Theories of Human Aging of Molecules to Society

Abstract

Aging is a process of deterioration of physiological functions, time-dependent, leading to homeoestenosis. The causes and mechanisms of this process are not yet fully clarified so have issued numerous theories to explain it.The article reviews the major theories of human aging, while proposing a classification that covers the fundamental mechanisms, including deregulation of the immune response (oxy-inflamm-aging) as one of the most cited today.The theories also include those relating to the psychosocial aspects of aging, taking a holistic approach to this biological process of humans.

Keywords: Aging theories elderly

Review Article

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Introduction

Human aging comprising the progressive deterioration, time-dependent functions of the adult organism with a homeoestenosis key physiological systems (nervous, endocrine, immune, cardiovascular and musculoskeletal), which goes from the robustness to frailty, illness, disability and ending in death [1,2]. Sources and methods to study aging are imperfect because of the obvious difficulty of bioethics human experimentation, and so laborious it is for researchers to conduct longitudinal studies of aging populations, so that the main efforts have been directed to experimental study short-lived animals (flies, worms, mice), the results are difficult to extrapolate to humans, and cross-sectional studies in aged sectors, such as the recent studies on long-lived Italian [3,4].

Man has always tried to find the cause of aging, reaching so laborious and useless "cure" for a process that is not a disease, and by which all inexorably have to navigate. Thus, over the years, from Cicero until today many theories have been proposed to support a variety of assumptions aging patterns [5]. Many of these theories have been disproved or abandoned for lack of evidence to support them and others continue providing plausible explanations, although insufficient to cover all sides of a process holistically speaking, is multifactorial and in which are embedded the genetic background, biological changes and psychological, social and environmental conditions where human beings live and develop. It has been rightly said that while there are more theories about an issue, it shows that it is less understood. It is for this reason that the author is proposed in this paper to review and condense the most current theories of aging that show "the best evidence" to gain insight into this very relieved and unenlightened.

Towards a Classification of Theories of Aging

Although sometimes proclaims that there are hundreds of theories of aging that is wrong. Perhaps there are hundreds of specific cellular and molecular mechanisms that contribute to the intrinsic biology of aging but these can be arranged upon a small number of major theories [6]. In past decades, theories were divided for your understanding in two main aspects: they considered aging as a process scheduled under the control of genes and other mechanisms due to unscheduled (stochastic) [7-9].

This traditional approach has now changed as well as considering the role of evolution in aging, divide the theories into two broad categories: biological, where it continues case aging as an evolutionary event genetically controlled, and chemical as a somatic process resulting from the cumulative chemical damage to bio molecules [10,11]. In the psychological theories that attempt to explain the changes that occur in this area as cognitive theories and the so-called theory of mind (Theory of Mind) [12,13] appear. In social gerontology from the 60 various theories linking human behavior with the mechanisms involved in the aging process [14] appear. Condensing theories and taking into account all essential to consider the human being as being (biological, psychological and social), the author proposes a classification which, although not complete, intended to be comprehensive (Table 1) spheres.

Biological Theories

Evolutionary theories of aging

Evolutionary theories of aging seek to explain why this process occurs through natural selection [15]. Classically predict that aging rates should be higher in animal populations in which the extrinsic mortality is high [16]. These theories take into account the fact that, in the wild, the mean extent of life of a species is usually shorter than would be protected media as in animals in captivity. In such conditions, because most animals die before reaching old age, there is no selection for or against the alleles is expressed at later ages [17]. Thus, humans, throughout history have achieved live more protected media achieved express these alleles would result in a phenotype of aging. If man does exceed the insults of the early ages (infections, accidents, wars, murders) safely become old.

These theories have evolved since 1962 and propose that the wider benefits to groups (species, kins), the spread of genes or the process of evolution can offset some degree of individual disadvantage and result in the retention of an adverse trait as would be the mammalian aging [18]. In short, the evolutionary theories consider that no specific genes for aging, this is an unscheduled process and depends on natural selection [6,18].

Table 1: Theories of aging.

Biological Theories
• Evolutionary Aging.
• Theory of soma disposable
1. Genetic mechanisms :
Mutation - accumulation
Pleiotropicantagonism
Error catastrophe theory
• Chemical mechanisms
Free radical theory
Mitochondrial theory
Immune theory
No enzymatic damage
Improper repair the damage
• Deregulation of cell number
Replicative senescence
Telomere shortening
Stem cell theory
2. Cognitive theories :
• Theory of Mind
• Theory of scaffolding
3. Psychosocial Theories:
• Role Theory
• Theory of untying
• Activity Theory
• Theory gerotrascendencia

Theory of soma available

Enunciated by Kirkwood [19], this theory proposes that organisms balance and direct resources to invest between reproduction and somatic maintenance [20]. Thus, it is postulated that a greater investment of resources in reproduction decreases investment in the maintenance and repair of soma, thus extending the life is shortened, while the reduction in reproduction lead to the redistribution of resources to the protection and repair, increasing life extension.

Another assumption of this hypothesis is that at early ages resources are diverted from the soma to the germline under stress conditions in order to ensure the adequacy of the next generation [21]. Explicit mathematical models make predictions of this theory that the optimal investment in somatic maintenance is less than that which may be required for a prolonged longevity [22]. Current reviews of this theory consider time as an important resource, as long-lived species like man prolong cell cycle time compared to lower short-lived species, allowing greater fidelity for repair, aspect that could be decisive in extending the life of the species [23]. There is abundant evidence linking with reduced longevity in lower life forms fertility, but human data have been conflicting. A study that compared a cohort of Ashkenazi Jewish centenarians with similar but not exceptional longevity reported that the former had fewer children, the children had them at later ages, along both men and women [24]. Review this phrase because what I mean is that in the population or population aging one of the determinants is to reduce fertility rates and birthrate. It Could Be: We now not only know That Have to Consider this theory in the individually aging demographic aging but Where also [9,25].

Genetic mechanisms

According to this theory, aging is programmed by genes, which presupposes that slow or stop gerontogenes metabolic biochemical pathways essential for homeostasis [26]. The study of the genetics of animals and humans is gradually identifying new genes that increase lifespan when they are mutated or overexpressed. These gerontogenes are classified as regulators of life extension, mediators, effectors, maintainers, genes involved in mitochondrial function and genes that regulate cell senescence and apoptosis [27].

Among the facts that support the involvement of genes as determinants of aging are the following:

1) The relatives of people with exceptional longevity have high probability of being well or long lived

2) The maximum life of each species is fixed and specific to that species

3) The existence of genetic diseases that are expressed phenotypes of aging (progeria and progeroid syndromes).

Linkage studies of families of people exceptionally long life support the existence of a longevity locus on chromosome 3; other putative locus differ between studies [28]. It is noted that the heritability of age at time of death was estimated at about 25% and it seems clear that epigenetic modifications (influence of environment on the genome) have a key role, as individuals with a genetic background similar (eg. homozygous twins) can age very differently according to the environment where they live [29,30].

The analysis of data from longitudinal studies such as the Framingham suggests that the factors responsible for the exceptional longevity and health are not necessarily the same, and that the genetic changes that increase the life span associated with physiological changes typical health and long individual life, reduced risk of mortality from cancer and cardiovascular disease and better adaptive capacity [31]. Premature aging disorders (progeroid syndromes) as Werner syndrome and progeria syndrome Hutchinson-Gilford have been subject of immense interest and that recapitulate many of the phenotypes observed in physiological aging. They not only serve as models for studying the processes of normal aging but also provide valuable insight into the intricate mechanism of senescence [32].

The classic example of progeroid syndrome Werner syndrome is an adult progeria caused by a mutation of the gene encoding the RecQ helicase, with the appearance of visible signs of aging such as skeletal changes and graying of hair early age and premature death [33,34]. Syndrome Hutchinson-Gilford progeriais a child progeria, a rare genetic disorder first described by Jonathan Hutchinson in 1886 and Hastings Gilford in 1897. Since then, about 100 cases have been reported and there are currently 40 worldwide. Most cases occur by mutations “de novo” and are rarely hereditary. The mutation in the LMNA gene, located in band 1q21.1 - 1q21.3 is responsible for most cases. The mutation replaces cytosine for thymine causing the abnormal transcription of the structural protein of the nuclear envelope called prelamine

A. It is characterized by premature aging phenotype with involvement of skin, bones, heart and blood vessels. Patients have the appearance of "plucked bird" and die prematurely from cardiovascular disease in the second decade of life [33,35,36].

Mutation – accumulation

The somatic mutation theory states that the accumulation of mutations in the genetic material of somatic cells in function of time causes a decrease in cell function. In particular, the accumulation of random mutations inactive genes that is important for the functioning of the somatic cells of several organ systems adult, resulting in a decrease in function [37] thereof.

Some authors [38] apply similar principles to those of Koch to defend this theory arises between individuals of species of longer life should have a lower rate of DNA damage than shorter lifespan and that interventions modulate the level of damage and DNA repair capacity must also modulate the rate of aging and longevity, and vice versa.However there are still many gaps to clarify and others criticize the theory of age-dependent mutation noting that laboratory experiments show that the deleterious nature of specific accumulated mutations of aging is reduced and becomes less variables with increasing age [40].

Pleiotropic antagonism

Another major hypothesis for human aging is the antagonistic pleiotropic (AP) presupposes the existence of genes or alleles with beneficial effects in early life and harmful after effects, with the inevitable cost of aging. As noted above, at an early age, before natural selection is strong with an increase of vital rates (maturation, reproduction and survival) and older ages, where natural selection is weak detriment of vital physiological events occurs and homeostasis [40-42].

It is now accepted that the permanent arrest of cell division, known as cellular senescence contributes to aging mechanism AP: cellular senescence could act beneficially in the early suppressing cancer, but at the expense later that involves system cells causing immune frailty and, paradoxically, cancer [43]. The discovery of mutants like the worm Caenorhabditis elegans longevity has changed the view of aging as a liability and entropic process. In these organisms, by mutation, it is possible to reduce the function of certain genes to prolong their maximum life [44]. Although a major focus of evolutionary studies have evaluated the relative abundance of AP aging type alleles that emerge through mutation, senescence per se the likelihood that these alleles arise [45] is reduced. Recently it has been argued that there are few clear examples of genes identified by AP: including the p-53, considered as a gerosupresor and related genes encoding metabolic pathway target of rapamycin (TOR - target of rapamycin). Early in life, the TOR pathway leads development programs that persist in later life as aging programs and related diseases [46].

Error catastrophe theory

The error catastrophe hypothesis, enunciated by Orgel in 1963, is recorded as historical detail. Its postulated is that, over time, the information transmitted in the processes of transcription-translation of the genetic message (in particular, protein synthesis), would be subject to a progressive number of errors, which would occur carrier proteins error, if reached a certain level (threshold) would fall to the cell in a metabolic "catastrophe" [47]. This he announced decrease in the accuracy of the information transfer that would accelerate the progress of aging does not hold, it has not been shown to work reliably comparing macromolecules (DNA polymerase, transfer RNA and proteins) of young animals and old, where no significant variations [48] are located.

Chemical Theories

Free radical theory

This theory, defined by Harman [49], has been one of the handiest in recent decades. Based on this theory, free radicals, which are reactive oxygen species (ROS) produced in metabolic pathways, may play a critical role in aging. These ROS extremely reactive chemical molecules, are considered toxic to produce oxidative damage to various cellular components (membrane lipids, proteins, nuclear and mitochondrial DNA) which causes cellular dysfunction that accompanies aging [50] aging process. Physiologically, a series of enzymatic and non-enzymatic systems act in concert to counteract this toxicity [51], among which are those of superoxide dismutase, catalase, glutathione peroxidase and glutathione reductase [52]. Theoretically, when the concentration of ROS exceeds the antioxidant capacity of the system to lighten defense, the toxic effects appear oxidative damage (Figure 1).

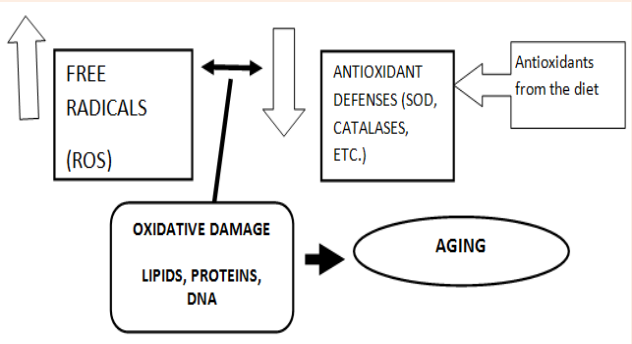

Figure 1: Imbalance between oxidants and antioxidant defenses produce the toxic effects.

Because of its high atomic instability free radicals collide with a biomolecule and subtract one electron oxidizing it. The biomolecule thus loses its specific function in the cell. If it lipid structures that are rich in them, essentially cell membranes and lipoproteins are damaged. In the case of the first altered permeability leading to edema and cell death. In the case of oxidized protein is preferably the amino acids phenylalanine, tyrosine, tryptophan, histidine and methionine. Crosslinks resulting peptide chains, fragmentation of the protein and formation of carbonyl groups are formed. The damage to nucleic acids modified bases, which has serious consequences for the development of mutations and carcinogenesis first, or loss of expression or synthesis of a protein by specific gene damage [53].

Although the theory of free radicals generated multiple investigations in different animal models that have produced abundant data that support, in recent years there have been

critics who suggest strong evidence against it [50]. Some authors suggest that oxidative damage, as other specific damage seen singly or in combination, do not represent the cause of aging [54], since this theory does not explain the causal relationships or the inevitability of the accumulation of damage. Moreover, some laboratories have shown that in some cases, with increased oxidative stress there is an increased longevity [55]. The finding that free radicals not only cause damage to cells but also serve as signal molecules has led to propose that under certain circumstances, might be useful as modulators of physiological processes such as adaptation to exercise [56-58] . Also, if aging is given or conditioned by free radical reactions, the maximum life of animals should be extended by the administration of exogenous antioxidants. However, research in this field is not fully illuminating in this regard [52,59,60].

Although it has been shown that caloric restriction, a method for reducing ROS production, slows aging and extends the maximum life in various animal species [61-63], their effects on disease resistance and mortality in primates - the closest man mammals - are not very consistent. A first study of 20 years of follow up in rhesus monkeys in which caloric restriction without malnutrition was used showed a decrease in the incidence of age-related (diabetes, cancer, cardiovascular disease and cerebral atrophy) [64] diseases. However, another study follow-up study of 23 years young primates to which was subjected to caloric restriction also showed a delay in the onset of diseases associated with aging, but no improvement in survival curves [65].

Mitochondrial Theory

The mitochondrial aging theory, a variant of the free radical theory was enunciated by Miquel [66]. This was named the mitochondrial respiration defined as the main source of ROS, which primarily produce cumulative damage to lipids, proteins and mitochondrial DNA (mtDNA) and lead to cellular aging [67]. ROS formed in mitochondrial respiration deteriorate mtDNA and its functions, which in turn causes a vicious circle by increasing ROS production. The formation of mtDNA mutations can be accelerated by this vicious circle, which could cause accelerated aging [68].

Studies in several animal species show a wide spectrum of disorders in the mitochondria with aging including: disruption of structure, function decline in oxidative phosphorylation, accumulation of mtDNA mutations, increased production of ROS and the oxidative damage to their structures [69]. Mitochondrial biogenesis product decreases with aging of these alterations and inhibition mitophagy. That Eliminates an autophagic process dysfunctional mitochondria in aging tissues increasing mitochondrial mediated apoptosis contributes to the percentage of apoptotic cells [70]. It is also noted that mergers and fission contribute to the accumulation of mutated mitochondria during aging because mtDNA is located near the oxidative phosphorylation complexes where most of the ERO [71] are generated. An important question to be answered and that is the main gap theory is to what extent changes in the structure and function of mitochondria are causes or consequences of aging [72].

Immunological theory: oxidative-inflammatory aging

With aging deregulation of the immune system called immune senescence, which predisposes people to frail elderly infections, cancer and decreased response to vaccines [73,74] appears. There is accumulating evidence that the deregulation of the innate immune system leads to increased production of inflammatory cytokines (TNF-α, IL-6 and others) that lead to a chronic inflammatory state of low grade has been termed the inflamm -aging (inflammatory aging) [75,76].

A strong correlation exists between inflammations and aging as is demonstrated in studies indicating that, on average, there is increased 2-4 times in serum levels of proinflammatory mediators in older individuals (> 50 years) compared to younger individuals. Also, people who have an unusual healthy aging (e.g. healthy centenarians) have a lower inflammatory profile than fragile centenarians [77] centenarians. Joining aspects of the theory of free radicals and chronic inflammation that underlies aging has proposed a new theory: oxidative-inflammatory aging (oxy-inflamm-aging) [78]. This new integrative variant provides that the oxidative stress associated with aging and generated ROS production in mitochondria mainly affects all body cells, especially those of (nervous, endocrine and immune) regulatory systems [79]. These systems, as a result, decrease its ability to preserve its redox state with functional losses incompatible with proper maintenance of homeostasis, distinctive physiological fact of aging (Figure 2). The oxy-inflamm-aging is one of the consequences of immune senescence and a frequent companion of fragility and pathological aging and is linked with major age-related diseases showing an inflammatory pathogenesis common as Alzheimer's disease, atherosclerosis, cancer, osteoporosis and frailty [80-83].

Non enzymatic damage

This general chemical theory refers to chronic, cumulative changes, not mediated by enzymes that increase the damage to biomolecules such as proteins and DNA, which may explain some manifestations associated with aging phenotype. These spontaneous reactions are that occurring between glucose and proteins known as glycosylation [84]. Glucose in its aldehyde form reacts with free amino groups of proteins to form Schiff bases and Amadori stable products. Other subsequent reactions involving ROS (hence the link with the theory of free radicals) generated more complex products, known as advanced glycation end products (AGP) Other non-enzymatic chemical damage in bio molecules can occur by oxidation, deamination, lipoxidation and cross links, among others [10].

Non-enzymatic modifications of proteins can alter the structure and often function. Although these systems can eliminate proteolytic modified proteins, it is demonstrated that the activity of these proteases declines with aging, leading to accumulation of damaged tissue proteins and protein aggregates [85]. These enzymatic changes are sufficient to explain how the glycosylation of proteins leads to the lens opacification and cataract formation [11]. Cross linking in the collagen proteins links mediated PGA leads to increase in rigidity of the connective tissue in blood vessels, tendons, and joint tissue with advancing age [86].

Improper repair the damage

For many years it was thought that aging is natural and inevitable consequence of wear and tear maintainer's mechanisms of life over time [87]. Many biogerontologists currently believe

that the agents causing damage during life are not really the cause of aging but this occurs as a consequence of progressive loss of ability to repair this damage [11,88]. Given the central role of DNA in life noted that insufficient reparations for these molecules may play an important role in the aging process to trigger cell signaling pathways such as apoptosis, which in turn results in cell depletion mother's physiological accelerating decline [89].

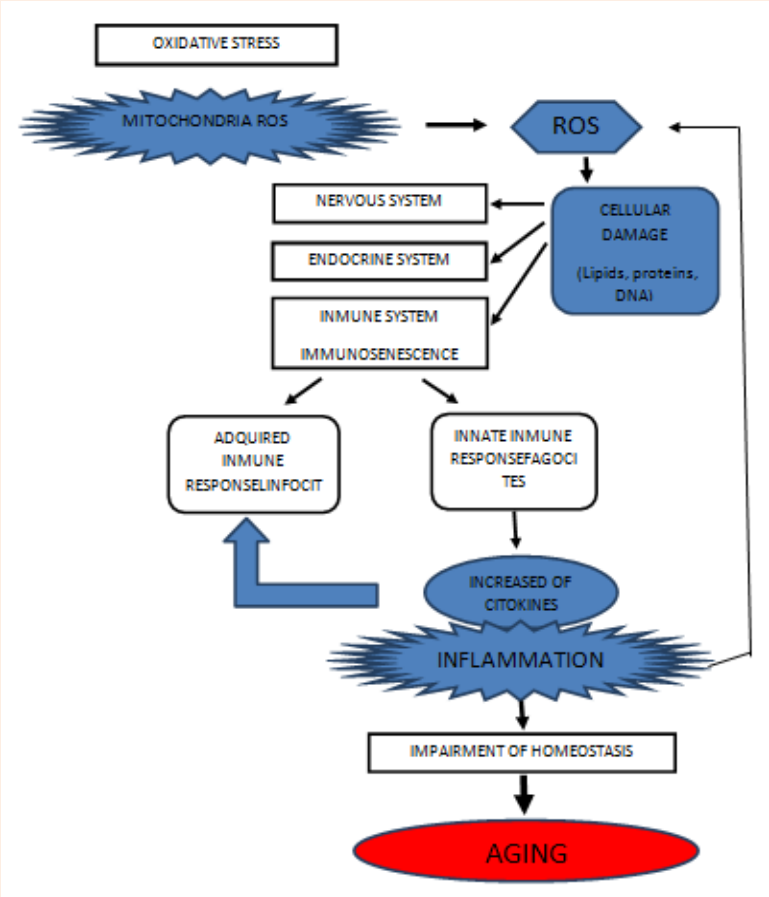

Figure 2: Theory of oxy-inflamm-aging.

Experiments in unicellular organisms predict that they must have active mechanisms to repair the damage before aging for segregation of it [90]. Genetic manipulations of DNA repair in mice support this view and indicate that the commitment of specific pathways such as repair and nucleotide excision of non-homologous end joining is associated with premature aging phenotypes [88]. In humans, it has been discovered that there are associated with defects in DNA repair and signs of premature aging as the xeroderma pigmentosum, skin cancer and progeroid syndromes [91] diseases. Critics of theories of wear and tear pose, however, that aging is not from a physical expectation but evolutionary. In support of this view point to experimental facts like that hyperactive mice live longer than controls and that worms with impaired antioxidant systems in their live longer than normal [92].

Deregulation in cell

Currently the thesis that one of the reasons for the aging of the whole organism is cellular senescence is supported. We age because cells that build and remodel our tissues develop senescence. Cellular senescence is defined as an irreversible cell cycle arrest by which tissues mitotic cells stop dividing and eventually die [77,93]. Recent data have demonstrated the in vivo accumulation of senescent cells in old age, so this approach [77] is reinforced.

50 years ago? Leonard Hayflick [94] experimentally demonstrated that human neonatal fibroblasts growing in culture have a finite life maximum [94], that is, have a limited number of cell divisions (about 60), after which die is and become senescent. This phenomenon is known as the "Hayflick limit" or replicative senescence [95]. The potential number of cell divisions is higher in long-lived animals suggesting a close relationship between the potential of cell division and longevity. Although the Hayflick hypothesis has been questioned in some quarters because only work for mitotic cells and not for mitotic postmitotic (muscle and brain), the results have provided the experimental basis for the understanding of other molecular and cellular mechanisms of aging [96,97].

Telomere shortening

Telomeres are gene sequences present at the ends of chromosomes that are responsible for maintaining genome integrity. Telomeres theory postulates that normal somatic cells have a finite maximum life and lose telomeric DNA with each cell division as a function of aging as found in vitro [98] studies. The length of telomeres is high at birth and gradually decreases with age so that shortening is considered as a biomarker of chronological age [99] advances. For many researchers this seems to be the explanation called "Hayflick limit".

Models in mice deficient in telomerase - an enzyme that prevents shortening of telomeres - have shown that these chromosomal structures dysfunction impairs the ability of tissue renewal and shortens the life of the animals [100]. The limits telomere shortening activating cell proliferation check points inducing replicative senescence and apoptosis. When telomeres become dysfunctional excessive shortening, p53 / pRb pathway that limits the maximum life proliferative cell [101] is triggered.

At present, opinions vary from those who believe that the telomere shortening is the only explanation of aging [102,103] to those who criticize this theory to consider that there is not a proportional relationship between telomere length and biological age because in the meta-analysis studies the association of this parameter with mortality decreases with age [104].

Apoptosis and theory of stem cells

Since the initial description of apoptosis, defined as programmed cell death, described other mechanisms of cell death, such as autophagy. Alterations of these mechanisms and signaling pathways may play a role in aging and diseases related to it [105,106]. Adult stem cells from mammalian tissues are essential for tissue homeostasis and repair throughout life, by replacing damaged or senescent mesenchymal progenitor cells [107]. As age advances, highly regulated, necessary to ensure proper cell homeostasis molecular signals, tissues and organs lose coordination and leads, as a result, the potential compromise of regeneration and repair of cells, inducing mechanisms of senescence and cell death scheduled [108].

The aging of fabric made from post-mitotic cells such as skeletal muscle could be partly explained by apoptotic mechanisms. In sarcopenia - decreased skeletal muscle mass associated with aging - proteins associated with apoptosis are increased and has been found apoptosis inducing inter mitochondrial factor located in the membrane of muscle cells (109). Cellular linked deregulation has been with other theories of aging. In this regard, it is suggested that the oxidative damage to DNA (free radical theory, inflammatory oxidative aging) can lead to cell death and senescence through the p-53 protein, which in turn leads to the induction of p-21, which is involved in caspase activation, key enzymes in apoptosis [110].

Cognitive theories

The development of the life of a human being is conditioned, importantly, for brain development. The brain processes related to cognition and behavior have been classified into general cognitive functions, including memory, learning, modulating moods, constructive and abstract thinking ability among others are, and function social cognitive, cognitive activities consisting of social relations, fundamental attribute of man as a specie [111].

Cognitive theories of aging relate more to how cognitive functions are presented in aging at why deficits are present in this area. In this regard based on a general cognitive theory called theory of mind. The theory of mind refers to the ability to represent the social mental state, i.e. the ability to attribute the wide range of mental ourselves states and others already use these attributes to have a proper sense helping to predict behavior [112]. A recent model distinguishes three subcomponents of social cognition: cognitive component itself, the affective component and empathy, a kind of bridge that interacts with these two. Research suggests that there is a decline in social cognition with normal aging, which is increased in the presence of neurodegenerative diseases such as dementia and Parkinson's disease. This decline appears to be independent of that which occurs in the general cognitive functions [113].

It has been shown that with long-term aging and a decrease in size of brain structures and the integrity of white matter occurs a decline in the speed of information processing, memory, working memory. Faced with these declines, functional brain imaging studies have found, surprisingly, compensatory increases in prefrontal activity that correlate well with better behavioral performance in the elderly [114].

This has given rise to state Scaffolding Theory of aging and cognition, which states that frontal activity increased with age, represents a scaffold (or frame) compensatory brain, most likely caused by a mechanism of neuro plasticity in response to the challenges posed by the decline of the neural structures and function [115] thereof. This process is a protective scaffolding cognitive function may explain the fact that some elders who die without apparent deterioration of their mental functions have, at necropsy, similar to Alzheimer's disease neuro pathological findings, which aim to the existence of a "cognitive reserve" in these people.

Psychological theories

Studies that have addressed the elderly from the social dimension focus their attention on issues related to cultural patterns, forms of social organization, attitudes and values in their historical development are giving old age and aging social meaning [116]. Psychosocial theories of aging have been happening over time and according to the historical development and the context where they were laid (many in industrialized countries), most of which were not disclosed, although some can retain nuances applicable in the today. Thus, an article that reviewed the theories used more frequently in social gerontology found 1046 between 1990-2004, in which, 39% mentioned theories [117].

Some authors divide the history of psychosocial theories of aging in three periods since the 2nd World War: a classic period (1940-1970), a modern period (1970-1990) and a postmodern period (after 1990) [118]. In this review only briefly discuss some theories of the first two periods and focus our attention on the postmodern period, stage focuses primarily on the construction of the definition and attributes of successful aging through gerotrascendence [119].

Between classical and modern period include the following (9)

a) Role Theory (1940) postulates that the loss of social role generates identity crisis, causing isolation, and demoralization and decreased self-esteem in older people.

b) Theory of decoupling (1960) argues that as we age, with retirement, the company facilitates the separation of the individual causing a decrease in the interaction between the elderly and the social system. A retirement is equivalent to widowhood in elderly.

c) Activity Theory (1963): It has a more optimistic view since it assumes that the social integration of the elderly is positively related to life satisfaction and successful aging.

Although there are many challenges to operationally define successful aging, from a psychological point of view there are accumulated evidence that optimism, resilience, positive attitudes toward aging and spirituality are associated with lower risk of morbidity and mortality in the elderly [120].

The postmodern psychosocial theory comes closest to the construct of successful aging is the Theory of gerotrascendence This was developed by sociology Swedish professor Lars Tornstam in the 90s and establishes that human development is a process that lasts a lifetime and remains in old age and, when optimized, ends in a new perspective. The gerotrascendence focuses on both the elderly person as in the aging process, describing the experience of growing old and characteristics of a normal and positive aging [121,122].

This theory, provide a key to understanding the development of the aging process and the transition to old age concepts, provides the possibility of structuring the attention and care for the elderly medical equipment and nursing. In this regard were generated to assess the gerotrascendence instruments that have demonstrated reliability and validity [123,124] and guide practical use for elderly care by nurses [125,126]. Despite being attractive and useful theory is causing controversy and generated a large volume of literature. A key question would be: Is the gerotrascendence really age-related or dependent on culture and personality? [127]. As you see, is not done deal.

Conclusions

The causes of aging are not yet fully clarified, what is revealed through the formulation of countless theories discussed throughout the different eras through which passed the gerontological science though, we could say that more than theories, there are numerous aging mechanisms. The spectrum of these mechanisms varies from micro to macro levels molecular and psychological integration and social behavior of human beings.It is impossible to ignore the influence of genetic, epigenetic, psychosocial and environmental factors and their inevitable interaction, so it is necessary to consider as a whole the overlapping of many of these mechanisms to obtain a holistic view of the aging process. Taking into consideration the theoretical, methodological, experimental and practical aspects that were exposed in this review will allow straighten the long and winding road that opens in the broad field of research to design and implement new strategies for successful aging.

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