ISSN: 3048-6181 www.wjphr.org 2025, Volume 2, Issue 12. Page: 01-09.

World Journal of Pharmaceutical and Healthcare Research

Review Article

INSIGHTS FOR CLINICAL INTERVENTIONS AND PUBLIC HEALTH STRATEGIES IN OBESITY AND DIABETES ASSOCIATED WITH THE COVID-19

Dr. Chrysanthus Chukwuma Sr*

Executive Director, The Chrysanthus Centre for Future-Oriented Studies, Abakaliki, Ebonyi State, Nigeria.

Article Info
Article Received: 03 November 2025,
Article Revised: 25 November 2025,
Published on: 01 December 2025.

***Corresponding author:**

Dr. Chrysanthus Chukwuma Sr

Executive Director, The Chrysanthus Centre for Future-Oriented Studies, Abakaliki, Ebonyi State, Nigeria.

https://doi.org/10.5281/zenodo.17909209

How to cite this Article:
Dr. Chrysanthus Chukwuma Sr, (2025). INSIGHTS FOR CLINICAL INTERVENTIONS AND PUBLIC HEALTH STRATEGIES IN OBESITY AND DIABETES ASSOCIATED WITH THE COVID-19. World Journal of Pharmaceutical and Healthcare Research, 2(12), 1–9.

ABSTRACT

This review provides a valuable contribution to elucidating how metabolic derangements influence COVID-19 susceptibility, outcome and prognosis. It engages complex metabolic interplays and proposes dietary and therapeutic modalities targeted at these vulnerabilities. In conclusion, this review emerges as a pertinent resource for clinicians, public health experts, and researchers dedicated to address the dual burden of the similar COVID-19 pandemic and pre-existing metabolic aberrations. Its insights will be instrumental in carving more effective and efficient clinical interventions and public health approaches. Thus, the study provides a conceptus of the predominant characteristics of diabetes, obesity and SARS-CoV-2/COVID-19 pandemic as well as the associated carbohydrate-lipid interactions and metabolism which may potentially impact public health and the clinical process, including socioeconomic burden. Obesity and diabetes are pronounced comorbidities for SARS-CoV-2. The complex interactions between carbohydrate and lipid metabolism establish a reverting cycle that exacerbates the hallmarks of SARS-CoV-2. Obesity and diabetes both highlight systemic dysfunctions in carbohydrate and lipid metabolism which are aggravated by COVID-19 infection creating an anomalous feedback loop worsening metabolic disorders from COVID-19 outcomes, while the infection, in turn, exacerbates prevailing metabolic issues or triggers nascent problems. The COVID-19 pandemic culminated in an expansive mortality as a repercussion of the SARS-CoV-2. The SARS-COV-2 encompassed by a lipid bilayer promotes fusion of the viral membrane to the host cell, replication, endocytosis, exocytosis and functionality of lipid metabolism in viral infectivity. Deficiency in proper drugs and vaccines, constitute never-ending opportunities for antiviral treatments or therapies. Diet suffused with carbohydrates and saturated fats enhance obesity and diabetes prevalence, oxidative stress and comorbidities occurrence as risk attributes for COVID-19 pandemic as a public health enigma. COVID-19 outbreak has invariably constituted a traumatic albatross to global public health system with resultant stumbling strategies to obviate the disorder. However, carbohydrates may offer rapid diagnostics, proper, effective and efficient vaccines and therapeutic regimen.

KEYWORDS: SARS-CoV-2, carbohydrate-lipid interactions, comorbidity, pathogenesis, pathophysiology.

INTRODUCTION

This study seeks to delve into newfangled principles for evaluating and monitoring risk factors of communicable and non-communicable diseases, substantiation and integration of the processes into intersectoral healthcare and clinical research. Coronavirus disease 2019 (COVID-19), a designated novel coronavirus severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) reached pandemic levels causing pronounced morbidity and mortality globally. Obesity, diabetes, and metabolic syndrome (MetS) patients were mostly vulnerable, susceptible and easily succumbed to severe side effects of the virus. The metabolic perturbations in obesity and diabetes can establish a suitable ambient for viral interaction, infection and severe discomfort. The global epidemic of obesity and type 2 diabetes increased pari passu with adverse metabolic events. Expansive evidence exists that the type of carbohydrate consumed is commensurate to the development or prevention of insulin resistance, obesity, and the metabolic syndrome, whereas

there is paucity of published data on the combined impacts of interactions between micronutrients, carbohydrates and lipids. However, extensive data exist undergirding the essentials of both carbohydrates and lipids in various processes of energy balance and disorders. Adequate modalities are pertinent to control the impact of carbohydrate-lipid interactions on obesity and diabetes. The impact of foods and nutrients as complimentary strategies on COVID-19 treatment, recovery[1], restoration and sustainability[2] are evident, even though, the intricate complexity of the disease, scarcity of veritable vaccines[3], emerging variants of concern[4], syndemics or comorbidities[5] geopolitics and gain-of-function research[6,7] have been expansive stumbling blocks. The pro-inflammatory presenting milieu in patients with metabolic disruption culminated in COVID-19-mediated host immune dysregulation, such as immune dysfunction response, severe inflammatory disorder, microvascular dysfunction, and thrombotic disorders. The review expresses the extant data regarding the impact of obesity, diabetes mellitus, and MetS on COVID-19 infection, severity, and the pathophysiologic mechanisms. The pro-inflammatory conditions exhibited in patients with metabolic disruption can lead to COVID-19-mediated host immune dysregulation, such as immune derailment, exacerbated inflammation, microvascular impair8, and thrombosis. This entry expresses the available data on the resultant iSARS-CoV-2 impact on obesity, type 2 diabetes mellitus, and MetS and concomitant pathophysiological processes.

Parametric factors exacerbating obesity and diabetes in coronavirus sequelae

The study finally delves into dietary strategies and therapeutic interventions aimed at mitigating adverse outcomes, emphasizing a nuanced understanding of macronutrient interactions in metabolic health and disease scenarios. Prior obesity and metabolic disease propound the severity of acute disease, but SARS-CoV-2 infection is also contributory in exacerbating comorbidities. COVID-19 pandemic with its associated variants depict clinical findings and variants with several risk factors for adverse morbidity, sequelae and mortality in susceptible and vulnerable persons presenting diverse risk factors, for instance, obesity and diabetes with resultant untoward trajectories of COVID-19.[7,8] The comorbid epidemic of obesity and type 2 diabetes globally exhibited the metabolic disruptions critical to aetiological events. Adipose tissue (fat) in obese patients has a high ACE2 receptor expression, the major entrant for SARS-CoV-2 into host cells. This depicts adipose tissue as a viral reservoir, increasing the total viral body load. Virus infection of the host reduces ACE2 receptor functionality. In persons with pre-existing obesity and diabetes, presenting compromised systems, this downregulation may culminate in severe perturbations in ACE2 regulated systems, such as the renin-angiotensin system, with further damage to organs. Diet becomes a potent functionality in modulating metabolic syndrome expression correlated with concentrations and varieties of carbohydrates and lipids as well as their interactions as vital parameters.[9] Insulin resistance is crucial in the metabolic syndrome due to relative insulin failure to regulate the several biological effects on carbohydrate and lipid metabolism. There is lack of published data on the combined impacts on interaction between micronutrients, dietary fats and carbohydrate varieties. Currently, there are expansive data undergirding the benefits of both fat and carbohydrates moieties on various processes of metabolism, energy balance, and disorders. There are processes which depict how the quality of dietary carbohydrate may influence weight gain by the absorption rate or the extent of carbohydrate fermentation.

The type of carbohydrate type is significant to the development or obviating of insulin resistance, obesity, and the metabolic syndrome as to prevent, control and treat the presenting condition.[10,11] Diabetes is characteristically a metabolic disorder presenting chronic hyperglycaemia and immune-mediated type 1 diabetes, insulin-resistant type 2 diabetes, gestational, genetic, environmental, infectious, behavioural or drug-induced impairments[12], and in syndemics or comorbidity with obesity[13,14], and relatively with SARS-CoV-2. Evolutionarily, the adaptation of living organisms is related to utilization of divergent nutrient resources including carbohydrates, lipids and proteins. Both carbohydrates and lipids are the major components of macromolecular ingredients regarding intracellular storage for energy production. Of significance, the anabolism and catabolism of these macromolecules are inextricably-linked in organs linked with nutrient regulation, such as the brain, liver, adipose tissue, pancreas and muscle.[15] Globally, obesity rate has been grossly exacerbated, with imposition on already economic and social burden regarding quality of healthcare and life.

Metabolic, pathogenetic and pathophysiological alterations

It is not easy to define a pathway for carbohydrate and lipid metabolism as well as their elusive interactions. Plants and animals conserve lipids from carbohydrates, but the reverse is portrayed in plants and, not evidentially depicted in animals.[8] In normal human metabolism, carbohydrate-lipid interactions are not easily definable, but the disruptions in diabetic metabolic exhibit these interactions. Carbohydrate or lipid constitutes the prominent source of body fuel but the metabolic pathway differs in chemical mechanisms and functions. It is manifest in the low concentration of body carbohydrate, as observed in severe fulminating diabetes and starvation whereby lipids make provision for body fuel, and it is also detected in the blood and internal organs, especially in the liver.[12,16] With reduced availability of carbohydrate required for metabolism, depot lipid is delivered to the liver and catabolized to ketone bodies, and peripherally burned in the muscles distinctly carbohydrate metabolism.[15] This functions as a normal process, but mainly deleterious in diabetes. Diabetic obesity is commonly diagnosed at the commencement and soon after insulin therapy. It is likely

that hyperglycaemia induces the obesity as determined in unique conditions of lipaemia, diabetes and lipodystrophy. Lipaemia is detectable in two divergent metabolism phases: (i) anabolic for storage; and (ii) catabolic in the storage flow to the liver for manifest disease.[16] The presentation is inter alia fatty enlargement of the liver in diabetic children that may rapidly not be evident during therapy and control, including chronic ketosis. As observed in high-income societies, increased obesity and metabolic syndrome prevalence predispose to pathophysiological changes which lead to non-alcoholic fatty liver disorder that triggers acute hepatic organ dysfunctionality, simultaneously with cirrhosis and oncological hepatocellular degeneration.[17] Disparities in insulin response, beta-oxidation, lipid transport and storage perturbations, autophagy, disequilibrium in chemokines and nuclear receptor signaling are involved in these alterations. Insulin, adipokines, epinephrine and other agonists stimulate regulatory pathways which key metabolic enzymes play a role in the integration of carbohydrate and lipid metabolism. Specific overnutrition distorts these pathways with sequelae of insulin resistance and type 2 diabetes.[17] The pathogenesis of obesity, type 2 diabetes and insulin resistance is associated with deranged lipid and carbohydrate metabolism. Obesity development is a complex mechanism associated with genetic vulnerability and environmental attributes which are not clearly explicated The complexity of communicable and non-communicable diseases (NCDs) in vulnerable populations arises from the encompassing of these two disease burdens, which are associated socioeconomic variants aggravated by conditions poverty and limited access and resources to healthcare and healthcare delivery. Susceptible communities counteract higher risks of both communicable diseases, such as HIV/AIDS, tuberculosis, malaria, and neglected tropical diseases and noncommunicable diseases NCDs, such as cardiovascular diseases, cancer, obesity, diabetes, and chronic respiratory diseases. The combination of these diseases creates a "double burden" that is more challenging to manage, especially in low-income countries and marginalized communities.[18] Lipid aggregation in inflammation and ER stress are clinically evidenced in liver regeneration and cancer degeneration.[19] The diabetes subclass characterised as not insulin-responsive or noninsulin dependent, NIDDM portrays insulin resistance and hyper-insulinaemia, with resultant glucose intolerance, hyperglycaemia and deteriorating diabetes, and comorbidity of ketosis and obesity.[13] Perspectively, obesity and dysfunctional metabolic disposition are specific risk factors for noncommunicable diseases, such as type 2 diabetes which are obviously complicated with coronavirus disease 2019 or COVID-19.[20]

Inextricably-linked gene-environment complexities

The review relates that individuals having obesity and T2DM, due to pre-existing metabolic interruptions constitute a vulnerable cohort for grossly untoward COVID-19 outcomes. It articulates biochemical and pathophysiological interactions between viral activities and metabolic dysfunctions in these communities. The study highlights the contribution of carbohydrate-lipid dynamics to increase insulin resistance and inflammation, which are leveraged by the virus to augment its pathogenicity. Also, the efect of environmental and genetic factors in mediating these comorbidities is explored. Advanced age, comorbidities and vulnerable populations are susceptible to SARS-CoV-2 as genetic, epigenetic, environmental and interaction attributes[21] detectable in obesity and diabetes.

Obesity is a crucial aspect of the metabolic syndrome, predisposing to the development of type 2 diabetes. There is unexplained and unexplained aetiology for the elevated incidence of diabetes and metabolic syndrome globally. This may be genetically influenced, and due to the prevalence of these diseases.[22,23] It has not been elucidated how genetic variables interact with environmental and dietary factors to accelerate their incidence.[22,23] However, genes associated with pathways of carbohydrate, lipid and amino acid metabolism, glycan biosynthesis, as well as pathways in the metabolism of cofactors and vitamins, ubiquitin mediated proteolysis, signal transduction, interactions of neuroactive ligand-receptor interactions, nervous system, and neurodegenerative perturbation pathways are upregulated in obesity in contrast to normal individuals.[24] On the contrary, genes associated with molecules of cell adhesion, cytokine-cytokine receptor interaction, insulin signaling and immune system pathways are downregulated in obesity. Genes involved in signal transduction, actin cytoskeleton regulation, processing and delivery of antigen, complement and coagulation cascades, axon guidance and pathways of neurodegenerative impairment are upregulated in type 2 diabetic persons with family history of diabetes in contrast to diabetic individuals without any family history. Genes associated with pathways of oxidative phosphorylation, immune, nervous system and metabolic disorders are upregulated in diabetic persons presenting diabetes family history, but not in diabetes without family history.[24] Obversely, genes related in lipid and amino acid pathways, ubiquitin mediated proteolysis, signal transduction, insulin and PPAR signaling pathways are downregulated in diabetic persons, and concomitant family history. Genes related in inflammatory pathways are differentially expressed in both obesity and type 2 diabetes. These indicate that genes associated with carbohydrate, lipid, and amino acid metabolic pathways, neuronal functionality and inflammation are important in the pathology of obesity and type 2diabetes. Genetic predisposition contributes expansively to obesity as established by familial aggregation, twin and adoption investigations.[22,23] Obesity emerges due to energy intake, mostly accumulated as triglycerides in excess of energy expenditure[24]; and governed by age, diet, developmental stage, genes and physical activity.[25] The increase of obesity prevalence correlates with type 2 diabetes and impaired glucose tolerance prevalence[26] and multiplicity of sequelae of both disorders, such as arthritis, hypertension, sleep apnoea, cardiovascular disorders and organ

derangement.[27] Due to the global increase in obesity and diabetes prevalence, future life expectancy of affected individuals decline[28] with or without further interactions with SARS-CoV-2.

Carbohydrate- and lipid metabolism in the SARS-CoV-2 ERA and normal milieu

The study provides an expansive investigation of the interrelations between carbohydrate-lipid interactions, obesity, type 2 diabetes mellitus (T2DM), and the repercussions in the SARS-CoV-2 era. It explores the mechanisms which worsen health outcomes for individuals presenting with these metabolic disorders amidst the COVID-19 pandemic. The article endeavours to conglomerate several reflections and perspectives on the viral interaction with an established deranged metabolic condition, thus, creating a comprehensive exposition of the intricate biological systems. COVID-19 is associated with perturbed glucose metabolism, even in the absence of erstwhile diabetes. Several patients develop hyperglycaemia upon hospital admission, being a predictor of poor outcome or prognosis. New-onset diabetes and insulin resistance have been detected following COVID-19 infection, partly because the virus infects and deranges insulin-producing pancreatic beta cells which concomitantly express the ACE2 receptor. SARS-CoV-2 influences lipid metabolism of the host to promote viral replication and assembly. The outer lipid envelope of the virus acquired from the host cell is pertinent for its functionality. The spike protein of the virus also binds to high-density lipoprotein (HDL) cholesterol, and low HDL levels correlate with increasingly severe disorders. Distinctly, in obese individuals, obesity and diabetes characterized by chronic, low-grade inflammation in SARS-CoV-2 infection can hyperactivate the immune system with resultant "cytokine storm." The unmitigated inflammation may lead to acute respiratory distress syndrome (ARDS), multi-organ dysfunction, and mortality. The pathogenic SARS-COV-2 is potentiated for untoward metabolic alterations in viral infection that enhances its survival, cell morphology modifications, inflammation, tissue derangement and vicious alterations in glucose and lipid metabolism.[29] Competition for respiration within substrates in animal tissues is observed in the quantitative interaction between glucose and fatty acids as manifested in the Glucose Fatty Acid Cycle of the reciprocal metabolic association between glucose and fatty acids. Glucose production translates to glucose oxidation including glucose and lipid storage, with suppression of fatty acid oxidation. Free fatty acid availability predisposes to fatty acid oxidation and oxidation, with suppression of glucose oxidation and increased glucose storage during glycogen reserve depletion.[30] Fatty acid oxidation inhibits glucose production in liver with specific enzyme processes. Permissively, fatty acids, during insulin secretory response of islet beta-cells is observed to be a protective mechanism in incessant availability of respiratory substrate. Extended islet beta-cell exposure to fatty acids degrades the insulin secretory response to glucose and the extant processes. Fatty acid oxidation correlates with dysfunctional glucose oxidation in uncontrolled type 1 and type 2 diabetes mellitus. Type 2 diabetes perturbation exhibits glucose storage inhibition leading to prolonged of plasma FFA I elevation in humans and experimental animals that is associated with glycogen depletion. Conversely, glucose storage inhibition in type 2 diabetes is proportional to glycogen reduction.[30] The specific functionality of fatty acids in impaired carbohydrate metabolism in type 2 diabetes creates the trajectory for future research, especially in the milieu of SARS-CoV-2.

It is evident that combustion pervades life, and stringent research directs itself on the significant fuel substrates which make provision for sustenance. These are in competitive interaction among themselves for respiratory combustion. Several characteristics of imminent mutual interaction depicted in both reciprocal and dependent attributes between glucose and lipid metabolism[31] include (i) the inhibitory effects of elevated levels of fatty acids on glucose oxidation through inactivation of mitochondrial pyruvate dehydrogenase or by means of desensitization of insulin-mediated glucose transport; (ii) the inhibitory effects of elevated glucose levels on fatty acid oxidation through malonyl-CoA regulation of fatty acid entrant to the mitochondria; and (iii) the stimulatory impacts of exacerbated glucose abundance on de novo lipogenesis, i.e. lipid synthesis from glucose via SREBP1c glycolytic and lipogenic enzyme regulation. Energy-lowering substrate cycling connecting glucose and lipid metabolism to thermogenesis acts as a modulating strategy that regulates intramyocellular lipid homeostasis leading to the undergirding of skeletal muscle against lipotoxicity.[30-34]

The rate of lipid oxidation may be proportional to plasma free fatty acid levels. Lipid contribution to energy expenditure is higher in obese in comparison to control individuals. This is taken over by lipids in the energy metabolism of both diabetic and non-diabetic obese subjects, and it is the repercussion of increased fat levels. The simultaneous suppressed carbohydrate metabolism ostensibly occurs as complications to glucose tolerance variations, culminating slow glucose storage and oxidation in the obese individual.[35] In unperturbed pregnancy, plasma triglycerides are increased 2-3 fold. Absence of a pronounced impact of diabetes, body weight, and enhanced carbohydrate intake on blood lipids during gestation distinguishes pregnancy from atherosclerosis-associated hypertriglyceridaemias; and identifies to a specific physiologic regulatory mechanism.[36]

Carbohydrate and lipid metabolism is markedly dependent on mitochondria regulated by NRF-1.[37] The metabolic pathways are controlled due to metabolic variations at disparate magnitudes of high energy substances, such as AMP/ATP and the NAD+/NADH ratios. Decreased levels of ATP activate AMPK that activates eclectic transcription factors, NRF-1, which regulate both carbohydrate and lipid metabolism. Obversely, changes in the NAD+/NADH ratios

affect the activity of other metabolic regulators such as the sirtuins which are protein deacetylases.[37] Sirtuins promote the activity of transcriptional co-activator, PGC-1 through deacetylation, the principal co-activator of NRF-1. Future research is necessary to establish the direct regulation of sirtuins on NRF-1 activity regarding carbohydrate and lipid metabolism. SARS-CoV-2 infection as indicated in numerous infections incites an inflammatory response that is generally limited to the respiratory system, but in excruciating events, a vast magnitude of the entire body is impaired.[38] Therefore, obesity is associated with chronic oxidative stress, that patterns towards dietary restriction and weight loss which result in marked decrease in oxidative stress indices within a short period.[39]

Strategic diet manipulation and therapeutics

This study provides a comprehensive analysis of the intricate relationships between SARS-CoV-2 infection, obesity, and T2DM. Its magnitude which enhances the understanding of the multifactorial impact of COVID-19 on metabolic health by successfully integrating discussions on genetic predispositions and dietary interventions, with sustained focus on the real-world implications of these interactions. This section discusses nutrient-gene interactions as particularly novel and aligns properly with contemporaneous precision medicine and personalised nutrition. Diabetic patients exhibit both very high and very low blood glucose levels which are associated with worse SARS-CoV-2 outcomes. Inadequately managed diabetes can obstruct both innate and adaptive immune responses, enhancing vulnerability to severe infection and sequelae. Additionally, hyperglycaemia exacerbates chronic inflammation, causing a pro-inflammatory ambient exploited by the virus. As stated supra[38,39], dietary approaches are pertinent in diabetes and obesity management. Pertinent dietary regulation with restricted carbohydrate diet have been expansively utilised, which diminished with novel therapeutic applications. The main goal is to mitigate dietary fat intake as to obviate atheriosclerotic disease risk, with reduced focus on carbohydrate quality and abundance. With increased global obesity and diabetes, the trend and target focus on the diet micronutrient composition. Low carbohydrate diets remain effective in initial weight dissipation and glycaemic control improvement.[40] Lowering saturated fat is ostensibly pertinent to diminish low-density lipoprotein, LD2 cholesterol and retarding of deranging effects of normal low carbohydrate diets. Enhanced dietary protein predisposes satiety, diminished energy intake and associated improved glycaemic homeostasis, but deficient in substandard improvement in glycaemic regulation or cardiovascular sequelae regarding the effect of weight loss. Type 1 diabetes response to regulatory effects with low carbohydrate diets, depicted no ketosis and hypoglycaemic sequelae. Carbohydrate-restricted diets show effective weight decrease as low fat diets; and fat surrogate for carbohydrate is invariably of benefit in cardiovascular disease risk without weight loss concern; and improves metabolic syndrome outcomes.[41] Low carbohydrate diets are influenced by glucose invariably via insulin as a pivotal regulatory component in gluconeogenesis, glycogen metabolism, lipogenesis and lipolysis.

The relative effects of fat compared to carbohydrate and the disparities among fatty acids and variants of carbohydrates on insulin resistance and associated risk factors for diabetes, cardiovascular disease and obesity[42] indicated that the fibre concentration of the carbohydrate diet ostensibly offers benefits in diabetic control, while lower cholesterol and postprandial blood glucose are associated with viscous fibres. Resting energy expenditure and both carbohydrate and lipid oxidation rates as well as rates detected in both fasting and parenterally fed states.[15] No differences in plasma insulin, glucose, and insulin resistance are observed in pre- and post-infliximab.[17] After anti-TNF-alpha therapy, Insulin resistance improvement occurred in inflammatory conditions in children, with no changes detected in adult patients with Chrons disease.[43]

Increasing evidence indicates that dietary plant polyphenols are potential nutraceuticals and supplementary therapeutic agents for type 2 diabetes due to their high content of phytochemicals and antioxidant potential.[44] In vitro animal models and human investigations revealed that plant-food polyphenols and polyphenol-rich products (i) ameliorate dyslipidaemia, hyperglycaemia and insulin resistance, (ii) enhance adipose tissue metabolism, (iii) attenuate oxidative stress and stress-sensitive signaling pathways and inflammatory responses[45], as well as (iv) modulate carbohydrate and lipid metabolism. Polyphenols may play a role obviate and diminish protracted diabetic complications, not restricted to cardiovascular disease, nephropathy and retinopathy.[46,47] Carbohydrates are inextricably-linked with immune ingredients at the molecular level. Invariably, carbohydrate-based systems inherently constitute attributes to counteract COVID-19.[48] Furthermore, carbohydrates demonstrate antifungal, antimicrobial and antiviral pertinence which equip them to morphologically confront SARS-COV-2 regarding vaccines and therapeutic agents.

DISCUSSION

The interdisciplinary potential of this work significantly bridges epidemiology, metabolic research, virology, and public health, with a substantial contribution to these disciplines. Furthermore, ethical preferences are inherently linked to healthcare resource allocation during pandemics, especially for those with underlying conditions. These merit future exploration to address the socio-economic variables which confound these health challenges and the global implications for health policy and equitable distribution of resources. Diabetes constitutes a metabolic disorder characterized by chronic hyperglycaemia and immune-mediated type 1 diabetes, insulin-resistant type 2 diabetes, gestational, genetic, environmental, infectious, behavioural or drug-induced perturbations.[12] Generally, obesity and type 2 diabetes prevalence accelerated globally

with concomitant sequelae in morbidity and mortality as well as comorbidities with obesity and SARS-CoV-2. Their pathophysiology[49,50] or pathobiology have been difficult to elucidate. Numerous hormone-like signaling agents which influence energy metabolism are elicited from adipocytes, such as leptin, including immature cells, such as tumour necrosis factor-alpha.

Obesity and type 2 diabetes have characteristic gene-environment interaction.[51] Adipose tissue in obesity discharges pro-inflammatory cytokines such as TNF-\(\alpha \), IL-6, and leptin, presenting a steady state of inflammation. Also, diabetes is an inflammatory disorder with high cytokine levels. Insulin resistance impairs glucose uptake in muscle, liver, and adipose tissue resulting in hyperglycaemia. Obese and diabetic persons frequently present anomalous lipid profiles characterized by high triglycerides and low HDL cholesterol. High blood glucose and inflammation can degrade internal vascular walls, thereby increasing blood clot risk. Excess fat can accumulate in tissues not designated for fat storage, such as the liver and pancreas, leading to aggravated organ dysfunctionality.[52-54] Deficient data pervade the single-gene defects which emanate in these conditions, but in multiple instances, polygenic contributions have been observed.[10,55] The devastating impacts of the COVID-19 pandemic on present and future human growth and development invariably depend on numerous factors including the socioeconomic correlates to ideologically behavioural imprint, macromolecular interactions, birth weight, obesity, diabetes, cardiovascular diseases, other communicable and noncommunicable diseases as indubitably evidenced in SARS-CoV-2 or COVID-19 degradations.[56-61] Thus, infections due to SARS-CoV-2 invariably causes aberrant alterations in carbohydrate, lipid, and amino acid metabolism, with severe impact on health outcomes. The viral shift from carbohydrate to lipid as the primordial energy reservoir undergirds and augments its replication. Metabolic changes offer the potential to predict disease prognosis, outcome and identify nascent therapeutic targets.[62,63] Contextually, the coronavirus disease 2019 (COVID-19) pandemic aetiologically influenced by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has become pertinent for metabolic research to undertake future exploration in viral infection processes and therapeutic regimen.[62-64]

CONCLUSION

This article provides a set of modalities for the analysis of carbohydrate-lipid interactions as extrapolated thematically for SARS-CoV-2, the aetiology of the COVID-19 pandemic. Carbohydrate-lipid dysfunctions in persons having obesity and diabetes combinatorially with SARS-CoV-2 infection creates a metabolic "perfect storm". This aggravates poor outcome and prognosis as well as increased morbidity and mortality. These individuals have prior dysfunctional metabolic processes, thus creating a susceptible avenue for the virus to increasingly degrade these critical pathways with resultant negative outcomes. The correlations and comorbidities between obesity and T2DM, the impacts of SARS-COV-2 on these conditions, and the impacts of carbohydrate-lipid interactions on these conditions are analysed to present data from specialized literature. There have been articulations of disparate theories to contribute to the adverse prognosis and outcomes in patients with diabetes, obesity and COVID-19 predicaments. SARS-CoV-2 infection disrupts carbohydrate, lipid, and amino acid metabolism, instigating and enhancing disease progression. Pre-existing obesity and diabetes, with their associated inflammation and insulin resistance, facilitate SARS-CoV-2 infection. The viral infection then intensifies this metabolic and inflammatory dysfunction. Elevated hyperglycaemia, inflammation, and insulin resistance establish an increasingly favourable environment for viral replication and disease progression, culminating in a poorer prognosis. Numerous environmental states including ageing ambience, lifestyle and social disruptions have been indicted in the predisposition of these disorders. Basically, a disequilibrium between caloric ingress and energy dissipation elicits endocrine and/or metabolic responses exacerbating the age-dependent dysregulation in lipid and carbohydrate metabolism in vulnerable populations and SARS-CoV-2 susceptible individuals. This review explores the prevalence, underlying aetiologies, clinical repercussions, and management strategies in persons with T2DM, focusing on its impact on glycaemic control, treatment safety, and correlated outcomes.

REFERENCES

1. Jiang Y, Xu L, Zheng X &Shi H. Recent advances in nutritional metabolism studies on SARS-CoV-2 infection. Infectious Medicine, 2025; 4(1): 100162. https://doi.org/10.1016/j.imj.2025.100162.
2. Chukwuma Sr C. Ecological analysis of the COVID-19 pandemic for restoration and sustainability. Research and Advances in Pharmacy and Life Sciences, 2021; 3(1).
3. Chukwuma Sr C. Accessing vaccines with compliance to stem the emergence of the SARS-COV-2 or COVID-19 variants. eBook: Research Trends of Microbiology. MedDocs Publishers LLC., 3(2): 12-15.
4. Chukwuma Sr C. Challenges and opportunities in accessing veritable vaccines and curbing the excesses of the SARS-COV-2 or COVID-19 variants. Middle East Publishers Scholars International Journal of Biochemistry, 2021; 4(9): 108-111. doi: 10.36348/sijb.2021v04i09.003.
5. Chukwuma Sr C. Syndemics of chronic and acute diseases in vulnerable populations, 2021. doi: 10.13140/RG.2.2.33288.14089.
6. Chukwuma Sr C. Challenges, Issues and Opportunities of Glp-1/Glp Receptor Agonists in Research and Patient Care. J Cur Tre Clin Case Rep., 6(3): 1-12.
7. Chukwuma Sr C. Pathophysiological Carbohydrate-Lipid Interactions in Covid-19, Obesity, and Diabetic Individuals. Medwin Publishers. Diabetes & Obesity

international Journal, 2022. DOI: 10.38125/OAJBS.000427.
8. Liu D, Zhang T, Wang Y and Xia L. The centrality of obesity in the course of severe COVID-19. Front Endocrinol (Lausanne), 2021; 12: 620566. doi: 10.3389/fendo.2021.620566.
9. Brown MA, Storlein LH, Huang X-F, et al. Dietary fat and carbohydrate composition: Metabolic diseases, Chapter 21. In: Montmayeur JP, le Coutre J, eds. Fat Detection, Task, Texture, and Post-ingestive Effects. Boca Raton (FC): CRC Press/Taylor & Francis, 2010.
10. Chukwuma Sr C. Health, risks and disease in therapeutic functionalities of Glucagon-like peptide-1 receptor agonists (GLP-1RAs) concerning human susceptibility. Grn Int J Apl Med Sci., Sep-Oct. 2025; 3(5): 240-254. DOI: 10.62046/gijams.2025.v03i05.002.
11. Chukwuma Sr C. Dynamic attributes of lipid and carbohydrate interactions in biosystems. Recent trends in pharmaceutical sciences and research, 2019; 2(1): 45-53.
12. Baynes HW. Classification, pathophysiology, diagnosis and management of diabetes mellitus. J Diabetes Metab., 2015; 6: 541.
13. Candib LM. Obesity and diabetes in vulnerable populations: reflection and proximal causes. Ann Fam Med., 2007; 5(6): 547-556.
14. Das UN, Rao AA. Gene expression profile in obesity and type 2 diabetes mellitus. Lipids in Health and Disease, 2007; 6: 35.
15. Kim JB. Dynamic cross talk, between metabolic organs in obesity and metabolic diseases. Exp Mol Med., 2016; 48: e214.
16. Chukwuma Sr C. Macromolecular interactions: Trends and Targets. Asian Journal of Microbiology and Biotechnology, 2016; 1(1): 7-13. https://mail.ikprress.org/index.php/AJMAB/article/view/11.
17. Hardie DG. Organismal carbohydrate and lipid homeostasis. Cold Spring Harbor Perspectives in Biology, 2012; doi: 10.1101/cshperspe.
18. Chukwuma Sr C. The complexity of communicable and non-communicable diseases in vulnerable populations. Advances in Bioengineering and Biomedical Science Research, 2020; 3(1). DOI:10.33140/ABBSR.03.01.01.
19. Chukwuma Sr C. Human hepatocytes response to pathological shifts in liver fibrosis. The Journal of Medical Research, 2024; 10(1): 37-41. DOI: 10.31254/jmr.2024.10108.
20. Stefan N, Birkenfeld AL and Schulze MB. Global pandemics interconnected - obesity, impaired metabolic health and COVID-19. Nature Reviews Endocrinology, 2021; 17: 135-149.
21. Alberti A, Schuelter-Trevisol F, User BPM, Traebert E, Freiberger V, Ventura L et all. Obesity in people with diabetes in COVID-19 times: Important considerations and precautions to be taken. World J Clin Cases, 2021; 9(20): 5358-5357.
22. Stunkard AJ, Harris JR, Pedersen NL, McLearn GE. The body mass index of twins who have been reared apart. N Engl J Med., 1990; 332: 1483-1487.
23. Bouchard C, Tremblay A. Genetic influences on the response of body fat distribution to positive and negative energy balances in human identical twins. Nutr., 1997; 127: 943S-947S.
24. Chukwuma Sr C. Molecular Mechanisms Which Regulate Skeletal Muscle Mass Against Obesity and The Clinicopathological Correlates. International Journal of Medical Case Reports and Medical Research, 2024; 3(1); DOI: 10.61148/2994-6905/IJMCRMR/043.
25. Kopelman PG. Obesity as a medical problem. Nature, 2000; 404: 635-643.
26. Harris MI. Diabetes in America: epidemiology and scope of the problem. Diabetes Care, 1998; 21(3): C11-C14.
27. Singla P, Bardoloi A, Parkash AA. Metabolic effects of obesity: A review. World J Diabetes, 2010; 1(3): 76-88.
28. Olshansky SJ, Passaro DJ, Hershow et al. A potential decline in life expectancy in the United States in the 21st century. N Engl J Med., 2005; 352: 1138-1145.
29. Silva MA, Alves da Silva ARP, Abrantes do Amaral M, Fragas MG and Canada NOS. Metabolic alterations in SARS-COV-2 infection and its implication in kidney dysfunction. Front Physiology, 2021. doi: 10.3389/fphys.2021.624698.
30. Randle PJ. Regulatory interactions between lipids and carbohydrates: the glucose fatty acid cycle after 35 years. Diab Metab Res Rev., 1998; 14(4): 263-283.
31. Dulloo AG, Gubler M, Montani JP, et al. Substrate cycling between de novo lipogenesis, and lipid oxidation: a thermogenic mechanism against skeletal muscle lipotoxicity and glucolipotoxicity. Proceedings of the Second Fribourg Obesity Research Conference (FORC-2003). Int J Obesity, 2004; 28: S29-S37.
32. Kelley DE, Mokan M, Simoneau JA, Mandarino LJ. Interaction between glucose and free fatty acid metabolism in human skeletal muscle. J Clin Invest, 1993; 92: 91-98.
33. Frayn KN. The glucose-fatty acid cycle: a physiologic perspective. Biochem Soc Trans, 2003; 31: 1115-1119.
34. Ferrannini E, Barrett EJ, Bevilacqua S, DeFronzo RA. Effects of fatty acids on glucose production and utilization in man. J Clin Invest, 1983; 72: 1737-1747.
35. Golay A, Felber JP, Meyer HU et al. Study on lipid metabolism in obesity diabetes. Metabolism, 1984; 33(2): 111-116.
36. Chukwuma Sr C. Pathophysiologic Functions of Carbohydrate-Lipid Interactions: Ascertainment in Obesity, Diabetes and SARS-CoV-2/COVID-19. Journal of Biotechnology and Its Applications. SciVision Publishers LLC., 2022. doi:10.33425/2771-9014.1005.
37. Ferreira Z, Ayeleso A, Mukwevh OE. Control of carbohydrate and lipid metabolism by NRF-1 and sirtuins: Implications on type 2 diabetes and obesity. Chem Biol Lett., 2014; 1(2).

38. Dandona P and Ghanim H. Diabetes, obesity, COVID-19, and other anti diabetes drugs. Diabetes Care., 2021; 44: 1929-1933. doi: 10.2337/dci21-0003.
39. Chukwuma Sr C. Regulatory and metabolic interactions of carbohydrates and lipids in diabetes and obesity. J Biotech Res., 2019; 5(11): 123-127. doi.org/10.32861/jbr/511.123.127.
40. Chukwuma Sr C. Dynamic attributes of lipid and carbohydrate interactions in biosystems. Recent trends in pharmaceutical sciences and research, 2019; 2(1): 45-53. https://doi.org/10.5281/zenodo.3595688.
41. Beinstein Dr. Dietary carbohydrate restriction in type 2 diabetes and metabolic syndrome: time for a critical appraisal. Nutrition and Metabolism, 2008. http://www.diabetes.book.com/dietary-carbohydrate-restriction/.
42. Hung T, Sievenpiper JL, Marchie A, et al. Fat versus carbohydrate in insulin resistance, obesity, diabetes and cardiovascular disease. Curr OpinClin Nutr Metab Care, 2003; 6(2): 165-176.
43. Steiner SJ, Pfefferkorn MD, Fitzgerald JF & Denne SC. Carbohydrate and lipid metabolism following infliximab therapy in pediatric Chrons Disease. Pediatric Research, 2008; 64: 673-676.
44. Bahadoran Z, Mirmiran P, Azizi F. Dietary polyphenols as potential nutraceuticals in management of diabetes: a review. J Diab Metab Disorders, 2013; 12: 43.
45. Evans JL, Goldfine, ID, Maddux BA, Grodsky GM. Oxidative stress and stress-activated signaling pathways: a unifying hypothesis of type 2 diabetes. Endocr Rev., 2002; 23: 599-622.
46. Pandey KB, Rizvi SI. Plant polyphenols as dietary antioxidants in human health and disease. Oxid Med Cell Longev, 2009; 2: 270-278.
47. Bahadoran Z, Golzarand M, Mirmiran P, Saadati N, Azizi F. The association of dietary phytochemical index and cardiometabolic risk factors in adults: Tehran lipid and glucose study. Nutri Metab (Lond), 2012; 9: 70.
48. Kumbhar PS, Pandya AK, Manjappa AS et al. Carbohydrate-based diagnosis, prophylaxis and treatment of infectious diseases: Special emphasis on COVID-19. Carbohydrate Polymer Technologies and Applications, 2021; 2. doi: 10.1016/j.carpta.2021.100052.
49. Chukwuma Sr C. The complex interplay in the regulation of cardiac pathophysiologic functionalities by protein kinases and phosphatases. Jjournal of Cardiology & Cardiovascular Medicine. JCCM., 2021; 6: 048-054. doi: 10.29328/journal.jccm.1001118.
50. Chukwuma Sr C. Exploring the complex interplay in the regulation of cardiac pathophysiological functions by protein kinases and phosphatases. International Journal of Biosciences and Biochemistry, 2021; 3(2): 1-7.
51. Chukwuma Sr C. Is Diabetes a Model for Gene-environment Interaction in Premature Senescence? Journal of Biology, Agriculture & Healthcare, 2014; 4: 25. http://www.iiste.org/Journals/index.php/JBAH/article/view/17380.
52. Morais AH de A, Passos TS, de Lima Vale SH, da Silva Maia JK, Maciel BLL. Obesity and the increased risk for COVID-19: mechanisms and nutritional management. Nutrition Research Reviews, 2021; 34(2): 209-221. doi:10.1017/S095442242000027X.
53. Sabri S, Bourron O, Phan F, Nguyen LS. Interactions between diabetes and COVID-19: A narrative review. World J Diabetes, 2021; 12(10): 1674-1692 [PMID: 34754370 DOI: 10.4239/wjd.v12.i10.1674.
54. Jin X, Qiu T, Li L, Yu R, Chen X, Li C, Proud CG, Jiang T. Pathophysiology of obesity and its associated diseases. Acta Pharmaceutica Sinica B., 2023; 13(6): 2403-2424. https://doi.org/10.1016/j.apsb.2023.01.012.
55. Romao I, Roth J. Genetic and environmental interactions in obesity and type 2 diabetes. J Am Dietetic Assoc, 2008; 108(4): S24-S28.
56. Chukwuma C Sr. Etiopathophysiological Functions of Carbohydrate-Lipid Interactions Associated with Obesity, Diabetes and Covid-19. Diabetes & Obesity International Journal. Medwin Publishers, 2022. doi:10.23880/doij-16000252.
57. Bogin B and Varea C. COVID-19, crisis, and emotional stress: A biocultural perspective of their impact on growth and development for the next generation. Am J Him Biol., 2020. doi: 10.1002/ajhb.23474.
58. Yaribeygi H, Sathyapalan T, Jamialahmadi T and Sahebkar A. The impact of diabetes mellitus in COVID-19: A mechanistic review of molecular interactions. Journal of Diabetes Research, 2020. doi: 10.1155/2020/5436832.
59. Chukwuma Sr C. Probing research precincts focused on systematic determination of the biological effects of macromolecular interactions. International Journal of Applied Science and Engineering Review, 2020; 1(6).
60. Chukwuma Sr C. Convergence on the constraints and challenges in the awareness, prevention, treatment and control of type 2 diabetes and related conditions. Global Journal of Medical Research, GJMR, 2018; 17(3): version 1.0.
61. Chukwuma Sr C. Bioinformatics-base and determinants in the spatiotemporal variations of emerging and reemerging infectious diseases - Journal of Infectious Diseases and Preventive Medicine. Journal of Ancient Diseases & Preventive Remedies, 2018; 06(02). DOI:10.4172/2329.8731.1000182.
62. Jiang Y, Xu L, Zheng X, Shi H. Recent advances in nutritional metabolism studies on SARS-CoV-2 infection. Infectious Medicine, 2025; 4(1): 100162. https://doi.org/10.1016/j.imj.2025.100162.
63. Li L, Li L, Cai X, Pan Z. New Insights into the Effects of SARS-CoV-2 on Metabolic Organs: A Narrative Review of COVID-19 Induced Diabetes.Diabetes, Metabolic Syndrome and Obesity. 2024. https://doi.org/10.2147/dmso.s454408.
64. Chukwuma Sr C. Approaches in diverse issues of glucagon-like peptide-1 receptor agonists (GLP-1 RAs) in global health strategy, economic burden, geopolitical

implications and gain-of-function research. World Journal of Pharmaceutical and Healthcare Research, 2025; 2(11): 1–6. https://doi.org/10.5281/zenodo.17510578.