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Ageing, cellular senescence, and diabetes, probing the associations between these mechanisms and consequences for therapeutics, treatment and management

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Abstract

The defined disparity between ageing and senescence is that ageing constitutes an intricately complex and definite biological mechanism characterized by a progressive depreciation in physiological functionalities and elevated mortality risk. Senescence features a cellular mechanism of a conditioned state of the arrest of cell cycle and secretion of inflammatory molecules. Cellular senescence correlates to the ageing process as the ageing immune system declines in efficiency culminating in senescent cells accumulation with superimposition on healthy cells. This impacts ability to cope with stress or impaired health, injury recovery, and configure or inculcate new ideas as brain senescent cells are capable of the impairment of cognitive activities. The aetiologies of cellular senescence are triggered by multifarious factors which include reiterated cell culture, telomere attrition, irradiation, oncogene activation, and oxidative derangement, as well as impairment of mitochondrial homeostasis, with propensity to accelerate age-associated phenotypes. The pathophysiologic mechanisms related with diabetes impacting the ageing elderly population due to advanced age culminate in the exacerbation of systemic chronic inflammation, oxidative stress, DNA damage, decline of mitochondrial function, cellular senescence, and tissue dysfunction, all conditions which contribute to generate metabolic disorders. With increasing age, the pancreas produces diminished insulin and resultant prolonged increased blood sugar. The process of cellular ageing is associated with increasing cell size and diminished capabilities to divide and/or multiply. In addition to other alterations, there is augmentation of pigments and fatty structures or lipids within the cells. Ageing impacts cellular actions which contribute to age-related disorders as ageing cells accumulate misfolded and perturbed proteins due to functional deterioration in the homeostasis or proteostasis process of the proteins.The cellular senescence mechanism results since cellular senescence is a highly formidable cell cycle arrest that is generated in reaction to disparate stresses. With the imposition of any growth arrest, senescence restricts aged or deranged cell replication. Age constitutes a major risk factor for type 2 diabetes However, It is not clearly explicated how senescence contributes to the pathogenesis of diabetes. Thus, available treatment regimen has not targeted the defined portion of the disease. Suppression of the deranged presentations of cellular ageing is important as a novel type 2 diabetes treatment where pancreatic beta cells is impaired regarding insulin secretion. Adequate therapeutic trajectories necessitate characterization of senescent β-cell populations and the spatiotemporal variations of senescence gene expression.

Keywords: aetiopathogenesis; clinicopathologic correlates; SASP; age-related diseases; pancreatic β-Cell senescence; senotherapy

Introduction

With increasing human life expectancy, it becomes a global concern that fixates on underlying ageing processes. Increased longevity has resulted in an expanding elderly population in the turf of age-related diseases (ARDs) which encumber global healthcare systems and economies. The prevalence of type 2 diabetes increases with ageing. The type 2 diabetes presenting in the elderly population is accompanied frequently by slow degradation of beta cell functionality and decreased β-cell mass concomitantly with ageing. The input of β-cell senescence in type 2 diabetes pathogenesis remains unclear [1]. Elucidating the intricate associations between ageing and aetiopathophysiological processes is pertinent in the issues, challenges and opportunities presenting in diabetes [2], obesity [3], senescence [4] and other age-related disorders. Expansive evidence portrays interrelated mechanisms and pathways which are contributory factors to their development and progression, thereby necessitate intervention strategies to retard the onset of varied pathologic states [5]. In this regard, conditions such as ageing and diabetes culminate in identical organ impairment driven by parallel molecular processes, as well as cellular senescence. The preponderance of senescent cells in diverse tissues increases with age, diabetes and obesity. Senescent cells are invariably indicted in insulin resistance generation [6]

Cellular senescence is an irreversible condition of cell cycle arrest triggered by diverse stimuli extremely associated with ageing and varied chronic ailments. Research predominantly suggested that senescent cell accumulation was a significant promoter to diminished organ metabolism, culminating in metabolic aberrations. On the contrary, the senescent cells eradication may ameliorate or adjourn metabolic disease onset or progression. Therefore, a vital relatedness between senescent cells and metabolic anomalies is possibly determined, and targeting senescent cells emanated as an alternative therapy associated with metabolic disease treatment [7].

Aetiopathogenesis of concomitant ageing, cellular senescence and type 2 diabetes

Characteristically, type 2 diabetes presents with insulin resistance and dissipation of β cell mass and functionality. Ageing is regarded as a major risk factor for type 2 diabetes development. In elderly people, the contributions of pancreatic β cell senescence and systemic ageing in the type 2 diabetes pathogenesis is inadequately explicated [8]. Regarding this instance, cellular senescence is a cell fate that is a consequence of multiple sorts of stress which are capable of inducing tissue repair or drive inflammation as well as degradation of tissue homeostasis. Ageing and obesity result in elevated senescent cell burden in numerous organs. Senescent cells discharge an array of senescence-associated secretory phenotype factors which invariably mediate pancreatic β-cell and adipose tissue anomalies as well as insulin resistance in peripheral tissues and induce the onset of type 2 diabetes. Senescent cells discharge an array of senescence-related secretory phenotype factors which mediate pancreatic β-cell dysfunction, adipose tissue dysfunction, and insulin resistance in peripheral tissues with resultant development of type 2 diabetes. Furthermore, hyperglycaemia and metabolic alterations in diabetes induce cellular senescence. Diabetes-Diabetes enhanced cellular senescence undergirds multiple diabetes complications. Therefore, type 2 diabetes is both an aetiology and repercussion of cellular senescence [9].

As cellular senescence constitutes a primordial ageing mechanism in several age-associated anomalies, it is indicted as a significant aetiological agent in tissue dysfunctionality. Senescent cell accumulation results during ageing and the instance of obesity and diabetes. Senescent cells may contribute in type 2 diabetes pathogenesis invariably via impact on pancreatic β-cell function, senescence-associated secretory phenotype (SASP)-mediated tissue perturbation, and and interconnectivity in adipose tissue impairment [10]. Simultaneously,, metabolic and signaling alterations depicted in diabetes, such as elevated circulating glucose, deranged lipid metabolism, and growth hormone axis dysfunctions, can trigger senescent cell production. As indicated previously, senescent cells may contribute as a pathogenic loop in diabetes, combinatorially as the aetiology and resultant impact of metabolic alterations and tissue disruption. Therapeutic targeting of a fundamental ageing mechanism, such as cellular senescence may impact immensely on disease pathogenesis with expansive effectivity in curbing diabetes complication progression than available therapies which present restricted effect on extant deranged tissue. Thus, senescent cells and SASP pose vital opportunities in the face of issues and challenges for advances in the prevention and treatment of type 2 diabetes and its sequelae [10, 11].

There is extant vital discourse of biomedical dilemma in harnessing or treating type 2 diabetes. Type 2 diabetes contributes for an excess of 90% of the overall diagnosed cases of diabetes, probably due to ageing, inflammatory factors, obesity and β-cell senescence. The principal symptom present in both type 1 and type 2 diabetes is elevated blood glucose concentration. Whereas type 1 diabetes is insulin-dependent with linkage to degradation of pancreatic β-cells, the type 2 diabetes is not susceptible to protracted insulin intake. In this regard, pancreatic β-cells are extinguished, however, the functionality is deregulated. In type 2 diabetes, metabolic stress enhances the number of senescent β-cells with concomitant deterioration of glucose tolerance [12]. The potential paracrine impact of senescent β-cells signify the β-cell senescenc-associated secretory phenotype (SASP) in driving metabolic degradation. It is instructive that the deregulation of the functionality of pancreatic β-cells in type 2 diabetes is related to ageing or senescence due to induced stressors. In essence as ageing occurs, incessant exposure to stressors culminates in pancreatic β-cell senescence. Pancreatic β-cells regulate glucose homeostasis by secreting insulin, with concomitant resultant dysfunction to the metabolic control emanates as a primordial contributor to type 2 diabetes development. Sustained cell cycle arrest, impaired secretory presentations, and diminished proliferation are characteristic of pancreatic β-cell senescence [13]. Thus, β-cell extinguished prowess to regenerate, resulting in diminished β-cell mass and perturbation of glucose homeostasis, impact type 2 diabetes pathophysiology due to these alterations. The consequential diminished β-cell mass and functionality, in consonance with the degrading impacts of the senescence-associated secretory phenotype (SASP), do play a vital role in insulin resistance and type 2 diabetes progression. SASP constitutes a paramountinextricably linkedsecretory state associated with structural and metabolic alterations, protracted DNA degradation responses, and the upregulation of multiple cell cycle inhibitors,

which are inextricably-linked with pancreatic β-cell senescence [13]. It is pertinent to explore the intricate complexity of the interplay between these molecular factors and the rationale for type 2 diabetes pathogenesis.

Pathophysiological characteristics and mechanisms associated with ageing, cellular senescence and type 2 diabetes

Ageing results in elevated cellular stress as a result of organismic fragility and nonfunctional capacity to undergo stress. Thus, paving the increased latitude for the development varied cardiometabolic anomalies, for instance, diabetes. The duo of cellular senescence and autophagy represent hallmarks of ageing and stress-coping trajectories which have earned prominence for contribution in diabetes pathophysiology [14]. Senescence perturbation, both diminishes and harnesses diabetes onset and progression, whereas autophagy presents a divergent function, indicating a context- and disease-stage-dependent impact. Studies depict contradictory results regarding the impact of autophagy on senescence, but the about interaction in β-cells has not been clearly elucidated [14]. Understanding the inextricable linkage between autophagy and senescence in pancreatic β-cells can assist in establishing respective roles, and to what extent the impact each mechanism prevails on β-cells and in order to create nascent trajectories to develop novel and efficacious therapeutics. As senescence presents as an aspect of the cellular response to disparate stressors, with increased ageing, incessant exposure to stressors culminates in age-induced senescence. Also, pancreatic β-cell proliferation and glucose homeostasis diminish with ageing, with resultant diminished β-cell mass and, consequentially, the probability diabetes onset and progression. The mechanism is mediated via perturbed cell cycle regulators, in consonant with defined augmentation in cell cycle inhibitors, telomere shortening, and impaired DNA repair processes [15]. Diabetes plays a role β-cell senescence per hyperglycemia, dyslipidaemia, oxidative stress, and inflammation. β-cells isolated from type 2 diabetes patients depict senescence markers, such as senescence-related secretory phenotype genes and β-galactosidase.

Cellular senescence considerably a significant process in the development and progression of diverse aberrant states which may be inextricably associated with metabolic anomalies, such as obesity and type 2 diabetes. The featuring of obesity and diabetes is established as a major risk factor in accrued onset and progression of impaired health, likely heart, vascular, kidney, and cancer derangements. As senescent cells are capable of being drivers of disease presence, it is perspicuous that obesity and diabetes can potentiate the ambient that accelerates cell senescence within tissues [16]. This consequentially depicts as age-associated biological perturbations and secondary disorders. Cell senescence in cell types related with obesity and diabetes, such as adipocytes and pancreatic β-cells must be studied regarding the contribution of obesity and diabetes in accelerating ageing via the incorporation of premature cell senescence [4] mediated by elevated glucose concentrations and oxidised low-density lipoproteins. Studies have to concentrate on rapid cell senescence in endothelial progenitors and endothelial cells as well as vascular smooth muscle cells in combination with cardiovascular and proximal tubular cells deterioration as linked to kidney failure. Type 2 diabetes perspicuously increases with the ageing process. The significance of this inextricable linkage is remarkably profound by the resultant ageing of global population. Current data depict the vital contribution of β-cell impairment in the age-associated disability of pancreatic endocrine activity, and delineate the availability of novel therapeutic interventions [17].

The philosophy of therapeutics in ageing, cellular senescence and clinicopathologic correlates

Ageing is a dynamic public health enigma in current demographic transitions where there is an exponential increase in persons from 60 years of age. The relationship between age and diabetes may be decipherable because the onset of type 2 diabetes is usually present in persons aged 45–64 years. It is commonplace to detect the disease after 45 years of age but may appear at any age. Therapeutic regimen that specifically target senescent cells called senolytics are utilised in clinical trials [18]. The increasing effectiveness of healthcare definitely results in an accelerated enhancement in the global population of the elderly. With increased ageing, the population presents chronic disabilities, which predominantly augment the socioeconomic burden with concomitant diminished self-help, quality of life, and lifespan of the ageing population. Due to ageing being governed by multifactorial and multidimensional mechanisms involving gene-environment interactions, such as genetic predisposition and chronic sterile inflammation including cellular senescence contribute significantly, and targeted to revert the degrading impacts on tissue homeostasis and functional balance. Cellular senescence connotes the irreversible suppression of cellular proliferation due to the aberrant exposure of cells to extrinsic or endogenous stress [19]. Extant evidence characterizes multiple ageing phenotypes, and that eradication of senescent cells at the tissue level is capable of enhancing age-associated tissue integrity. These observations have renewed scientific interest in possible therapeutic interventions.

Cellular senescence as a state of irreversible cell cycle arrest, plays an intricately complex role in ageing and age-related anomalies, while senotherapeutics, drugs targeting senescent cells (SnCs), provide therapeutic procedures. Senotherapeutics are classifiable as senolytics (senescent cell expungement) or senomorphics (SASP attenuation), with preclinical and clinical trials exhibiting the potentialities of the strategies. Ardent research has arduously attempted to explicate senescence mechanisms by means of the identification of novel factors which interact with ageing and age-related complexities in treatment management, quality of life and lifespan in diabetes and its complications. An elucidation of the primordial mechanisms which lead to ageing and research-oriented trajectory on successful ageing will alleviate multiple socioeconomic and healthcare encumbrances presently and in the future for diabetes and associated presentations [20, 21]. Cellular senescence is a response to multiple stressors, such as DNA impairment, oncogene activation, physiological ageing, and anomalous rapidly promoted senescence are contributory factors to presentations of human disorders as well as diabetes. Research has depicted a role in pancreatic β-cell senescence in the pathogenesis of type 1 diabetes, type 2 diabetes and monogenic diabetes [22]. Small molecule or genetic targeting of senescent β-cells has depicted a newfangled therapeutic strategy for diabetes prevention and treatment. Irrespective of the progress, constraints and challenges abound in elucidating the molecular mechanisms which drive senescence in the β-cell, identifying molecular markers

which demarcate, detect or differentiate senescent and non-senescent β-cell subpopulations, including translation of proof-of-concept therapeutics into nascent human diabetes treatment.

Discussion

Regarding the resultant diseases of civilization, the population presenting with type 2 diabetes is anticipated to expand to more than an excess of one billion in less than two decades in clinicopathologic correlation with inter alia ageing population, poor dietary factors, sedentary lifestyle, dire genetic predispositions and immunological variables [23]. Type 2 diabetes is implicated in the dysfunction of multiple organs with characterisation of insulin resistance, elevated glucose concentration, and adipocyte impairment in correlation with senescence. This variety of cellular ageing is of vital biological functionalities but can be detrimental due to resistance of senescent adipocytes to apoptosis, augmentation of cytokine secretion, downregulation of cell identity genes, and inculcation of the senescence-associated secretory phenotype that provides a more oxidative ambient. Harnessing type 2 diabetes is achievable through a vast array of senotherapies, senolytics and senomorphics, irrespective of the prevailing challenge, issues and opportunities [23]. The attendant sequelae to be envisaged in further research may incorporate secretory phenotype, chronic inflammation, increased insulin resistance, aberrant adipogenesis and adipocyte cell functionalities as well as association of type 2 diabetes and fat tissue senescence, novel adipocyte-associated anti-diabetes further research trajectories.

Undergirded by enhanced ageing population, type 2 diabetes prevalence is concomitantly increasing . Ageing has impacts on the tissues and organs of the entire human body due to diverse pathophysiological intrusions. Adipose tissue possesses an elevated magnitude of plasticity and alters with ageing. Ageing modifies adipose tissue dissemination, impacts on adipogenesis, browning features, inflammatory characterisation and adipokine secretion, as well as enhances lipotoxicity [24]. These age-dependent modifications in adipose tissue contribute as an pivotal aetiology of insulin resistance and type 2 diabetes. (Ref) Elucidating the alterations of adipose tissue invariably advances a healthy or successful ageing trajectory, with aggregate concern on the contribution of ageing adipose tissue in insulin resistance and type 2 diabetes. Ageing is a potent independent risk factor for cardiovascular diseases such as atherosclerosis and heart failure. Concomitant diabetes ardently undergirds this impact of ageing on cardiovascular disease. Cellular senescence is a primordial mechanism of ageing, and ostensibly contributes to the onset and prognosis of cardiovascular disease in the context of both ageing and diabetes [25]. Although, senescent cells are in a condition of cell cycle arrest, these cells are metabolically active due to the ability to secrete inflammatory factors.

The pertinence of insulin sensitivity versus secretion and insulin resistance in the clinical strategy for the treatment, lifestyle modification, early onset interventions and regulation of diabetes cannot be underestimated [26]. Progressive declination of glucose tolerance during the ageing process is associated with the aetiopathogenesis of type 2 diabetes due to peripheral insulin resistance and derangement of β-cell functions [26]. The presenting comorbidities or clinicopathologic correlates, age-associated anomalies and functionality with obesity is common with the global prevalence of sarcopenic obesity, type 2, cancer and other diseases in older adults. Due to no extant cognizant drugs which target comorbidities, it is vital to designate or create trajectories for therapeutic formulations with the approaches of functional genomics which are increasingly feasible in disease aetiopathogenesis, drug target and discovery, as well as genetic variations [27] in ageing, cellular senescence and type 2 diabetes.

Conclusion

Type 2 diabetes is a principal metabolic disease of the global population that requires a vast latitude of therapeutics for healthcare. The derangement of pancreatic β-cells, hyperglycaemia, and systemic inflammation barring insulin signaling are crucial in type 2 diabetes therapeutic regimen. Insulin resistance and adipocyte remodeling emanating from overnutrition may result in adipocyte senescence. The complications and consequences which necessitate expansive research encompass SASP phenotype, chronic inflammation, insulin resistance increase, including debilitated and debilitating adipogenesis and adipocyte cell functionality. Senotherapy constitutes the primordial strategy to suppress senescent adipocytes that inculcates senolytics and SASP-inhibiting therapeutics. Available therapeutic strategies directed at glucose concentration, insulin resistance, and senescence must incorporate focused strategies on immunological, adipocytic, and signaling concerns. Several clinicopathologic correlates must be attended to, particularly senescence, eradication of adipocytes and SASP phenotype, as well as type 2 diabetes . The treatment trajectory incorporating senolytics and SASP inhibitors necessitates amongst other modus operandi, a strategic personalised focus. Furthermore, the therapeutic latitude of targeting senescent cells using senotherapeutics, such as senolytic and senomorphic, and compensatory research to explicate or elucidate the underlying process for clinical application optimizations

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