# The Aging Process and Hormones

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## Introduction

The ebbs and flows of hormones around the body play an important role in maintaining the circadian rhythms of life as well as the body's ability to regulate energy metabolism and cope with internal and external stresses. Hormone production, efficacy, and clearance all change as we age. The smooth, oscillatory release of hormones, in particular, has changed to a more chaotic pattern of release. This, combined with a decrease in the function of hormonal receptors and their post receptor effector mechanisms, frequently results in a sluggish response of the endocrine system. When the reserves of the ageing endocrine system are pushed to their limits in older people, the decline in function is often more pronounced.

Brown-Sequard pioneered the idea that endocrine system failure may play a role in the physiological changes associated with ageing in the nineteenth century. He believed that testicular failure was the cause of ageing and performed a series of experiments on himself, injecting testicular extracts to reverse the ageing process. Although the outcome was most likely a placebo effect, his reported findings led to the world's wealthy elderly paying large sums of money for injections of testicular extracts, human testicular transplants, monkey-gland transplants, and goat testicular transplants. This concept was extended to women in the 1950s with the publication of Wilson's book, "Feminine Forever," which suggested that oestrogen could delay the ageing process. Victor Horsley proposed in 1886 that older people resembled myxedematous monkeys and that thyroid hormone deficiency could result in "mere senility." Dan Rudman developed the concept of somatopause more recently, ushering in a new era of growth hormone replacement therapy for the wealthy elderly. Other hormones, such as melatonin and Dehydroepiandrosterone (DHEA), have also been promoted as "hormonal fountains of youth." Denckla, on the other hand, proposed the existence of a pituitary hormone he dubbed the "death hormone." According to his theory, an excess of this hormone causes accelerated ageing. Unfortunately, these science fiction origins of ageing endocrinology still have some sway today, as evidenced by many of the mythological, quasiscientific claims of some antiaging industry adherents.

The role of hormones in hip fracture prevention and the effects of diabetes mellitus on function in older people will be investigated. The role of hormones in aging-related changes in energy metabolism is an important emerging area for explaining the proclivity of older people to develop malnutrition.

## Fratility and Hormones

Androgens and growth hormone are anabolic hormones that are linked to muscular mass and strength in young people. As a result, it is reasonable to believe that, as they deteriorate with age, they may play a role in the loss of strength and muscle mass that occurs with ageing, and thus play a role in the development of frailty syndromes.

Cross-sectional epidemiological studies have found that testosterone and, to a lesser extent, Insulin Growth Factor-1 (IGF-1) play a role in the loss of muscle mass and strength as we age.

Numerous cross-sectional and longitudinal studies have shown that testosterone declines with age in men, with an even greater decline in free or bioavailable (free plus albumin bound) testosterone due to an increase in Sex Hormone Binding Globulin (SHBG). Testosterone replacement therapy increases muscle mass and strength in hypogonadal men, according to interventional studies.Testosterone also helps to reduce body fat and boost bone mineral density. There is evidence that testosterone contributes to the cognitive decline associated with ageing. Testosterone decline has been linked to functional impairment in older men. Testosterone replacement therapy has been shown to improve the functional index measure in older men undergoing post-hospitalization rehabilitation. Overall, these findings lend credence to the idea that testosterone decline may play a role in the pathogenesis of frailty in older men. However, there have been insufficient long-term replacement studies to back this up. Furthermore, the potential risks of testosterone replacement therapy on the prostate and cardiovascular system have yet to be determined.

Total testosterone level is an insufficient measure of tissue-available testosterone because of the increase in SHBG with ageing and changes in its binding characteristics. As a result, it is necessary to measure either free testosterone (as measured by dialysis or ultracentrifugation techniques) or bioavailable testosterone (as measured by the ammonium sulphate precipitation technique). The measurement of free testosterone using a commonly available analogue assay technique has been shown to be an ineffective test.

To make a diagnosis of hypogonadism in men, it is recommended that men be screened for symptoms first, and only if symptoms are present, should a measurement of free or bioavailable testosterone be taken. The St. Louis University Androgen Deficiency in Aging Men (ADAM) questionnaire is a well-known symptom checklist. Although it correctly identifies the majority of men with hypogonadism, it has a relatively high non specificity rate. The most common cause of a false positive is the presence of depression. Depression should be treated before testosterone levels are measured. A false-positive ADAM result can also be caused by hypothyroidism.

Women's testosterone use is more contentious. Between the ages of 20 and 40, women's testosterone levels decline. During menopause, oestrogen replacement lowers free testosterone levels even further.

Only a few studies have shown that testosterone replacement therapy improves libido, muscle mass and strength, and bone mineral density in menopausal women. At this time, the long-term effects of testosterone replacement therapy on women are unknown. Tibolone, an estrogenic-progestogenic-testosterone agonist, appears to improve muscle strength and quality of life. It has a good safety profile and is widely used in Europe.

Overall, the role of anabolic hormones in frailty prevention or treatment is, at best, unproven. Most studies have been of poor quality, and belief has driven the literature far more than data has.

## Hormones, Energy Metabolism and The Ageing Process

It is now widely accepted that there is a physiological decline in food intake over the course of a person's life. This is referred to as "ageing anorexia." Hormones may play a significant role in the pathogenesis of this physiological anorexia of ageing, according to recent research. This ageing physiology is a major reason why older people have a greater proclivity to lose weight when they have a disease than younger people.

Older people are more likely to become satiated early in life. There are two reasons for this. Aging causes a reduction in stomach fundus compliance. This happens because food does not cause an adequate release of nitric oxide to relax the fundus smooth muscles.

When the fundus fails to relax, the reservoir becomes smaller, allowing food to enter the antrum more quickly. Antral stretch sends signals to the central nervous system that the stomach is full, causing the person to stop eating.

Cholecystokinin (CCK) is a gastrointestinal hormone produced in the duodenum in response to fat. CCK is a powerful satiating agent. Older people produce more CCK in the duodenum, both basally and in response to fat. The increase in CCK is primarily due to slower clearance in older people than in younger people. When given to elderly people, CCK is more satiating than when given to younger people. As a result, CCK represents a second reason why older people develop early satiation in response to a meal.

Adipose cell signals influenced appetite in part. The peptide hormone leptin is the primary hormone involved in this. Leptin is anorectic and increases metabolic rate. As a result, it's a catabolic hormone. TNF, a cytokine produced by adipose cells, is also anorectic. As a result, as fat mass increases, anorectic messages to the central nervous system increase in an attempt to curb eating. However, as fat tissue accumulates, it produces substances such as triglycerides, which result in resistance to the leptin effect.

Men experience more physiological anorexia as they age than women. Men's fat mass decreases after the age of 70. This would be expected to cause a drop in leptin levels, but a long-term study found that leptin levels increase in older men. The rise in leptin levels was linked to the decline in testosterone levels that occurs with age. In older men, testosterone replacement therapy reduced leptin levels. As a result, it appears likely that leptin plays a role in the ageing anorexia seen in older men.

Ghrelin, a peptide hormone, was recently isolated from the stomach fundus. Ghrelin stimulates the release of growth hormone and increases feeding. Ghrelin or an analogue appears to have potential as a treatment for ageing anorexia.

## Thyroid

Aging causes a decrease in thyroxine production rate, which is offset by a slowing in plasma clearance rate, resulting in unchanged circulating thyroxine levels. Triiodothyronine (T3) levels have decreased slightly.

Thyroid Stimulating Hormone (TSH) levels are unchanged, but 2.7 percent to 3.5 percent of older men and 7.1 percent to 17.6 percent of older women have early hypothyroidism. 32 Thyroid peroxidase antibodies rise with age. There is emerging evidence that as people age, thyroid hormone becomes less capable of activating a variety of postreceptor biomarkers of thyroid function. The supersensitive TSH assays are less sensitive in older people, making their use for diagnosing hyperthyroidism problematic. Thyroid hormone excess and deficiency are two classic examples of atypical disease presentation in older people. Apathetic thyrotoxicosis affects about 7% of older people and is characterised by blepharoptosis (hooded eyes), weight loss, anorexia, atrial fibrillation, congestive heart failure, proximal muscle weakness, and depression. Autoimmune thyroiditis is the most common cause of hypothyroidism in older people. Early symptoms and signs of hypothyroidism are frequently indistinguishable from nonspecific manifestations seen in elderly people with nonthyroid disease. Thus, in the elderly, early hypothyroidism is a biochemical diagnosis.

Low T3 syndrome, also known as euthyroid sick syndrome, is common in the elderly. T3 levels in these people are significantly lower, and thyroxine levels may be normal or low. TSH levels can fall, stay the same, or rise. There is no evidence that thyroid replacement improves outcomes in this condition.

Because of the slower plasma clearance rate, older people often require a lower thyroid replacement dose than younger people. Physicians are frequently unaware of this and fail to adjust thyroxine doses appropriately in elderly patients. Older people with hypothyroidism and dementia may be resistant to treatment and require intramuscular thyroid hormone replacement.

## Conclusion

Multiple hormonal changes occur as we age. They frequently occur gradually, and their relationship with the functional changes associated with ageing is unknown. The possibility that certain hormones, such as testosterone, may slow the development of sarcopenia and frailty syndromes is of great interest in geriatric research. A typical manifestations of hormonal disorders in the elderly frequently present diagnostic challenges to the geriatrician.

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