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Review Article

ECOLOGICAL ANALYSIS OF THE ADVANCES IN COMPOSITE INTERDISCIPLINARY RESEARCH IN UNDERSTANDING SHARED RECIPROCAL INTERPLAY AND COMPLEXITIES BETWEEN AGEING, DIABETES, (RE)EMERGING DISORDERS, CLINICAL IMPLICATIONS AND THERAPEUTIC MODALITIES FOR THE FUTURE

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ABSTRACT

Recent advances in integrative research have preeminently emphasized on the tri-directional, reciprocal interplay between ageing, diabetes mellitus (DM), and (re)emerging infectious diseases. Pivotal results earmark that diabetes functions as a significant predisposing factor for severe infection, especially among the elderly population, emanating from immune system senescence and chronic inflammation, whereas infections on their own are capable of exacerbating metabolic dysfunction. We live in a challenging era, highlighted by strife and wars, political pressure, deadly diseases, inchoate sustainable governance and systems. Generally, focus on a specific research discipline or country, may not be considered appropriate for publication and global consumption, thus researchers ought to think locally and act globally. Diverse research across disciplines has enriched understanding of inter alia intricately complex geopolitical and socioeconomic spheres, health and environmental challenges, opportunities and priorities. Epidemiology involves the study, analysis and understanding of the dissemination, patterns, and determinants of health and disease states in specifically distinct populations. Research into the various dynamics of disease emergence and reemergence frequently focuses on the identification of key driving parameters which elevate outbreak risk. The primary drivers for emergence, reemergence and distribution inculcate human-animal interactions (zoonoses). The incessant frequency and interaction between humans and wildlife, frequently driven by agricultural expansive diversification and habitat encroachment, grants the latitude for pathogens to jump species. Due to climate change, the anomalous altering of global temperatures and weather trajectories expand the geographic magnitude of disease vectors mosquitoes which introduce pathogenic organisms to nascent precincts. In accelerated global mobility, air travel and trade expose the dissemination of infectious diseases rapidly across regions, from local outbreaks to global pandemics. Research abound on the primordial mechanisms of ageing, the process of cellular senescence, and lifespan, expansively. Research goals prevail to understand the clinicopathological correlates of diabetes, enhancing diagnostic techniques, therapeutic strategies, and the prevention of sequelae inextricably linked to the disease. Invariably, investigations are focused on the dynamics of extant, emerging and reemerging infectious diseases, tracking and defining transmission patterns and trajectories, new vaccine development, and enhanced preparedness for future pandemics. These incessant research inputs constrict the lacunae between basic biology and clinical medicine, culminating in enhanced health and quality of life. Public health strategies are associated with research and concern on transmission and dissemination dynamics which direct critical policies for outbreak prevention, preparedness, abatement, control, with pertinent strategic surveillance systems, contact tracing, and vaccination programmes. These strategies function in synergy to ameliorate or obviate disease dissemination by the provisions of detailed information and guidance through domestic and international organisations on the implementation of these measures effectively. This research review tends to enhance predictive stances by providing academia, health organizations and the general public for better anticipation and preparedness for future disease outbreaks.

KEYWORDS: Climate change, artificial intelligence, sarcopenia, gene-environment interaction, SARS-CoV-2/COVID-19, Glp-1/Glp Receptor Agonists, geopolitics, gain-of-function research.

INTRODUCTION

Research has significantly advanced understanding of the clinicopathological correlates of ageing, diabetes mellitus, and the dynamics of extant, emerging and reemerging infectious disorders through the identification of shared molecular processes, advancing diagnostic tools, and pioneering new therapeutic modalities.[1-3]] Diabetes and ageing correlate with identical organ and system perturbations which are enhanced by concomitant molecular processes such as cellular senescence. Age represents a major risk factor for type 2 diabetes mellitus. It is unclear how senescence contributes to diabetes pathogenesis. Thus, available treatment modalities have not targeted the vital area of the disease Chukwuma.[1,2] A recent review article[4] citing the following articles[5-8] enunciating that ageing is characterized by the age-dependent depreciation of physiological functionality that invariably tends to mortality vulnerability. The declination mechanism is evident in life and characterises a significant aspect of several disorders, such as obesity, type 2 diabetes, Alzheimers and cardiac aberrations. Insulin functionality impairment in the regulation of metabolism, development, cell division and differentiation has been linked with numerous age-related anomalies. Insulin is mainly regulated by dual processes involving release and clearance of the hormone from blood and tissue sensitivity. Ageing deranges these mechanisms, leading to insulin dysfunctional presentations, and elevating disease and mortality incidence. The activity of Insulin during the ageing process is significant in contributing to age-related diseases. Thus, research in improving insulin functionality may enhance effectiveness in advancing longevity and quality of life. On the whole, research across disciplines inculcates and emphasizes that diverse conditions are not restricted but constitute aspects of broader and intricately complex, inextricably-linked pathological landscapes, dependent on integrative strategies for prevention, diagnosis, and therapy. This article delineates current information on the inextricably-linked processes which underly ageing-related disorders, such as type 2 diabetes, obesity, sarcopenia, cardiovascular diseases and neurodegenerative disorders. The review explores the critical role of contributing factors including microbiome dysbiosis, sex variations, and modulating disease progression. Progress and improvement in multi-omics technologies and epigenetic clocks are highlighted regarding their precise quantification of biological ageing and stratification of disease risk. A discourse of the therapeutic potential and advances in clinical trials of targeting the shared processes with senolytics, for instance, provide a paradigm shift from disease-specific therapy to holistic interventions patterned to extend healthspan and longevity.[9] Research stipulates that dealing with the intersection of diverse conditions necessitates a holistic "one-health" strategy that inculcates metabolic management and immune system resilience to enhance outcomes in ageing populations. Recent progress in research, specifically addressed during the COVID-19 pandemic era exposed an intricately complex, bidirectional, and reciprocal interplay between ageing, diabetes, particularly type 2 diabetes, and (re)emerging infectious diseases. The concurrent convergence of this "three-way association", rapidly induces metabolic deterioration, debilitates immune defenses, and accelerates vulnerability to adverse morbidity and to a certain extent, mortality.

Perspectives and reflections in ageing research

Gerontology research has gone past descriptive investigations towards the molecular and cellular mechanisms of ageing, for instance, mitochondrial dysfunction, cellular senescence, and chronic low-grade inflammation or inflammageing, with resultant determination of numerous pivotal hallmarks. The mechanistic explication is to develope targeted interventions or geroprotectors for enhanced healthspan and potential lifespan. The reflection and perspective of ageing is the encompassing organism genotype presentation and gene interaction with the internal and external environment.[7] Ageing depicts a risk of evolving age-related disruptions, such as diabetes, cardiovascular disorders, cancer, and neurological degenerative disorders, with eventual resultant mortality. Type 2 diabetes is inextricably linked to both environmental and genetic components and aetiologies, which a simple ageing biological model is well-nigh impossible to explicate due to expansive organismic intricate complexity. That sort of model may be important as a trajectory for observing both the clinical and theoretical strategies as pathways to elucidate the mechanisms of gene-environment interaction for diabetes and early onset senescence. Ageing is characterised as biological and chronological age, with the latter being a fixed dimension based on the calendar, whereas the former depicts inter-individual growth and ageing variations.[10] Biological gerontology focuses on the parameters of average ageing, whereas little attention has been paid to the individual variations regarding the rates of ageing. The objective of ageing research is the stemming or retardation of the deleterious physical changes in old age. Normal ageing depicts usual ageing whereby the extrinsic factors merely exhibit the impact of ageing; and whereas a neutral or positive role is displayed in successful ageing with extrinsic factors. Age-related disorders occur rapidly or start sooner in unsuccessful ageing than in successful ageing. The clinical presentations of these disorders are increasingly elevated and manifested at a youthful (chronological) age, culminating in early onset morbidity and mortality.[6] Therefore, unsuccessful ageing pertains to the process of premature ageing or accelerated ageing, with restriction to a singular organ system, isolated progression in certain organ systems, or uniform procession encompassing the whole body. The main heterogeneity in age groups has either suffered neglect or linked with disparities in genetic factors. This perspective disregards the vital aspect of intrinsic factors, psychosocial and physiologic variables interact, including the heterogeneity within disparate age groups. Individuals ageing successfully tend to manifest slight or no age debilitating physiologic functionality, whereas those with usual ageing

present with disease-associated degradations depicted as the impacts of ageing. The distinct significant association between diabetes and disease-related anomalies are observed as the impacts of ageing, whereas the association between diabetes and unsuccessful ageing distinctively reflects the higher prevalence of varied sequelae within diabetic patients.[7]

Significant progress in unravelling the molecular, cellular, and supracellular processes which underly ageing has occurred. This spurred geroscience inception, with goals to decipher functional hallmarks of ageing. Ageing is perceived as a process enhanced by gerogene overactivation, explicated as genes and molecular pathways which correlate with biological ageing, and alternatively retarded by gerosuppressors, just as cancers result from the activation of oncogenes and inhibited by tumour suppressors. These gerogenes and gerosuppressors are usually associated with age-related disorders in human population research, also as targets for modelling age-related disorders in animals and preventing or treating the disorders in humans. Gerogenes and gerosuppressors are in interactions with environmental, psychological and behavioural risk factors for the identification of the heterogeneous pathways of biological ageing and disease presentation. Nascent molecular profiling technologies enhance the features of gerogenic and gerosuppressive trajectories, which depict as ageing biomarkers, thereby enacting the precision geromedicine era. Combined with findings from randomized clinical trials and regulatory accent, gerotherapeutics will be patterned to every individual depending on genetic profiles, high-dimensional omics-dependent ageing biomarkers, clinical and digital biomarkers of ageing, psychosocial profile, and erstwhile or current exposures.[11] Biological ageing is a complex, multifactorial mechanism with characteristic progressive dissipation of cellular homeostasis, directed by intersecting pathways of inflammageing, immune senescence, mitochondrial dysfunctionality, and genomic disruptions.[12]

Numerous human diseases or disorders are invariably linked with the complex gene functionalities and regulations. These are also connected in the environmental signal integration for cells in the modulation of the functional genome output. The perspective of the ageing process is a reflection of the entire manifestation of an organism and gene-environment interaction. Ageing occurs due to a plethora of processes which inculcate diabetes, immune system deterioration, oxidative degradation, apoptosis rates, and telomere shortening. The detection of reduced telomere lengths frequently depict rapid biological ageing, particularly in those presenting with hypertension, diabetes mellitus, and glucose intolerance disparately or comorbidity. However, adverse social factors may contribute to a worse prognosis or outcome for diabetes in early ageing. It is deduced that diabetes is inextricably linked with "premature ageing", with perceptible variation in the progression rate of ageing in humans. Thus, a simple biological model may not suffice to depict both the clinical and theoretical spheres in order to explicate or elucidate the gene-environment interaction mechanisms linking diabetes mellitus and premature ageing. The potential clinical and gerontological implications are ascertainment of the population at risk for premature ageing development among diabetic patients as to enact prompt therapeutic strategies and reduce pecuniary costs and obviate precarious sequelae.[7]

Biomarker development in ageing

Robust biomarkers identification, for instance, DNA methylation patterns ("epigenetic clocks"), a provides the latitude to quantitatively assess and predict biological age, but not chronological age.[7] DNA methylation-based epigenetic clocks are potent biomarkers which quantify biological age, predict healthspan and disease risk beyond chronological age via detecting rapid or retarded ageing patterns, providing insights for individualized health interventions. These "clocks" employ machine learning for the analysis of methylation extent at specific DNA sites or CpGs for the estimation of the biological age of an individual, capable of unravelling the underlying health conditions and magnitude for age-related states. Due to its intricately complex trajectories, ageing is characterized by diverse alterations at the cellular, subcellular and nuclear dimensions, such as epigenetic ageing. Due to the the critical role of epigenetic alternations in ageing, DNA methylation patterns are being applied for determination of biological age, presently ascribed to as the epigenetic clock. In this regard, the epigenetic clock constitutes a biomarker of ageing and a resourceful tool to evaluate, monitor and manage healthy ageing. This burgeoning scientific discipline attracts diverse emerging epigenetic clocks, such as Horvath and Hannum, DNA PhenoAge and DNA GrimAge. Epigenetic age correlations with morbidity and mortality, for instance, are suggestive of the potential for epigenetic clocks to predict and identify risk contextually in ageing. There are extant studies on advances in age-reversing interventions with the application of epigenetic clocks as practical tools in efficacy monitoring and evaluation. However, paucity of higher-quality information constitutes an aggravating issue that must be addressed. For the future, pertinent reasonable understanding of epigenetic clocks may potentiate novel insights into the ageing mechanism in order to manipulate and promote healthy ageing.[13]

Research on the role of epigenetics, especially in human development, health, diseases such as ageing, muscle disorders, and infections, as well as the impact of the environmental on gene expression, frequently associating molecular processes to wider health outcomes and evolutionary biology, investigate factors beyond DNA, such as diet and pollutants, drive gene activity and disease vulnerability, advancing theories on transgenerational health and therapeutic strategies. The works at the Chrysanthus Centre for Future-Oriented Studies.[14] emphasize understanding of these mechanisms for future

health solutions and preventative tendencies and strategies, as well as on the molecular basis of disease, epigenetics, and the interplay between genetics, environment, and health. In essence, Chukwuma Sr C employs an epigenetic lens to understand how the experiences in life and the ambient can "scribble" on our genes, impacting on health from conception through life and generations of the future.[15,16]

Ageing exacerbates insulin resistance

Ageing fuels type 2 diabetes risk via elevated insulin resistance due to gain of body fat, muscle dissipation per sarcopenia in obesity[17], chronic inflammation, and cellular dysfunctionality (mitochondrial issues, oxidative stress), suppressing the ability of cells to utilise glucose, as the pancreas, particularly exceeding age 45, is in dire straits to compensate, imposing a profound storm for high blood sugar. Alterations in body composition manifest due to elevated fat (particularly visceral) and reduced muscle mass, resulting in inadequate glucose utilisation. This is because skeletal muscle is the primary site of postprandial glucose uptake, and reduced mass, combined with the inflammatory effects and insulin resistance associated with excess visceral fat, impairs the body's ability to clear glucose from the bloodstream efficiently.[18] Skeletal muscle dysfunction presents as ageing muscle develops mitochondrial derangement, inflammation, and fat accretion, deteriorating insulin signaling.[19] Cellular and molecular stress manifest due to Increased oxidative stress per reactive oxygen species and inflammation associated with cytokines such as IL-6, TNF-α distort insulin trajectories.[20] Pancreatic beta-cell decline is associated with age, beta cells producing insulin become deficient in effectivity, and vulnerable to mortality, and failure to correspond with the augmented demand due to resistance.[5] Inflammageing results due to chronic, low-grade inflammation associated with aging directly aggravates insulin resistance.[22] Physical inactivity or lack of exercise in older adults who frequently are sedentary, causing decrement in the protective impacts of ambulatory states.[23] Also, metabolic alterations in ageing impact hormones and enzymes with glucose regulation, such as GLP-1.[24,25] In essence, ageing exhibits a metabolic milieu wherein tissues inadequately respond to insulin (resistance), and the pancreas is incapacitated, thus, culminating in hyperglycaemia and type 2 diabetes.[26]

Diabetes mellitus and the pivotal spheres of advancement highlights the intersection

Current research stipulates that diabetes is a complex and multisystemic disorder that profoundly accelerates the biological ageing process encompassing diverse organs, spurred by inextricably-linked molecular processes, such as insulin resistance and chronic inflammation.[27,28] Progress in diabetes research highlights its complex, multisystemic features and the intersection with ageing. Advances in diabetes research have surpassed merely as a blood sugar anomalous presentation but as a complex, multisystemic state significantly intertwined with the ageing mechanism. Conventionally, AGE levels have been most proximally associated with diabetes and its sequelae, such as CKD and CVDs, as their severity positively correlate with the accumulation of AGE, due to persistent hyperglycaemic ambient in diabetic patients that normally assists AGE production. With advancements in AGE research, there have been emergence of new theories, indicating that AGEs are not just byproducts of diabetes but also involved in its development.[29]

Cellular Senescence: Research shows that both diabetes and ageing accelerate cellular senescence, whereby cells terminate dividing but neglect to die, this, aggregating in tissues, contributing to inflammation and organ dysfunction.[7] This produces a vicious cycle, as cellular senescence in adipose tissue and the pancreas, increased insulin resistance and impairment of beta-cell functionality.

Inflammageing: Chronic, low-grade inflammation or "inflammageing" constitutes a feature of both ageing and diabetes.[1,2] At a molecular level, diabetic persons usually present with premature ageing, characterized by elevated inflammatory markers which enhance long-run sequelae, such as cardiovascular and renal deterioration.

Mitochondrial Dysfunction: A retardation in mitochondrial as powerhouse of the cell functionality is observed in both ageing and type 2 diabetes. Research explores how mitochondrial dysfunction assists in dysfunctional glucose metabolism central to type 2 diabetes, as well as the overall decline observed in ageing.[1,2]

Epigenetic Modifications: In both ageing and diabetes, modifications are induced in gene expression without changes in the underlying DNA sequence. These epigenetic shifts explicate the contribution of lifestyle, environmental factors, and spatiotemporal variations contribute to disease development and progression, depicting a shared molecular basis.[15,16,30] Thus, there are pertinent needs for integrated therapeutic modalities to address both metabolic health and the underlying biological ageing. Future therapeutic strategies may target conventional pathways for the prevention or treatment of expansive age-related disorders associated with diabetes.[7,27]

Shared Pathophysiological Mechanisms with Ageing: Research depicts that ageing constitutes a substantial risk factor for type 2 diabetes, with shared processes, such as oxidative stress, deranged insulin formation resulting from beta-cell senescence, and advanced glycation end products (AGEs) aggregation. Also, diabetes, on its own, is capable of accelerating ageing in numerous organs, characteristic of type 2 diabetes, a metabolic disease presenting chronic high blood sugar concentrations and insulin resistance, with diabetes contributing to the the ageing process despite age, citing it an age-related ageing disorder. The oxidative stress attributed to chronic high blood sugar and IR may result in dysfunctional mitochondria, that enhances distortions in epigenetic regulation, telomere shortening,

and cellular senescence. Research is pertinent in the synthesis of epidemiological and clinical concerns on ageing within diverse organs, with focus on insulin resistance and oxidative stress as primordial processes, as well as diabetes-specific ageing precincts, such as toxicity of glucose, advanced glycation end-products (AGEs), immunoinflammatory ageing, and protein amyloidosis, as integrated in the "metabolism-inflammation-ageing" network, including interventions targeting diabetes ageing[31], and as an accelerating public health dilemma[32], and as a model evidenced by direct correlation between diabetes, telomere shortening, and epigenetic ageing.[33-35]

(Re)emerging infectious diseases and climate change issues

Global climate change and accelerated mobility are pronounced drivers of expansive infectious diseases dissemination, especially by the mosquito vector. Universally, rising temperatures and changes in weather patterns spur new habitats for disease vectors, as accelerated air travel and trade enhance fast pathogen distribution.[36] This work focuses on human health to understand management of the global predicament related to the determinants and impact of susceptibilities to extreme hydrologic events and climate change without risk reduction and the accompanying anomalies. The major effective strategy for risk reduction associated with biodiversity, environmental, and health susceptibilities from climate change and extreme hydrological events, should inculcate an ecological framework that recognises exposure, vulnerability, and resilience. Research focus has been on the preeminence of understanding the correlations between ecosystems and human communities on exposure and vulnerability to hazards, hazard sensitivity, and coping, adaptive and recuperative capacity. Risk reduction inculcates structurally mitigating exposure, undergirding resilience, and enhancing sustenance and vulnerability management. Extreme weather- and climate-related events impact on human health with resultant morbidity, mortality and socioeconomic constraints and priorities. Climate change and extreme hydrologic events have varied the frequency, intensity, geographic pattern, and pertinence as drivers for future sustainability and change. The implicated susceptible variables encompass hydrologic events, such as precipitation, floods and droughts as well as heat waves, wildfires, global warming, extreme temperatures, and hurricanes. The trajectories associated with extreme events to economic development, human health prognosis and outcomes are invariably and inexplicably varied and complex; and therefore, impervious to easy prediction as a result of their emergence and reemergence from local, global and environmental factors which impact disease burden.[37,38]

Research and publication pose progressive perspectives and initiatives in the governance of local and global health as disease is invariably a geopolitical issue and concern associated with gain-of-function research whereby health diplomacy must be modelled within the ecological framework in present and future global emergence and reemergence of infectious diseases. These have spurred reactions within the spheres in extreme geopolitics and gain-of-function research, natural and anthropogenic activities. Geopolitical concerns and gain-of-function challenges and priorities influence the social determinants of health and vice versa. The unprecedented convergence of countries for an epidemic or pandemic treaty settings or other formulations to harness or curb emerging and reemerging infectious diseases provides appreciable opportunities and challenges in action, preparedness and response. It is pertinent to foster legal instruments, effective and efficient platforms to prevent future infectious disease threats and outbreaks.[39]

Emerging and reemerging infectious diseases including parasitoses are expansively distributed in flora and fauna, and cause pronounced morbidity and mortality. Modifications in environmental states due to anthropogenic factors usually lead to microbial or viral traffic. Recently, there have been progress in medical research and therapeutic regimen but infectious diseases have ranked alongside wars and famine as principal issues inhibiting human development, progress and survival; and constitute major aetiologies of global morbidity and mortality. It is pertinent to take into cognizance that actions and policies taken towards countries, policies, agricultural subsidies, and geopolitics may have untoward repercussions on global health. The prevention or mitigation of the effect of vector-borne disease agents through the identification of peculiar features which augment their evolutionary capacity, emergence, reemergence and dissemination. Most of these are a varied group of diseases and infections which are the neglected diseases made up of a medically diverse group of tropical infections, strictly endemic in low income communities in developing regions of Africa, Asia, and the Americas. These necessitate the research of systems of increasing complexity via the application of models. National and world governments must not shirk their responsibilities to respond and fully inform their constituencies of the dire consequences of not facing their obligations because the constituencies are frequently incapacitated to grasp the nature and repercussions of parasitoses, emerging and reemerging infectious diseases, and, may be incapable to imbibe the magnitude of change needed to confront them.[40] The dynamics of parasitoses, emerging and reemerging infectious diseases[41-44] have been properly explicated through interdisciplinary presentations, collaborations and technological innovations.

Explicating Zoonotic Origin: Research has identified, determined and established that a vast majority of emerging and reemerging infections, such as COVID-19 and Ebola[45] have zoonotic origins, propelled by factors, such as climate change, land use change, and augmented human-wildlife interaction. The significance of the "One Health" strategy, integrating human, animal, and environmental health come to the fore. The originating of zoonotic diseases in animals present a formidable threat to global

public health. The coronavirus disease 2019 (COVID-19) pandemic caused expansive morbidity, mortality, and socioeconomic debilitating incidents globally. In order to survive these diseases effectively, the strengthening of surveillance and establishing of rapid response systems become pertinent.[46] Research has to examine modern technologies and solutions which can enhance zoonotic disease surveillance, as well as outbreak responses, with valuable cutting-edge innovations which can be leveraged to prevent, detect, and regulate emerging and reemerging zoonotic disease outbreaks. These will make provisions for discourse on advanced tools, big data analytics, artificial intelligence, the Internet of Things, geographic information systems[47], early warning systems, remote sensing, molecular diagnostics, point-of-care testing, telemedicine, and digital contact tracing. These technologies provide real-time monitoring, outbreak risk prediction, early aberration detection, accelerated diagnosis, and outbreak targeted interventions. Collaborative partnerships, integrate and enhance these strategies to significantly accelerate and improve the effectiveness of zoonotic disease control. Numerous challenges are bound to persist, especially in resource-restricted settings, like infrastructure deficiencies, inequitable distribution and availability of resources, inadequate data integration and training, and poor ethical evaluation, implementation and monitoring.[47] A combination of strategic planning and coordination as well as modern technological innovations and solutions can potentiate and spur responsiveness in surveillance and outbreaks, as well as obviate emerging zoonotic disease threats.[48] and devastations due to reemerging comorbidities globally.[49]

Global Surveillance and Modelling: The applications of AI, machine learning, and real-time data from social media and global travel patterns have enhanced the timeliness and accuracy of prediction models, such as the Hawkes model, and detection of outbreaks of diseases. Disease outbreaks cause significant burdens on public health systems, frequently necessitating accelerated response modalities to ameliorate expansive health and economic impacts. Although, conventional modalities of outbreak prediction and surveillance could be effective, there may be frequent deficient capacity for processing and analysing of the vast array of heterogeneous data generated in current healthcare ecosystems. In this sphere, machine learning (ML) provides transformative potential, thus leveraging its ability for processing large datasets, identifying complex patterns, and providing real-time insights. Integration of various data sources, such as electronic health records (EHRs), genomic sequences, social media feeds, and climate data, ML algorithms are able with unprecedented convergence and accuracy predict disease outbreaks. For instance, supervised learning models have been employed successfully for the forecasting of spatiotemporal variations in influenza trends, whereas unregulated clustering techniques have been available to detect aberrations suggesting emerging infectious diseases. Furthermore, ML enhances advanced public health surveillance by automating data processing pipelines, facilitates real-time monitoring potentialities, and undergirding resource optimization for outbreak responses. Irrespective of these advances, the employment of ML in public health surveillance is not without its predicaments associated with data privacy, algorithm interpretability, ethical considerations, and integration with extant public health infrastructures and priorities. A multidimensional strategy[50], encompassing frameworks of robust data governance, advanced algorithm transparency, and collaborative partnerships incorporating technology enhancers and public health interests. The vital role of ML in the transformation of public health surveillance, addressing its usage in predicting disease outbreak, underscores the preeminence of incessant innovation, regulatory support, and ethics in progressive MLdriven positive outcomes for global health security and geopolitics.[51,52]

At an accelerated response, Artificial intelligence (AI) is rapidly restrategising public health by facilitating rapid, precise and scalable stances. With the progression of the global burden of communicable and non-communicable diseases[53], the prowess of AI in the expansive and multitudinous analyses of a vast majority of datasets, pattern identification and recognition, as well as production of practical insights validates it as a veritable asset to enhance global health outcomes with future directions for encompassing synthesis of AI applications in public health, enabling technologies, pivotal benefits, and extant challenges in order to guide researchers, practitioners, and policymakers to leverage AI for equitable and effective public health modalities.[54, 55]

Therapeutic modalities enacted from advancing research (Key interventions explored)

Novel Therapies: Despite available conventional therapies[56], researchers are exploring newfangled pharmacological modalities and agents, such as dual incretin receptor agonists and even gene-editing technologies to target immediate and remote aetiologies of type 2 diabetes, and metabolic diseases with multiple hormone pathways for better glucose regulation and weight dissipation, including triple agonists (GIP/GLP-1/Glucagon)[24,25] for better effect in comparison to surgery. Alongside these pharmacological advances, researchers are exploring gene-editing (CRISPR), microbiome manipulation, and targeted therapies for complications like diabetic retinopathy, such as gene silencing for VEGF, with the objective to amend the underlying pathophysiology, upgrade outcomes, and provide personalized therapies. Type 2 diabetes is characteristically a chronic metabolic disorder presenting insulin resistance and pancreatic beta cell dysfunction, that culminates in elevated blood glucose concentrations.[57]

Current therapies, indicative of metformin, sulfonylureas, and insulin, have been longstanding therapeutics, however, with issues, such as adverse effects, decreased efficacy in

the long-run, and restrictions in achieving topnotch glycaemic control. These have spurred pronounced interest in the development of nascent, novel and experimental therapeutic approaches to improve therapeutic outcomes. Recently, advances in diabetes management have presented dual incretin receptor agonists, such as tirzepatide, which combine GLP-1 and GIP receptor agonist, leading to elevated insulin secretion, suppressed glucagon discharge, and pronounced weight dissipation [Chukwuma x3]. Dual SGLT1/2 inhibitors, such as sotagliflozin also exhibit significant blood glucose decrement and enhanced weight dissipation by targeting the regulation of glucose in the gut and kidneys. Also of promise are glucagon receptor antagonists, GPR119 agonists, and FGF21 analogues, which enhance insulin sensitivity and glucose metabolism via innovative trajectories. Technologies involving gene editing, encompassing CRISPR-Cas9 and AMPK activators are researched to effectively solve the underlying type 2 diabetes pathophysiology, with interests and concerns on the long-run efficacy and safety. Future perspectives on conventional and innovative type 2 diabetes therapies focus on the potential of novel therapies to modify diabetes care and improve patient outcomes.[58]

Senolytic Drugs: The functions of these drugs is to eliminate senescent or ageing cells which enhance tissue dysfunction and age-related pathologies.[59] Senescence is a characteristic cellular condition of ostensibly permanent interrupted cell cycle and disparate secretory phenotype. Senescent cells present diverse advantageous physiological functions, but progressive aggregating of these cells as a result of ageing or other anomalous states induce debilitating impacts on the usual activity of the same or elevated hierarchy of biological organizations. The elimination of senescent cells in vivo, on application of senolytics, tend to mitigate anomalies established with an augmented amount of senescent cells. On that score, researchers have rigorously attempted to develop new senolytics with disparate selectivity and potency. Future studies must incorporate the classification of proposed senolytics and their mechanisms of action, the heterogeneity of senolytics per their impact magnitude, cell type specificity, and on the explored modalities to enhance these features, as well as prospective pathways for the novel technologies for senescent cell ablation.[60] Oxidative stress is due to disequilibrium between reactive species of oxygen and nitrogen formation and the antioxidant defense mechanisms of the body. Accumulation in excess of these reactive molecules deranges lipids, proteins, and DNA, thereby enhancing cellular impairment and ageing progression. The Free Radical Theory of Ageing, propounded by Denham Harman indicates that oxidative stress facilitates molecular degradation resulting age-associated functional debility; although, current research suggests oxidative stress is both deleterious and adaptive, in its impact on cellular signaling and stress responses. Concurrent evidence undergirds gene-environment interactions[7] and attributes in oxidative homeostasis, and latitude for therapeutic interventions for ageing-associated anomalies. Explicating oxidative stress as a dynamic and regulated mechanism other than an invariable degenerative impact unravels pathways for targeted approaches in ageing modulation, healthspan extension[61] and longevity.[62]

Mitochondrial transplantation, MT is an experimental technique in which damaged mitochondria undergoes replacement for the augmentation of cellular energy formation and functionality.[63] As an advancing therapeutic approach, mitochondrial transplantation is beneficial under the condition of transfer of healthy robust mitochondria bio-augments metabolically impaired cells or tissues.[63] The ambit of MT emerged in the early 1980s for the development of antibiotic resistance amongst cells. As an innovative therapy, MT has made diverse ventures in grappling with metabolic impairments in varied systemic disorders, such as metabolic diseases affect ocular tissues, heart, lung and brain, contributing to their pathogenesis.

Modulation of Key Pathways targets pathways such as NF-κB associated with inflammation and mTOR that is linked with cell growth and metabolism exhibiting potential to influence the ageing process and manage age-related disorders[64], as well as potentiated research of modulators, such as spermidine and metformin in human clinical trials, facilitating future interventions.

Therapeutic Targets: Novel interventions and senolytic drugs which eliminate senescent cells, and mitochondrial transplantation, modulation of pivotal pathways, such as NF-κB and mTOR are effective in animal models for retarding the ageing process and providing therapeutic effects in age-related disorders. Inasmuch as impactful interventions have been successful in animal models for ageing retardation and therapeutic modalities for age-related disorders, the strategies are in the infancy stage or of premature human mitochondrial medical practice. Mitochondrial health relates to a fragile balance of specific functions, such as metabolism, signaling, and dynamics which are debilitated in neurodegenerative disorders. The regeneration of mitochondrial function by selective targeting of mitochondrial stressors, such as reactive oxygen species, inflammation or proteotoxic insults as in “bottom-up” approaches, constitute an expansively explored therapeutic approach. Although, these strategies are effective in preclinical studies, they not been able to demonstrate perspicuous clinical benefits. Enhancing mitochondrial capacity and other cellular ingredients for the regeneration of a healthy cellular ambient is an advantageous complementary or alternative strategy. Further research provide a non-technical perspective in brain metabolism on neuroprotective approaches targeting mitochondria with emphasis on top-down interventions such as exercise or physical training, metabolic modulators, caloric or dietary restriction, brain stimulation and conditioning, with encompassing conceptual disparities to bottom-up strategies, and hypothetically in the manner these mechanistically comparatively inadequately defined top-

down therapies are liable to function, via the discourse of predominantly mitochondrial stress responses and mitohormesis.[65]

Accelerated Vaccine Development: Certain breakthroughs in platforms, such as mRNA technology, as evidenced in the COVID-19 pandemic era facilitated the accelerated production and accessibility of novel vaccines[66,67], with immunotherapy being considered a treatment option.[68] The COVID-19 pandemic exhibited how platforms, such as mRNA technology spurred rapid vaccine production, fostering wider application in cancer therapy, while immunotherapy provides profound treatments, with future emphasis on the refining of mRNA long-run impacts, delivery, and explicating immune responses for intricately complex diseases, encompassing diseases other than infectious disease prevention to personalized cancer vaccines. The manifestation of messenger RNA, mRNA vaccines depicted a seminal shift in immunization, highlighting an era featuring immeasurable rapidity and efficacy in profound infectious disease presentation. The global crisis due to the COVID-19 pandemic catalytically influenced the accelerated production and accessibility of two preeminent mRNA vaccines, Comirnaty and SpikeVax, defining not merely the technological mRNA premise, rather the transformative potential in public health approaches. Research provides an expansive avenue of the fundamental hallmarks of mRNA vaccine technology, elucidation of the exclusively defined benefits exceeding conventional vaccine spheres, analysis of extant challenges, opportunities and priorities encountered for the future of public health via integrated mRNA technology and public health precincts for the enhancement of global health security and adaptation during emerging[69] and reemerging infectious geopolitical threats.[70] The mRNA therapeutics continue to transform medicine with an interdisciplinary sphere to configure pathways to address erstwhile unmanageable morbidities. Current progress in biotechnology have enhanced efficiency and effectivity in the development of functional proteins, antibodies, and peptides by means of mRNA with accelerated and adaptable solutions for vaccine production and treatment interventions. The resultant benefits of mRNA vaccines, demonstrated in the COVID-19 pandemic era the potential to counteract infectious diseases with formidable intensity. The progress provide succour for patients with intricately complex or intractable conditions, such as cancer the trajectory for a newfangled era in targeted therapies and personalized healthcare[71] opportunities and priorities.

Elaborate experimental data from cell and animal tumor models indicate that hyaluronan–CD44 interactions are important in both malignancy and resistance to cancer treatment. Due to the close association between the hyaluronan–CD44 system and tumor cell survival, growth and development, there are expansive research applications to anticancer chemotherapeutics. Interference with the hyaluronan–CD44 interaction by targeting drugs to CD44 and the hyaluronan matrix, or interference with hyaluronan matrix/tumor cell-related CD44 interactions constitutes a tenable approach for cancer therapy. Several of these methodologies can retard or mitigate tumor burden in animal models but have not been amenable to observable clinical utility. Recent advances in nanomedicine have been valuable for cancer prevention, detection and therapy.[72] The augmented permeability and retention impact rationalizes application of nanoparticles for the treatment of solid tumours. The targeted and concerted delivery of these particles to every tumor-inundated precinct in ample amounts necessitates optimization. A conducive nanocarrier needs to be well-equipped with specific ligands which are increasingly or definitively expressed on target cells and, therefore undergird the carriers with select targeting capabilities, particularly the importance of the hyaluronan–CD44 system in the provision of this sort of alternative in tumours expressing defined CD44 variants.[72]

Precision Medicine: The integration of multi-omics data (genomics, epigenomics, metabolomics, and microbiomics)[73] and artificial intelligence is making headway to a highly personalized strategy for type 2 diabetes management, paving trajectories for specific therapies correlated with the unique profile of a person. Type 2 diabetes pathophysiology is intricately complex, displaying three major mechanisms which culminate in increased glucose concentrations. Insulin resistance inhibits glucose usage in muscles, adipose tissue, and the liver. Pancreatic dysfunction leads to untoward excessive glucose release and disruption of insulin and glucagon concentrations, thus augmenting hyperglycemia. Tailored management approaches to personalised requirements and stages of the disease is of importance. Genetic factors contribute to type 2 diabetes development, and needs to be inculcated in any therapeutic modality. Genome-Wide Association Studies (GWAS) have determined extensive genetic loci and Single Nucleotide Polymorphisms (SNPs) related with type 2 diabetes. A personalized pattern takes into consideration an expansive array of attributes, such as patient disposition or characteristics, medical history, sequelae, and genetic constitution in order to enhance outcomes and mitigate type 2 diabetes impact on entire health through Next Generation Sequencing (NGS) and pharmacotherapy.[74]

Drug-induced nephrotoxicity

Nephrotoxicity having drugs as aetiologic agents continues to constitute a gruesome impediment in pharmacotherapy of a vast majority of diseases, and represents circa 25 % of severe side-effects following drug administration.[75] Therefore, drug-induced nephrotoxicity is a general sequela of numerous medications and diagnostic agents, observed in both inpatient and outpatient settings with variations in presentations extending from mild, reversible damage to severe kidney disorder. Circa one-third of type I diabetic patients present with diabetic nephropathy with confounding pathogenesis--usually manifesting as heavy persistent proteinuria, decreased glomerular filtration rate, and elevated arterial hypertension.[76] Also, certain groups

of drugs, such as antibiotics, nonsteroidal anti-inflammatory, chemotherapeutic, antiviral and antifungal drugs, as well as immunosuppressants have the predilection for clinically degrading the kidney and may be alluded to as "nephrotic silent killer". The onset of acute kidney failure enmeshed with drug administration is exhibited in circa 20 % of patients and several become susceptible to chronic kidney disease vulnerability causing restricted application of these sort of drugs in clinical practice. The complex association between immunological, vascular and inflammatory incidents which induce kidney impairment necessitate future research for implementation in the bioengineering of nascent or novel biomarkers for prompt detection of drug-related kidney degradation, such as Kidney Injury Molecule (KIM-1), lipocalin related to neutrophil gelatinase (NGAL) and diverse microRNAs. The application of artificial intelligence (AI) to develop computer algorithms for the early or prompt detection of kidney deterioration is pertinent for future clinical research of novel potential drugs or natural products for accessible education to obviate or ameliorate drug-induced nephrotoxicity.[77]

DISCUSSION

Recent research has ingrained the understanding of the intricately complex, reciprocal interplay in the spheres of ageing, diabetes, and (re)emerging infectious diseases, unveiling shared underlying mechanisms and marked clinical implications[78], particularly on the aftermath of the COVID-19 pandemic. The association is driven by shared underlying approaches, essentially chronic inflammation and immune dysregulation, with pronounced clinical impacts in patient management and public health interests. Research has increasingly focused on cellular senescence as an incessant and frequent molecular process in ageing and diabetes. The senescent pancreatic beta cell accumulation, for example, assists in anomalous insulin secretion and glucose intolerance in elderly individuals.[79] Type 2 diabetes is associated as a model of premature senescence and accelerated ageing, whereby diabetic patients depict early decline in numerous body systems, such as rapid debilitating muscle mass in comparison to non-diabetic persons[7,80] Progress in biomarker Identification and technology as well as machine learning are enhancing biological age and associated risk prediction in diabetic patients. The application of advanced diabetes technologies, such as continuous glucose monitoring and smart insulin pens in elderly persons is a pertinent research sphere enhanced glycaemic regulation and decrement in hypoglycaemia risk.[81]

The dynamic Interplay with (re)emerging Infectious diseases[82,83] indicates that emerging and (re)emerging infectious diseases (EIDs and REIDs) constitute an evolving debacle to global health security manifesting impromptu outbreak of novel infections, such as COVID-19, SARS, Ebola or the reemergence of erstwhile regulated diseases, for instance, cholera, tuberculosis, and malaria malaria. These events are driven by an intricately complex, dynamic interplay of biological, environmental, and socio-political attributes, with circa 60% of extant and 75% of nascent human diseases resulting from animals.[84,85] Diabetes patients manifest elevated vulnerability and severity of 1.5- to 4-fold higher risk of diverse, frequent and deleterious infections, with worse clinical outcomes than the general population of non-diabetic persons, as a result of immune system anomalies, characterized by dysfunctional immune cell, chronic inflammation or "inflammageing", and vascular insufficiency.[86,87] A reciprocal relationship exists where certain infections, remarkably SARS-CoV-2 (COVID-19), increases the risk of new-onset diabetes or deteriorate extant glycaemic control, invariably infecting pancreatic beta cells.[88] Evidentially, a bidirectional or reciprocal association between COVID-19 and diabetes, with pre-existing diabetes augmenting severe COVID-19 risk, and obversely, COVID-19 induces new-onset diabetes or perspicuously deteriorate extant glycaemic regulation. Hyperglycaemia constitutes a pivotal factor whereby high blood glucose levels are sustained as a primordial predictor of disease severity and mortality due to infections, such as COVID-19, irrespective of the pre-existing diabetes state of the patient.[89]

The therapeutic potential due to the discovery of encompassing signaling pathways and comorbidities between ageing, diabetes, and infectious diseases spurred research into potential co-therapies. Natural agents, such as resveratrol and curcumin, possessing anti-diabetic, anti-ageing, and antiviral attributes, are incessantly exploited for target and delivery as therapeutic drugs. As ageing is a complex, multifactorial mechanism with characterisation of progressive physiological deterioration and increasing susceptibility to chronic disorders and syndromes, gerotherapeutics and the vital functionality of an aggregated, mechanism-focus for therapeutically targeting ageing biology senolytics, senomorphics, NAD+ precursors, mTOR inhibitors, and metabolic modifiers, such as metformin are pertinent. Future interdisciplinary research and publication emphasizing their mechanisms backed by preclinical evidence in efficacy and contextually translational clinical impetus is of benefit.[56,90] Research involving co-therapies for ageing, diabetes, and infectious diseases is undergirded by the emergence of shared molecular mechanisms, specifically chronic low-grade inflammation or "inflammageing", oxidative stress, and immune derangement. These shared pathways potentiate diabetes to increase the rate biological ageing trajectory, concurrently as both conditions debilitate the potential of the immune system to suppress pathogens.[91]

CONCLUSION

This review narrates the intricate intersections of research and publication in ageing, diabetes, chronic and (re)emerging infectious diseases, climate change, environmental perturbations and geopolitical foundations in understanding the complexities of modern public health challenges, opportunities and priorities in clinical and theoretical paradigms. The article grants an expansive

discourse of the interconnected dynamics between these factors in a bid seeking to underscore shared molecular mechanisms, clinical implications, and potential therapeutic strategies. Interdisciplinary potential, particularly through the lens of geroscience and public health, is considerable and suggests avenues for collaborative research and innovation. By enhancing these aspects, the manuscript has the potential to advance discourse in medical, epidemiological, and public health fields, prompting critical conversations and encouraging research collaboration. The convergence of molecular biology with public health presented herein is both timely and vital for addressing future disease challenges. Ageing and diabetes are placed in the broader context of geopolitical health challenges, climate change and global mobility. Delving into spheres such as cellular senescence, inflammageing, and mitochondrial dysfunction; and this work contributes to explicate how these factors impact disease (re)emergence and progression in shared responsibility, with a composite understanding of complex biological and environmental interactions as pertinent to extant scientific literature. The focus on the interplay of ageing, diabetes, and infectious diseases provides a multidimensional stance that can expand understanding in both scientific and public health discourses. The integration of concepts like cellular senescence and inflammageing is Hallmark for the interconnectedness of diverse health issues and disease burden in which research and publication have provided significant stride towards holistic health strategies. The discourse on therapeutic potentials based on understanding shared molecular mechanisms present the intendment to drive future research and publication agendae and clinical practices for sustainable governance, systems and development.

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