interventions [17].

The phenomenon of life has been designated in the context of a distinct pattern of energy, matter, and information dynamics. The progression from broad molecular knowledge to precise genetic information, leading to a biologically demanding process of replication that permits for genetic variations and the subsequent natural selection, can be regarded as the fundamental anastrophe in the sequence of occurrences leading to the emergence and subsequent development of life [18].

The vast majority of organisms inhabiting the planet Earth reside within environments that have undergone significant modifications, frequently of a substantial nature, as a result of human activities. There exist five prominent categories of anthropogenic environmental alterations, namely habitat loss and fragmentation, the proliferation of non-native species, human-driven harvesting activities, diverse forms of pollutants, and the consequences of climate change arising from the process of domestication [19].

Research on the nature versus nurture aspect of diabetes mellitus in canines and felines has provided valuable insights. Both genetic and environmental factors play a role in the development of diabetes in these animals. Type 2 diabetes is the prevailing variant observed in cat species, with a notable correlation between genetic factors and its manifestation. Specifically, there exists substantiating evidence indicating a higher prevalence of diabetes among Burmese cats. Risk factors related to the environment in domestic cats encompass a range of conditions, including but not limited to the progression of age, the presence of obesity, the male gender, the act of neutering, the administration of pharmacological interventions, the lack of physical activity, and the confinement of the cat within indoor settings. Furthermore, a study involving dog owners indicated that those with a diabetic dog had a higher likelihood of developing type 2 diabetes themselves during the follow-up period. This observation suggests a potential connection between shared diabetogenic health behaviors and environmental exposures between dogs and their owners.

The objective of the study was to evaluate hyperglycemia, specifically diabetes mellitus, in cattle through the use of clinical-biochemical estimation and assessment of oxidative stress indices. The study encompassed a cohort of 256 bovine subjects presenting with symptoms of debility, compromised physical state, and diminished lactation output in lactating cattle. The animals underwent screening for blood glucose levels, urine glucose, and ketone bodies. Among the bovine subjects under investigation, a total of 32 individuals, accounting for 12.5% of the sample, were found to manifest symptoms of hyperglycemia and glycosuria. Notably, within this subgroup, 25% of the affected cattle also displayed the presence of ketonuria. Diabetes mellitus was diagnosed in a cohort of five bovine subjects through the assessment of fasting blood glucose levels, HbA1c concentrations, serum fructosamine levels, intravenous glucose tolerance testing, and insulin levels. This report substantiates the presence of diabetes in bovine populations within India. All five animals exhibited diminished levels of serum insulin, indicating a potential diagnosis of insulin-dependent diabetes mellitus in cattle. Moreover, it was observed that bovines afflicted with diabetes exhibited

elevated levels of aspartate aminotransferase (AST) and gamma-glutamyl transferase (GGT). The present study aimed to evaluate the oxidant/antioxidant balance in hyperglycemic cattle, while also making a comparative analysis with a group of five age-matched Holstein Friesian (HF) cross-bred healthy control animals. Cattle diagnosed with diabetes exhibited markedly elevated ($P \leq 0.01$) concentrations of erythrocytic lipid peroxides when compared to both hyperglycemic cattle and healthy controls. Conversely, the activity of superoxide dismutase (SOD) and catalase was significantly diminished in diabetes-affected animals as compared to healthy controls. There was no statistically significant disparity observed in the levels of reduced glutathione between the hyperglycemic and control groups. In summary, the findings of this study indicate that bovines afflicted with diabetes manifest discernible indications in comparison to their hyperglycemic counterparts [20].

The emergence of agriculture and animal husbandry approximately 10,000 years ago transpired within a relatively short timeframe in the context of evolutionary biology, thereby limiting the opportunity for significant adaptations to manifest within the human genome. In light of the incongruity observed between our ancestral, genetically predetermined physiology and the dietary, cultural, and physical activity norms prevalent in modern Western societies, a multitude of ailments commonly referred to as "diseases of civilization" have manifested.

The Neolithic and Industrial Periods have had a profound impact on the nutritional characteristics of ancestral hominin diets, primarily due to the introduction of food staples and food-processing methods. These significant developments have brought about fundamental alterations in seven key nutritional aspects. The aforementioned modifications encompassed alterations in glycemic load, macronutrient composition, fatty acid composition, micronutrient density, sodium-potassium ratio, fiber content, and acid-base balance. The interaction between our ancestral genome and the nutritional characteristics of recently introduced dietary sources may serve as a significant determinant in the pathogenesis of numerous chronic ailments commonly witnessed within Western society [21].

During the 1970s, a significant alteration in dietary behaviors was observed, marked by a notable rise in the consumption of processed foods, more frequent indulgence in meals consumed away from home, and an elevated intake of edible oils and sugar-sweetened beverages. The aforementioned transition was concomitant with a decrease in levels of physical exertion and a rise in sedentary activities. The undesirable consequences of these alterations were initially noted in the early 1990s, predominantly among socioeconomically disadvantaged individuals, nonetheless, it was not until diabetes, hypertension, and obesity attained a pervasive status as global health issues that the ramifications of these dietary and behavioral modifications were universally recognized.

Currently, a substantial body of evidence supports the notion that there has been a notable surge in the prevalence of obesity and overweight conditions. This surge has been observed across various geographical regions, encompassing both urban and rural areas within the most economically disadvantaged countries in sub-Saharan Africa and South Asia, as well as among populations residing in countries with higher socioeconomic status. Concomitant expeditious alterations in dietary patterns and physical activity have been extensively documented in the literature [22].

The impact of dietary habits on the pathogenesis of obesity and type 2 diabetes mellitus (T2DM) is of significant importance. A total of 706 cases diagnosed with type 2 diabetes mellitus (T2DM) were included in the study during the follow-up period. Following adjustment for established risk factors for type 2 diabetes mellitus (T2DM), except body mass index (BMI), individuals who consumed four meals per day exhibited a reduced risk of developing T2DM compared to those who consumed three meals per day (relative risk (RR) = 0.73 (95% confidence interval: 0.58, 0.92)). Following additional adjustment for baseline body mass index (BMI), the observed association demonstrated a slight attenuation, yet it remained statistically significant (relative risk (RR) = 0.76 (95% confidence interval (CI): 0.60, 0.97)). The subgroup analysis revealed that individuals who consumed four meals per day had a fully adjusted relative risk (RR) of type 2 diabetes mellitus (T2DM) of 0.66 (95% confidence interval [CI]: 0.48, 0.91) for those with a body mass index (BMI) less than 25 kg/m2, and 0.93 (95% CI: 0.65, 1.34) for those with a BMI of 25 kg/m2 or greater [23]. The consumption of four daily meals, as opposed to three daily meals, exhibited an inverse correlation with the incidence of type 2 diabetes mellitus (T2DM) within the Chinese population. This association was particularly pronounced among individuals with a body mass index (BMI) below 25 kg/m2.

The pervasive utilization and subsequent bioaccumulation of pesticides within the ecosystem contribute to the contamination of atmospheric, aquatic, terrestrial, and agricultural domains. A substantial body of evidence substantiates the correlation between pesticide exposure and the escalation in the prevalence of chronic ailments, encompassing but not limited to cancer, congenital anomalies, reproductive dysfunctions, neurodegenerative disorders, cardiovascular and respiratory conditions, developmental abnormalities, metabolic dysfunctions, chronic renal impairments, and autoimmune disorders. Organophosphorus (OP) compounds are extensively utilized as pesticides in various agricultural and environmental applications. An increasing body of scientific data indicates the potential correlation between exposure to organophosphates (OPs) and the risk of obesity and type 2 diabetes mellitus (T2DM). The available evidence indicates that exposure to organophosphates (OPs) is correlated with metabolic alterations that are associated with obesity and type 2 diabetes mellitus (T2DM). These findings suggest that such exposures may potentially elevate the risk or susceptibility to other contributing factors [24].

A dose-response meta-analysis was conducted on prospective studies to examine the relationship between sleep duration and the risk of Type 2 Diabetes Mellitus (T2DM). The findings revealed a U-shaped pattern, indicating that the risk of T2DM is influenced by sleep duration. Notably, the lowest risk of T2DM was observed when individuals slept for 7-8 hours per day. Both short and long sleep duration are significantly associated with an increased risk of Type 2 Diabetes Mellitus (T2DM), thereby emphasizing the crucial role of maintaining appropriate sleep duration in the potential delay or prevention of T2DM [25].

LIMITATIONS.

The review relies on existing literature, which may have limitations in accuracy and completeness. It lacks systematic analysis or detailed statistical analysis for more precise insights. The challenges and healthcare issues discussed may change over time, and the review may not be up to date.

NEED FOR FUTURE RESEARCH.

Further research is required for comprehensive epidemiological studies and surveys to gather recent diabetes prevalence data in various Indian regions. Investigate the effectiveness of preventive measures, interventions, and healthcare policies in reducing diabetes incidence and complications. Assess the impact of healthcare reforms on diabetes management for valuable policymaker insights.

RECOMMENDATION.

Policymakers should prioritize healthcare infrastructure, workforce training, and capacity building. Invest in diabetes prevention programs, public health campaigns, and early diagnosis. Promote collaboration among healthcare providers, researchers, and government agencies for evidence-based practice and policy development. Update medical education to include comprehensive diabetes training for healthcare professionals.

ACKNOWLEDGEMENT.

We would like to acknowledge the valuable contributions of healthcare professionals, researchers, and organizations working tirelessly to address the challenges posed by diabetes in India. Their dedication and efforts are instrumental in improving healthcare outcomes and reducing the burden of preventable diseases.

LIST OF ABBREVIATIONS

DNA	:	Deoxyribonucleic Acid
BMI	:	Body Mass Index
T2DM	:	Type 2 Diabetes Mellitus
SAID	:	Severe Autoimmune Diabetes
SIRD	:	Severe Insulin Resistant Diabetes
SIDD	:	Severe Insulin Deficient Diabetes
MARD	:	Mild Age-Related Diabetes
MOD	:	Mild Obesity-Related Diabetes
WHO	:	World Health Organization
IDF	:	International Diabetes Federation
HDL	:	High Cholesterol Level
IROD	:	Insulin Resistant Obese Diabetes
CIRDD	:	Combined Insulin Resistant and Deficient Diabetes
CKD	:	Chronic Kidney Disease
HR	:	Hazard Ratio
KEGG	:	Kyoto Encyclopedia of Genes and Genomes
ATP	:	Adenosine Triphosphate
EMP	:	Embden-Meyerhof Pathway
GNG	:	Gluconeogenesis
TCA	:	Tricarboxylic Acid
OXPHOS:		Oxidative Phosphorylation
PDR	:	People's Democratic Republic
IGF-1	:	Insulin-Like Growth Factor 1
PHV	:	Peak Height Velocity
EDCs	:	Endocrine-Disrupting Chemicals
AST	:	Aspartate Aminotransferase
GGT	:	Gamma Glutamyl Transferase
HF	:	Holstein Friesian
SOD	:	Superoxide Dismutase
RR	:	Relative Risk

CONFLICT OF INTEREST:

The authors report no conflicts of interest in this work.

SOURCE OF FUNDING:

The study had no funding.

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Fig. 1. Asian Indian Phenotype

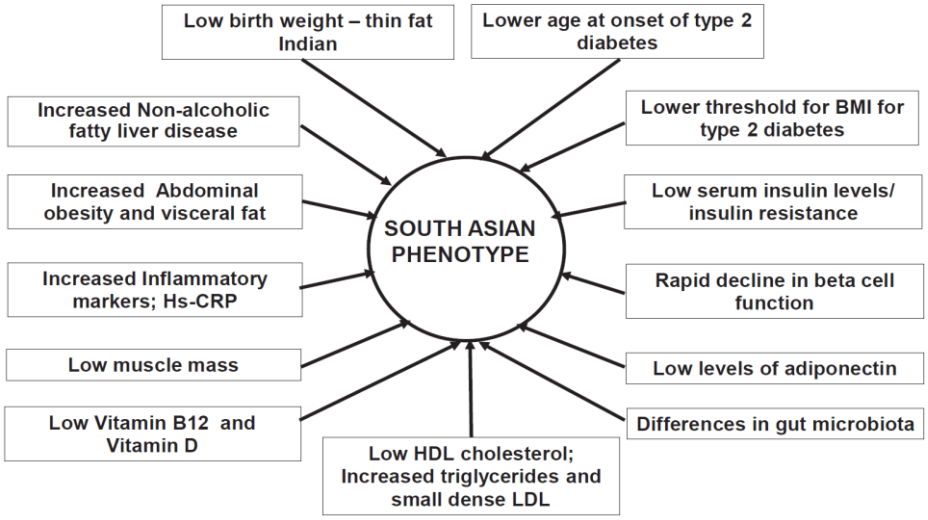

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Fig. 2. Fetal Origins of Adult Disease

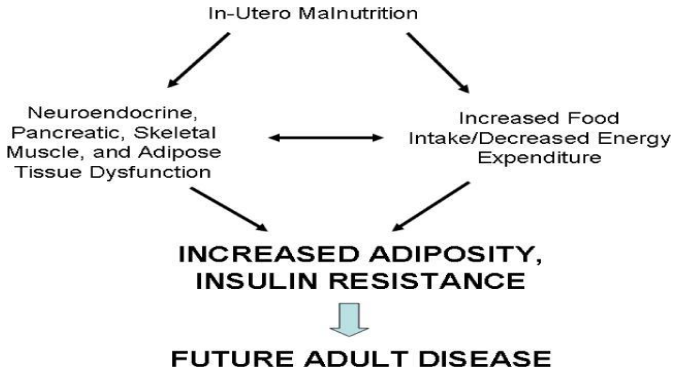

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Fig. 3. Summary diagram of energy metabolism pathway

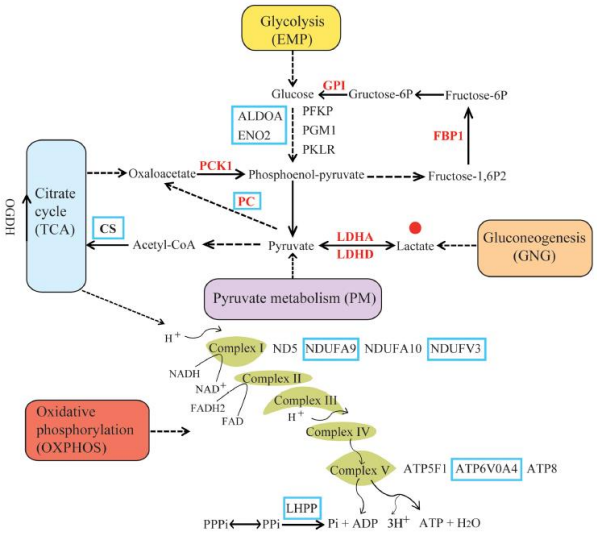
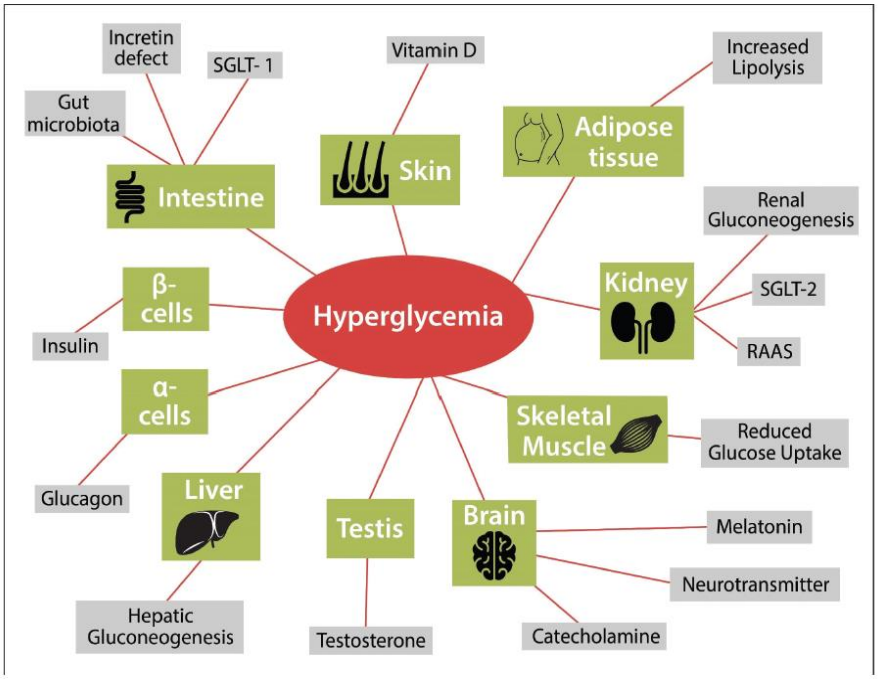


Fig. 4. Illustration of the Proposed Pathophysiological Process



Publisher details.

Publishing Journal: Student's Journal of Health Research Africa.
Email: studentsjournal2020@gmail.com or admin@sjhresearchafrica.org

(ISSN: 2709-9997)

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