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Review Article

Imbalance in Circulating Hormones May Accelerate the Aging of Key Internal Organs in Man

Gambhir Kanwal K [1,2]*, Bernard Rehkia [2], Morgan Gabrielle [3], Fluitt Maurice [1], Nunlee-Bland Gail [2]

[1]Professor, Director Molecular Endocrinology lab, Division Endocrinology and Metabolism, Department of Medicine, Bldg. HU Hospital, 2041 Georgia Ave, N.W., Washington, DC 20060.

[2]Diabetes Treatment Center, Howard U. Hospital, 2041 Georgia Ave, N.W. Washington, D.C. 20060.

[3]Honors, Undergraduate Pre-med Student, Howard University College of Arts and Sciences, 2400 6th St NW, Washington, D.C. 20059.

***Corresponding Author:** Gambhir Kanwal K, Professor, Director Molecular Endocrinology lab, Division Endocrinology and Metabolism, Department of Medicine, Bldg. HU Hospital, 2041 Georgia Ave, N.W., Washington, DC 20060

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Abstract

The objective of this review article is to provide a distinct overview of the vast literature suggesting that the imbalance in circulating hormones accelerates the aging of internal organs. Aging is described as an inevitable process that affects all living organisms, often accompanied by a decline in cellular function. By understanding the role of hormones in the process of aging, this article hopes to identify their effects on key organs in human systems which in turn lead to age-related diseases, namely Alzheimer's, breast cancer, cardiovascular disease, stroke, etc. These hormones are transported throughout the blood stream to their target organs or tissues to trigger a response to stimuli and maintain internal homeostasis. Previous studies have shown that an imbalance in the concentration of these hormones over time causes changes in bodily organs, aging them biologically, highlighting a contrast between human chronological age and the biological age of organs. This article aims to show that these differences in biological versus chronological age of key organs are because of the imbalances in hormones which often result in catastrophic effects on the human body. By identifying these hormones that affect the aging process of organs, future scientists would be able to facilitate improvements in the treatments of age-related diseases.

Key words: circulating hormones; internal organs; accelerate ageing; biomarkers; kidney; heart; lungs; liver; gastrointestinal track; biological age; chronological age

Abbreviations

ATP: Adenosine Triphosphate

BUN: Blood Urea Nitrogen

DNAm: Deoxyribonucleic Acid Methylation

EML: Epigenic Mutation Load

FSH: Follicle–Stimulating Hormone

GH: Growth Hormone

GHRH: Growth Hormone-Releasing Hormone

HRT: Hormone Replacement Therapy

IGF-1: Insulin-like Growth Factor 1

IGF-1R: Insulin-like Growth Factor 1 Receptor

LH: Luteinizing Hormone

PAX8: Paired-box Gene 8

PAS: Periodic Acid-Schiff Stain

PTH: Parathyroid Hormone

TGF: Transforming Growth Factor

T3: Thyroxine

T4: Triiodothyronine

TH: Thyroid Hormone

TSH: Thyroid Stimulating Hormone

Introduction

According to the World Health Organization, by 2050, the number of people age 60 and over will have doubled, making it increasing likely that age-related illnesses will become more common causes of death than seen

in previous years. An understanding of the aging process can help further our interpretation of disease and death. Identifying the biological age of the isolated organs can help reveal the association between age-related changes in metabolic hormones and their effects on other parts of the body. A multi-omic study suggests that there may be systemic clocks that overlay organ-specific counterparts [1]. Biomarkers, classified into nine categories by body system, were used for the generation of biological age and studied for patterns of correlation with chronological age. The biological age of the renal and sex hormone systems had the highest correlation. Sex hormone age was also associated with the renal and immune systems. The generation of biomarkers to classify signs of aging may also be useful for understanding how the aging of one system affects another, with further applications when considering the effects of lifestyle modifications through diet and exercise. In this paper, we review current literature on the metabolic and hormonal changes that occur during aging, with emphasis on the subsequent effects on vital organs including: the heart, kidney, lungs, GI tract and liver. This paper also examines the specific effects that change in the concentrations of hormones cause on these key organs, as well as their respective organ systems, and how this contributes to the health effects seen as aging gradually occur. Table 1 summarizes the organs which secrete and produce the various hormones.

Organ (Shape Description)	Hormones
Hypothalamus *(Funnel-shaped)*	Growth Hormone-releasing Hormone
Pituitary gland (anterior/posterior) *(Pea-shaped)*	Thyroid Stimulating Hormone, Growth Hormone
Thyroid gland *(Butterfly-shaped)*	Thyroxine (T4), Triiodothyronine (T3), Calcitonin,
Parathyroid Gland *(oval-shaped)*	Parathyroid Hormone (PTH)
Adrenal glands *(Triangular)*	Cortisol, Aldosterone, Epinephrine, Norepinephrine
Pancreas *(Flat leaf-shaped)*	Insulin, Glucagon
Pineal Gland *(cone-shaped)*	Melatonin
Liver *(Wedge-shaped)*	IGF-1, Calcidiol (Vitamin D precursor)
Stomach *(J-shaped)*	Gastrin, Ghrelin
Small Intestine (duodenum, jejunum, ileum) *(Long, coiled tube)*	Secretin, Cholecystokinin (CCK), Gastric Inhibitory Peptide (GIP), and Motilin
Kidneys *(Bean-shaped)*	Erythropoietin (EPO), Renin, Calcitriol (active Vitamin D)
Ovaries *(Almond-shaped)*	Estrogen, Progesterone
Testes *(Oval-shaped)*	Testosterone
Placenta *(Disc-shaped during pregnancy)*	Human Chronic Gonadotropin, Progesterone, Estrogen
Thymus *(bilobed-shaped)*	Thymosin, Thymopoietin

Table 1: A Representation of the Hormone Secreted from each of the Endocrine Glands *

**The information was derived from references number 26 and 27*

Hormonal Changes During the Aging Process

Changes In Growth Hormone Secretion During Aging

Growth hormone (GH), also referred to as somatotropin, is a peptide hormone secreted from the pituitary gland. It is directly responsible for an increased susceptibility to insulin resistance, lipolysis, and stimulating muscle growth. Growth hormone is secreted in a pulsatile fashion from the pituitary gland, with levels peaking at mid-puberty and declining by 50% every 7 to 10 years [2]. Insulin-like growth factor-1 (IGF-1) release is stimulated by growth hormones and acts as negative feedback. The decline of GH seen in aging is caused by a reduction in the amplitude of secretory episodes. Contrastingly, in the elderly, serum growth hormone concentration still rises at night as compared to young study subjects [2]. There is a parallel decline in serum IGF-1 levels and GH secretion as aging occurs. The lower output synthesis of growth hormone during the aging process is correlated with an increase in total body and visceral fat and declines in estrogen and androgen concentrations [2].

At present, there is no approved therapy to treat the reduction of growth hormone levels associated with aging. A single-center observational study on the effects of long-term growth hormone replacement in growth hormone deficient patients of all ages showed a decrease in waist circumference, waist-height ratio, and hip circumference in the adult group. It was concluded that the reduction in these parameters was to reflect the effect of GH on limiting central body fat deposits that occur with age [4].

Changes In Testosterone During Aging

Testosterone is the male sex hormone that is produced in the testes as part of the hypothalamic-pituitary-gonadal axis. Its effects are largely related to male reproductive health - androgenization, sexuality, and fertility. Testosterone levels are impacted by aging but have not been attributed to any progression of the aging process, though reproductive health has been linked with general health [2]. The recent studies on testosterone and aging are largely aimed at understanding the effects of the aging process on spermatogenesis and implications on children born to parents with advanced age. Increased paternal age and the changes that come with it carry an increased risk of infertility and impaired offspring health.

Changes in Thyroid Hormone Secretion During Aging

Thyroid hormones produced by the thyroid gland and stimulated by thyroid-stimulating hormones released from the pituitary gland, are the main metabolic peptide hormones. There is an inverse correlation between thyroid hormone (TH) levels, specifically T3 and T4, and longevity studied in different mammalian species and is a proposed biomarker of healthy aging and metabolic fitness [5]. Recent studies have largely been aimed at understanding the correlation between thyroid hormone levels and longevity, instead of the ways thyroid hormones fluctuate because of the aging process. In a study of thyroid hormone modulation and its effects on health and welfare in mice models, results were consistent with human studies that associate decreased life expectancy with higher levels of thyroid hormone [5]. Mice with mild hypothyroidism were found to have increased mitochondrial dysfunction and oxidative stress, two processes that are commonly found in aged tissues.

Changes in Estrogen During Aging

Estrogen is regulated through the ovarian axis and is related to the aging of the ovaries. Aging of the human ovary is predetermined for midlife senescence, unlike the other endocrine axes. The decline in ovarian follicle number ends with menopause, at which the menstrual period ceases. Aside from its widely known function in the reproductive system, estrogen also plays a role in maintaining bone density. The decline in estrogen levels seen in post-menopausal women contribute to the frailty

associated with old age, increasing the risk for osteoporosis and bone fragility.

Hormone replacement therapy (HRT) has been studied and used clinically in postmenopausal women to treat some associated symptoms of estrogen deficiency seen in advanced age. A London-based post hoc analysis of a randomized clinical trial on the effects of HRT on bone density showed that women with estrogen replacement-maintained collagen in the intervertebral discs and upregulated glycosaminoglycan synthesis that maintains water content in the discs. It was concluded that estrogen administration was associated with increased intervertebral disc heights, with implications on the increase in disc collagen and water content [6]. This effect could be partially responsible for reducing the risk of vertebral fractures.

Organ Specific Changes in Aging

Thyroid Hormone Control and Liver Aging

To understand the correlation between thyroid hormone levels and longevity, mice with hyper- and hypothyroidism were studied for their lifespan and health status. Hypothyroid mice demonstrated increased insulin resistance, hepatic steatosis, and increased chance of developing hepatocellular carcinoma. This information is consistent with studies that conflate the PAX8 gene mutation resulting in hypothyroidism with "development of hepatocellular carcinomas in Asian and Non-Hispanic white cohort [5]. Mice that displayed mild hypothyroidism did not live longer in comparison to wild type mice. Oleic acid was increased in the hypothyroid mice, consistent with the previous findings that show increased fatty acid intake promotes hepatoma progression. The mice also showed enhanced markers of mitochondrial beta oxidation, and there was evidence of altered antioxidant response and net accumulation of oxidative damage [4]. The study proposes the mice as appropriate models to study the mechanisms and effects of hypothyroidism.

The Aging Heart

Aging of the cardiac system promotes structural and functional dysregulation that leads to the development of various cardiovascular pathologies. Understanding what endocrine changes during aging may contribute to these changes may help outline what biomarkers can be used to accurately assess biological age as well as chronological age [20].

Insulin-Like Growth Factor and Effects on Cardiac Aging

Reduced activity of IGF-1 (insulin-like growth factor 1) is understood to extend the lifespan of model organisms and exhibits negative feedback on the somatotropic axis to regulate growth hormone-releasing hormone (GHRH), growth hormone, and IGF-1 [7]. Normal IGF-1 levels are shown to be protective against inflammation and endothelial damage [8]. It is primarily produced in the liver via stimulation from growth hormone and minds to IGF-1 receptors (IGF-1R) to exert its effects on organ growth [8]. It has a role in the cellular growth and metabolism of almost all organ systems, but there is conflicting evidence on the effect on age-related, non-proliferative pathologies. There is a peak of IGF-1 in the teenage years and a decline with age that is variable, related to fat mass, biological sex, hormonal status, and diet [8].

Recent studies aimed at defining the relationship between IGF-1R signaling and cardiac health and lifespan further indicate the possible biphasic effect of IGF-1R signaling in a lifespan. While it was previously understood to be a linear relationship, late-in-life treatment with IGF-1R monoclonal antibodies has been shown to improve cardiac function in female mice. In this study, the beneficial effects of overexpressed IGF-1R signaling were lost by 12 months of age in mouse models. When measuring autophagy and mitochondrial oxidative capacity, there was greater accumulation of autophagic substrate and autophagy-related lipidated form of a microtubule-associated protein that points to "either increased formation or reduced degradation of autophagosomes" [10]. Autophagy is linked to mitochondrial function, and it was found that mice IGF1R overexpression led to mitochondria with lower amounts of ATP, meaning there was impaired oxidative capacity and increased oxidative stress. There was also an increase in glycolytic intermediates including lactate in the myocardium that suggests increased anaerobic metabolism. Spermidine, an autophagy inducer, has been confirmed to prevent the IGF1-induced suppression of autophagy. Treating aged IGF-1R-overexpressed mice with spermidine improved multiple parameters of cardiac dysfunction, further suggesting autophagy as a factor in the cardiac dysfunction seen in this group [11]. When studying the translation potential to aged human hearts, there was no increase in IGF-1R expression in hypertrophic hearts as compared to control hearts. However, there was a nearly 2-fold increase in IGF-1R expression as compared with normal controls and non-failing hypertrophic human myocardium [10]. Overall, age was proposed as the determinant of the cardiac effects of IGF-1R signaling so that inhibition of cardiac IGF-1R signaling in late life likely suppresses the biological effects of cardiac aging, particularly those due to autophagy and mitochondrial dysfunction.

The Aging Liver

The aging process generally comes from impaired metabolism and accumulated oxidative stress that then manifests as a decline in physiologic function of an organ. In the liver, there is a deterioration of the liver function that is linked to systemic susceptibility to other age-related disease processes [12]. The liver produces most of the glutathione responsible for maintaining redox status. Aged hepatocytes produce a higher level of inflammatory cytokines that lead to increased reactive oxygen species-mediated activity. Interventions aimed at promoting healthy liver aging would be important in reducing the oxidative damage that speeds up aging.

The Aging Kidney

In a study on male rats aimed at characterization of age-related markers of kidney function, blood pressure and heart rate were not significantly different between young (3 months) and aged (24 months) rats. Serum BUN, uric acid, and glucose were among the conventional markers of kidney function. Total urinary albumin increased in the aged rats, but the total urinary protein levels did not differ between young and old rats. PAS-positive areas of the extracellular matrix of the glomeruli were "significantly increased" in 24-month-old rats. Interstitial fibrosis was shown in aged rats and was supported by the upregulation of inflammatory cytokines, including TGF-alpha and TGF-beta. It was determined that markers of inflammation and urine metabolites were the most promising proposed markers of kidney aging in human models [12].

Effects Of Diet and Exercise

Caloric Restriction and Cardiac Senescence

Understanding the role that diet, and exercise can play in promoting healthy aging will help define some of the measures of aging to be studied. Maintaining a healthy diet has demonstrated positive effects on metabolism and physiologic functioning. In a study of caloric restriction and its effects on cardiac aging in obese diabetic rats, it was found that caloric restriction increased the expression of markers of cardiac senescence (e.g. IGF-w) and improved myocardial degradation [14]. While the mechanism is still unknown, caloric restriction reduced the oxidative stress associated with aging and increased telomerase activity. These benefits are thought to be associated with improvement of diastolic dysfunction in the hearts of diabetic rats.

Age And Physiological Function in Active Older Adults

The relationship between age and physiologic function is of increased interest but misunderstood. Because of the large number of confounding factors, it is difficult to define parameters that accelerate or decelerate aging. A cross-sectional study aimed at removing these confounding factors suggests that the relationship between function and physiological age has many interrelated factors, but that physical activity should be

among the factors taken into consideration [15]. Genetic variation and lifestyle choices are among the variables to be taken into consideration to determine the relationship between age and function. A group of highly active men and women were studied and proposed as a viable model for healthy aging. The correlations found in this study were statistically significant, however none of the parameters studied were determined to be reliable markers that could consistently predict an individual's function at a given age [15].

Biomarkers Of Aging Slowed in Diet and Exercise Intervention Trial

Epigenetics based on DNA methylation is one proposed biomarker of aging that is increasing in accuracy. A study analyzing diet and exercise effects on aging biomarkers in healthy postmenopausal women showed evidence of a causal association with lifestyle-modification and the decline of DNA-methylation (DNAm) biomarkers. Higher consumption of fruits and vegetables was associated with slowed DNAm measured aging, while consumption of processed meats demonstrated an unfavorable increase in the epigenetic mutation load (EML). The lifestyle changes of improved diet quality and increased physical activity, specifically increased physical activity, lead to decrease of EML's. This study was conducted with women, so any differences attributed to gender cannot be assessed [17].

The imbalanced hormones that cause accelerated aging of the key internal organs are listed in table 2.

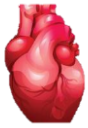
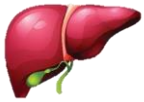
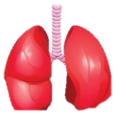
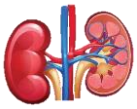
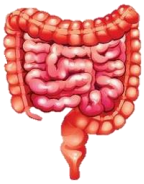
Key Internal Organ	Imbalanced Hormone
Heart 	Progesterone, Thyroid Hormones, Cortisol, Parathyroid Hormone, Sex Hormones
Liver 	Estrogen, Testosterone, Thyroid Hormones, GH, IGF-1
Lungs 	Glucocorticoids, Sex Hormones, Ediponectin, Leptin, Insulin, Thyroid Hormones
Kidney 	LH, FSH, GH, Testosterone (free), Estrogen, Calcitriol, Erythropoietin, PTH, Complex interplay of several hormones
Gastrointestinal Tract 	Sex Hormones, GH, IGF-1, TH, CCK, Leptin, Ghrelin

Table 2: A Representation of the Specific Imbalanced Hormone that Impacts Vital Organs during Aging in Man

Discussion

It is important to acknowledge that the endocrine organs responsible for producing hormones are controlled by other hormones, both of which are impacted by aging. For example, an endocrine tissue may produce less of its hormone than it did at a younger age, or it may produce the same amount at a slower rate. It is important to note that imbalance in hormonal levels can contribute to the accelerated aging of key internal organs through various mechanisms. Hormones are powerful signals, and an imbalance, particularly decline in key hormones, are closely linked to an acceleration in cellular aging. A sharp drop in estrogen production during menopause has been seen to accelerate biological aging in women [17]. A recent that explores the effect of estrogen deficiency and aging on organismal homeostasis during menopause provides an excellent viewpoint on overall women's health [34].

The markers of aging differ in the internal versus external environment of man. This difference is seen between the biological age of organs, some of which age slower than others due to lifestyle influences, among other factors and the chronological age which is fixed [21]. The dynamics of biological processes during aging are also because of the difference seen in organs since they do not age simultaneously and as a result of these biological functions have become more closely associated with the organ's pathological age, rather than the chronological age of the human [22].

This review clearly demonstrates that the imbalance in hormones accelerates the biological aging of key internal organs in the human body. The antiaging gene, sirtuin1, is important to the prevention of accelerated aging and diabetes seen in humans. Sirtuin 1 inhibitor causes *insulin resistance and reduces plasma Sirtuin 1 levels. Chronic exogenous*

*hyperinsulinemia might lead to the suppression of Sirtuin*1 activity [32, 33]. The study also clearly demonstrates that imbalances in insulin result in dysfunction of pancreatic tissue [33].

Conclusion

Hormonal changes represent a central mechanism through which aging exerts its effects on human physiology. The decline in anabolic hormones such as GH, IGF-1, melatonin, estrogen, and testosterone, alongside alterations in thyroid function, contribute to functional impairments across all organs. These endocrine changes intersect with inflammation, mitochondrial dysfunction, and metabolic stress, further accelerating the aging process. These functional impairments have been linked to a reduction in muscle mass and age-related insulin resistance, a contributing factor to the increase in type 2 Diabetes seen in older populations [19]. Importantly, lifestyle modifications, specifically improved diet quality, stress reduction and increased physical activity, have shown potential in mitigating these hormonal shifts and their downstream consequences. Continued investigation into the interplay between hormones, organ-specific aging, and modifiable behaviors holds promise for developing targeted interventions and reliable biomarkers to promote healthy aging and improved quality of life in later years.

Author Contributions

Author Contributions

Dr. Kanwal Gambhir conceived this idea, redrafted the original draft and edited the final draft. Rekhia Bernard drafted the original draft of the manuscript to fulfill the requirements for her MS IV elective. Gabrielle Morgan prepared the pre-final draft under the supervision of Dr Gambhir. Dr Maurice Fluitt and Dr Gail Nunlee-Bland edited the final draft as well.

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