

Applied Kinesiology and the Factors and Biomechanics of Androgenic Alopecia: A corrigible Look

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Narrative: Baldness and balding are epidemic problems that affect both sexes at varying ages on almost all levels of being. The subject is a large one. Understanding and correction require the identification of balding types by category, among many things. These include categories related to stress, improper nutrition, hormonal imbalances, and, of late, gravity, and COVID-19—a type of stress related hair loss. The types of baldness by common diagnostic name include *androgenic alopecia* which includes male pattern balding (MPB), female pattern balding (FPB), and receding hairline. Then other types such as *alopecia universalis*, *alopecia areata*, *traction alopecia*, and *central centrifugal cicatricial alopecia* (CCCA). In the last few years, we have identified causes of hair loss related to dysfunction of the stress system by way of adrenal, thyroid, and pituitary dysfunction, nutrient deficiencies, chemical toxicity and poisoning of the scalp and hair follicles, emotional trauma, and the focus of this paper, gradual biomechanical aberrations. It appears that when gradual biomechanical aberrations are identified and addressed, and if stem cells in the hair follicle are properly stimulated and supported, hair regrowth and the prevention of androgenic alopecia may be possible. Applied Kinesiology tools applied to the cranium and hair follicle allow the identification of causes and the creation of a care plan with a foundation in the restoration of sound cranial biomechanics and function. This means that for the management of improved scalp and hair health to be successful and long lasting, it must include correction and basic maintenance of the biomechanical structures of the cranial vault, osteo-myofascial system, and remaining stomatognathic system.

Indexing terms: Chiropractic; Applied Kinesiology; Male Pattern Balding; Androgenic Alopecia; Subluxation; Manual Muscle Test.

Introduction

Unanticipated hair loss affects untold numbers of the population of the world and the USA. There are many causes and types of hair loss. The causes include the more obvious gross nutrient deficiencies, traumatic stress, and then, more ambiguous and incomplete, are the various explanations for male pattern and female pattern baldness.

This paper will orient, arrange perspective, as well as validate work already performed on these two subjects and advance them to a new level not seen before, using tools we already own.

... To restore full normal function to the hair unit requires a panoply of factors for restoration and stabilisation of a weak and ailing hair units and the scalp they reside in. But this can be done ...'



Jargon relating to hair loss

Bone remodelling is the process of bones changing size, shape and sometimes growth. Holographic sublaxation is a shape based sublaxation that does not match the nervous systems expectation of shape and moulding, the process of changing shape of the skull intentionally or not. Growth includes:

- ▶ vellus hair as fine hair
- ▶ lanugo hair as baby hair
- ▶ anagen as the first stage of hair shaft development and the growth period
- ▶ catagen as the disruption of the growth process and end of hair shaft growth and nourishment
- ▶ telogen as the resting phase of a hair follicle
- ▶ telogen effluvium as the expulsion of a hair shaft from the follicle
- ▶ Androgenic alopecia as hair loss based on elevated androgens
- ▶ miniaturisation as thinning of hair so that a weak and fine hair shaft grows
- ▶ dihydrotestosterone from reduced testosterone

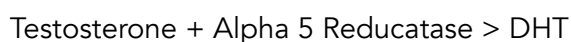
The superficial Musculoaponeurotic system (SMAS) is the fascial system in the head and neck that includes the *platysma* muscle superior to the clavicle rising to the superficial temporal fascia.

Background and preparation

A web search on the topic of hair loss provides millions of results. From gels to lotions to surgery and implants to wigs, hair is often on many people's minds.

Hair loss affects 80 million Americans, with approximately 60% of men and 40% of women suffering from it. The most prevalent type among the 80 million is androgenic alopecia (AA) (American Academy).

This type includes male and female pattern balding. (Skin Conditions by the Numbers, n.d.) In 1987, a seminal study was done on scalp Dihydrotestosterone (DHT) elevation on bald areas of the scalp of men suffering from male pattern balding (MPB). We know that testosterone irreversibly converts to dihydrotestosterone: (Caballero)



Male pattern balding by convention since 1987 was stated to be related to an elevated level of scalp DHT and scalp 5-alpha reductase, the enzyme that converts testosterone to DHT, which causes 'susceptible hair follicles' to produce weak hair shafts and then produce none at all. Blood tests for DHT in one study showed no difference between control and studied subjects. To date, there are no blood tests that accurately correlate blood androgen levels with balding. (Donovan 2014)

Steroids

The common agreement and background information is that androgens have been determined to target specific hair follicles that have more receptors for 5-alpha reductase and DHT, and more of these are located in the frontal lobe than the occipital. (Price 2003) This is the area of male pattern baldness.

These androgens are well documented to also be responsible for secondary sex characteristics such as the following in men:

- ▶ Pronounced body hair characteristics (beard, chest, etc.) and is usually coarser.
- ▶ Heavy musculature

- ▶ Angular features (i.e. square jaw, triangular mid region)
- ▶ Narrow hips
- ▶ Muscular pectorals (chest)
- ▶ Overall, there is less fat tissue.
- ▶ Deeper voice

In women, these secondary sex characteristics include:

- ▶ Less pronounced body hair characteristics (mostly in pubic region, hair all over is usually finer)
- ▶ Lighter musculature
- ▶ Rounded features (i.e. softer facial features, hourglass mid-region)
- ▶ Wider hips (for child bearing)
- ▶ More pronounced breasts with more fatty tissue
- ▶ More fat tissue overall
- ▶ Higher voice

The following androgens also play a role in hair growth:

- ▶ Testosterone creates hair growth and secondary sex characteristics that differentiate men from women.
- ▶ Cortisol is a stress and everyday adrenal hormone that is elevated during stressful times and is catabolic to all tissues. Chronic exposure is also catabolic and gene expression in many cells can change from an acute exposure as an anti-inflammatory to a chronic exposure that stimulates nuclear genomic increases in levels of NfKb, making the inflammation epigenetic or a chronic product of genetic expression instead of acute local irritation. Cortisol may also work in a feedback loop with DHT. Elevated cortisol levels cause local increases in DHT levels as these are meant to lower the cortisol (Toufexis).

Gravity

Another hypothesis proposed in a 2008 paper is that hair loss is a direct product of the forces of gravity. According to this new theory, the pressure created by the weight of the scalp is the cause of male pattern baldness (USTUMER 2008).

Genetics

Genes are thought to play a role in AA. The problem in each person is thought to be polygenic and a perfect storm between endocrine imbalances and polygenetic factors. Two genes have been identified. We do know that epigenetic factors can modify genetic expression. Improper nutrition has been put forth as a major factor in some types of baldness; not solely in the case of AA.

Phases of hair growth

At birth, or in an anorexic adult, we see lanugo hair. This hair eventually falls out after birth and is replaced by fine hair called vellus hair. Vellus hair is present until puberty. After that, a terminal or adult hair replaces it.

Parts of the scalp hair unit

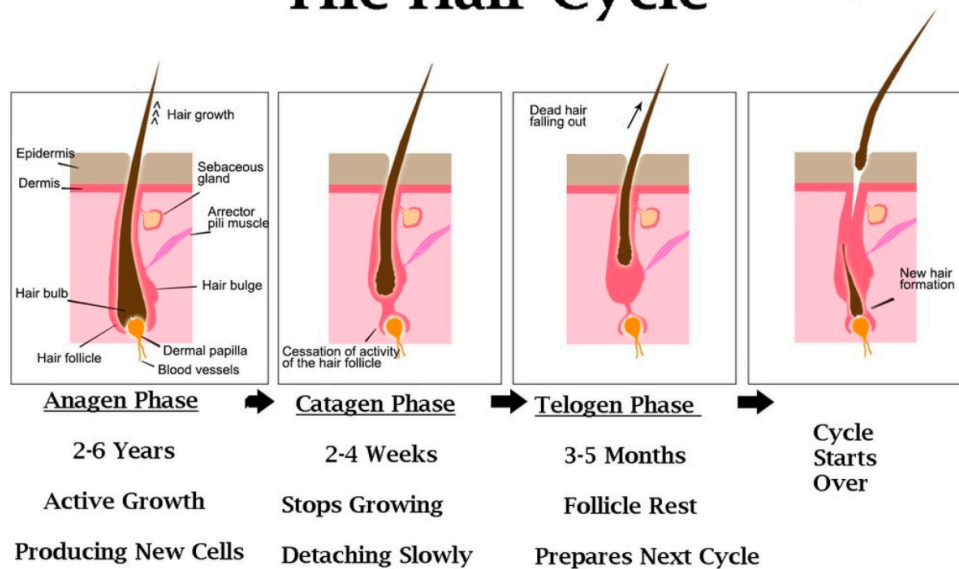
Hair is composed of a shaft, 'the hair', which grows from a follicle that has germ cells at the bottom of the follicle that receive blood from blood vessels in the underlying tissue.

Phases of terminal hair growth

Terminal hair growth occurs in stages of follicle and shaft activity. Four of the most basic stages are listed below: (Kiesewetter, 1990)

- ▶ Anagen, lasts 2-7 years and represents the growth of the hair shaft and how long it can become
- ▶ Catagen, the hair shaft shrinks and detaches from its nutrient source. It has a 10- day duration and can be gradual or acute and is impacted by cortisol.
- ▶ Telogen is the unit's resting phase. The shaft of hair is now detached but still in the follicle. The stem cells have made or are resting but can make a new hair shaft. At any given time, 10-15% of the hair is in this stage. This can last three months or longer.
- ▶ Exogen is still in the resting phase, but new hair will begin to grow, and the detached hair will leave the follicle and be shed.

The Hair Cycle



(The Hair Growth Cycle, n.d.)

Current treatments for androgenic alopecia

The current treatments for hair loss include:

1. Wigs/ barber's synthetic creations.
2. Surgery –via various implant methods with an unpublished success rates.
3. Minoxidil, Finasteride or alpha-5 reductase blocker medications.
4. Low level laser therapy to increase circulation.

A review of the skull, scalp, cranial blood supply, myology

The human cranium is composed of 29 separate flexible membranous bones, postulated and objectively measured to have movement at the joints or sutures between them. These movements are governed by factors such as muscular contraction, attachments and reciprocal tension membrane forces from inside the skull as well as fascial forces from local and remote locations of the human muscle and skeletal system.

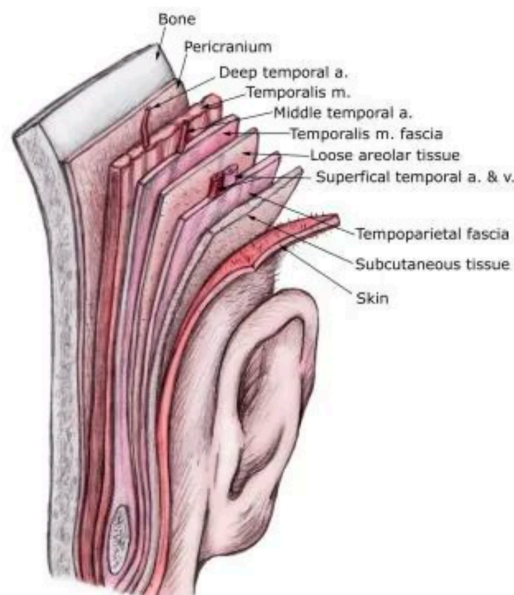
Depending on how they are counted, there are about 30 muscles on each side of the human skull. There are 20 muscles in the neck that directly attach to the skull and the cranio-cervical,

superficial and deep fascia, as well as the superficial muscular aponeurotic system (SMAS). (Whitney 2021) The fascial ties are common to the head, neck, and the entire body as one connected unit. Another fascial connection is the aponeurosis that covers the calvarium called the *galea aponeurotica*; this flat tendon is the insertion of two opposing muscles, the *frontalis* and *occipitalis*, also known as the *occipitofrontalis*. On either side of the skull is a location for the *temporalis* muscle.

Traveling through this vast network of muscles, bones, and fascia are the arteries that supply these structures. The scalp's blood supply arrives via the superficial temporal artery, a branch of the external and internal carotid arteries. The carotid arteries were named by Galen based on the Greek word for stupor, as he found when you compressed them, one could go into a stupor.

The scalp, which tops the *calvarium*, is a network of adipose tissue, skin, and hair follicles and sits on top of the *galea aponeurotica*.

The scalp layers from bone *calvarium* to the skin and hair progress as illustrated below;



Discussion

A novel pathogenesis model for male pattern baldness

The male pattern balding process from start to finish takes approximately 5-25 years to complete. The process is not an acute problem but chronic and progressive for most; stress chronically causes disease, but it is also episodic for most. The hair loss pathogenesis is from terminal hair to vellus to lanugo to bald and then dormancy.

Working backwards from a bald scalp to a healthy scalp with a full understanding of wholism, we see that the end result of hair loss is a dormant or weakly producing hair shaft from a hair follicle that still has stem cells in it.

The 'swan song' of a dying hair follicle

The dormant hair follicle once had a miniaturised hair shaft; we called this vellus hair. The hair follicle reacted to elevations of cortisol, and the cortisol caused the hair follicle to be weaker and smaller. This in turn caused a reactive elevation of DHT, which is used by the body to lower cortisol. Lower cortisol levels allow the hair follicle to strengthen or start a new but this does not happen and so elevation of DHT is the swan song of catabolised hair follicles, not the root cause of

hair loss-the small hair, now in catagen and doomed, will shed eventually in the exogen phase. Perhaps more rapidly if the levels of cortisol do not decrease. Now the question is, what increased the cortisol?

What triggered catagen, cortisol

We know that elevations of cortisol, either acute or chronic, can throw a hair follicle into catagen leading to hair loss. Stress, shock or trauma do this acutely but usually that happens with the loss of patches of the scalp.

Chronic stress causes the adrenal glands to increase their cortisol output to deal with various types of stress. One such type is structural stress. Structural stresses may present as abnormal cranial bone movement, myofascial dysfunctions and adhesions. Then changes in the shape of bones from years of cranial bone remodelling create a holographic mismatch, leading to increased stress and insidious increases in cortisol, decreases in blood supply and functional hypoxia for the scalp via the superior temporal artery. As this process insidiously happens, silently and slowly, so too does the loss of the terminal hair, propelling it into catagen status. (S 2020)

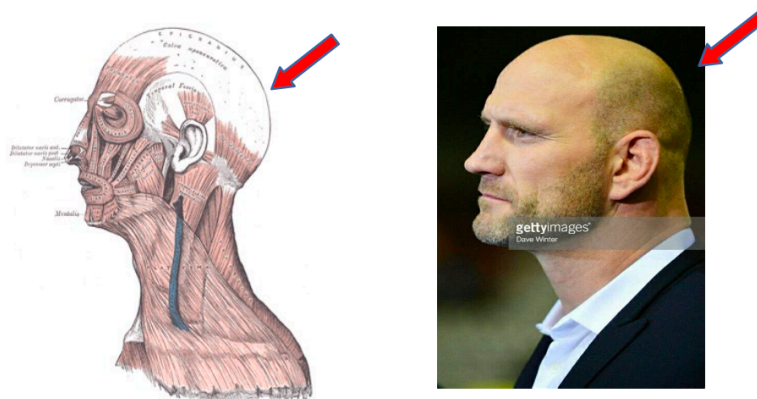
The causes of cortisol excesses are best reserved for future works but includes non- optimal interactions with, structural, chemical, thermal, emotional and geopathic and electromagnetic stressors. Chronic exposure may increase cortisol levels.

Hair follicle miniaturisation is complicatedly the product of chronic excess cortisol exposure and slow deprivation of oxygen and nutrients in the case of androgenic alopecia (AA). (Toufexis 2012)

The years of hypoxia also cause muscle dysfunction, which creates fascial distortions that lead to chronic but low force hypertonicity and imbalance; and as Dr. George Goodheart used to say, 'Muscles do move bones.' And so, Wolff's law takes effect to make the problem chronic. According to this law in part, a bone in a healthy animal will adapt to the load under which it is placed. Hence, we see actual moulding of the skull. (Wolff 1892)

We start to get growth and shape changes in the cranial bones as a result of persistence and consistent myofascial tension, imbalance, and muscle dysfunction. These osseous changes cause changes in the tension of the galea which cause further fascial distortions and further decreases in blood supply and resultant diminished nutrition to the scalp.

As a result, cortisol secretion is stimulated, miniaturisation of more follicles occurs, and DHT increases to neutralise the cortisol, and we start over again, year after year; thinner and thinner. The 'gravity theory' too has been in play all the while. Except the pulling is the myofascial system, not the gravitational forces of the planet. The end product is loss of hair, male and female pattern balding, and distortion of the shape of the cranial bones. (Graham Kennedy, 2013) Oddly and overlooked is the fact that the balding of male pattern balding is almost in pattern with *galea aponeurotica* in many individuals.



The illustration compared to the photo demonstrates galeaic hairloss in the male pattern balding fashion.

Well before the above scenario was probable, cranial imbalances and deviations from normal cranial respiratory movement were an issue. This caused a decrease in blood support through increases in tension and myofascial dysfunction from chronic and acute stress.

These structural stresses that affect cranial movement and scalp mobility and nourishment may occur anywhere in the human body, but most commonly in the neck and shoulders.

Correction

Now that we have identified the pathogenesis. Where do we start on the correction? At the end of course! In any situation where damage is being done you need to:

1. Get to a safe place. in this case we need to identify areas of cortisol stimulation and start addressing them. This may be very individual. (i.e. overwork, overtraining, emotional situations unhandled, etc.)
2. Stop the damage. The hair follicle has been deprived of vital blood, oxygen and nutrients. It may be using anaerobic glycolysis to survive. The end product is lactic acid. Some people notice this because of soreness, itching and burning. That means that NAD is being used for this process so testing for Niacin not niacinamide is a first step to stopping damage.
3. Next to control damage we must discourage the local testosterone conversion by 5 alpha reductases. We can use frequently applied essential oils and topicals for this.
4. Check each of the 29 bones of the cranium for holographic subluxations.
5. Check for cranial faults and sutural faults.
6. Release the *frontooccipitalis* muscles.
7. Evaluate all muscles of the skull for proprioceptive dysfunction.
8. Evaluate the entire somatagnathic system.
9. Evaluate fascial balance and for adhesions using the midline scalp pull test.
10. General fascial release per fulford may be achieved by using percussion at the following locations:
 1. left and right deltoid tuberosities
 2. left fibular head,
 3. C2, L2 and S2
11. Evaluate and release the sacrum and coccyx.

Now that you have addressed the various structural concerns all of the following nutrient areas need to be assessed and corrected:

12. Scalp cortisol challenge
13. Zinc status
14. B3
15. Methylation status – homocysteine protocol (Schmidt)
16. Essential fatty acid blood spot profile
17. Vital amino acids needs must be assessed and met.
18. Cranial remoulding exercises must be performed three times daily. Cranial moulding exercise may cause cranial faults so the cranium should be re-evaluated to ensure no harm has been done. These exercises must be prescribed based on the holographic imbalance in each cranial bone identified (e.g. parietal bulge is most common)
19. Calvarial muscles must be evaluated for any types of dysfunction

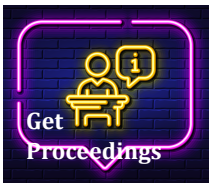
20. Each muscle of the facial skull muscle be evaluated for dysfunction
21. Topical and internal use herbs that improve blood supply are adjunctive and needed.
22. Adrenal function must be assessed and addressed per the AK Synopsis.

Conclusion

Thirty-five years prior to this paper, a discovery was made that described, not the cause but the end result of one process of hair loss. *Dihydrotestosterone* elevations are the end result and hair unit 'swan song' from years of blood deprivation and all of the life-embodying factors that it carries to the tissues and cells of the scalp.

Applied kinesiology allows the pieces of this puzzle to be found and arranged in a fashion that allows us to see the big picture of the puzzle completed. To restore full normal function to the hair unit requires a panoply of factors for restoration and stabilisation of a weak and ailing hair units and the scalp they reside in. But this can be done by those willing to put forth a little work and effort.

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References

1. Donovan JD. (2014, August 31). Should you get a blood test for dht. Donovan Hari Clinic. Retrieved August 31, 2014, <https://donovanmedical.com/hair-blog/dht>
2. Miller R, Clarren S. Longterm developmental outcomes in patients with deformational plagiocephaly. *Pediatrics*, 105(2). 2000.
3. Photo taken from www.londonorthotics.co.uk/plagiocephaly/locband.html which is the website for the London Orthotics Consultancy which specializes in the production and fitting of these helmets.
4. Aksoy H, Yildirim UM, Ergen P, Gürel MS. COVID-19 induced telogen effluvium. *Dermatol Ther.* 2021 Nov;34(6):e15175. DOI 10.1111/dth.15175.

5. Anawalt BD. (2017). Is Dihydrotestosterone a Classic Hormone?. *Endocr. Rev.* 38 (3): 170–72. DOI10.1210/er.2017-00091. PMID 28582536.
6. Caballero MJ, Mallol J. Physiopathological role of bald-scalp cytosolic proteins. *Rev Esp Fisiol.* 1987 Jun;43(2):229-37. PMID: 3498977.
7. Cicatricial alopecia. (n.d.) Miller-Keane Encyclopedia and Dictionary of Medicine, Nursing, and Allied Health, Seventh Edition. (2003). Retrieved March 12 2022 <https://medical-dictionary.thefreedictionary.com/cicatricial+alopecia>
8. Easley T. COVID-19 One Herbalist's Thoughts On The Coronapocalypse. <https://www.eclecticschoolofherbalmedicine.com/covid-19/>
9. Goodheart G. Fatigue and Its Management.
10. Goodheart G. 'Hiatus Hernia - The Great mimic.'
11. Gray H. *Anatomy of the Human Body* 1918 2H. The Large Intestine
12. Grymowicz M, Rudnicka E, Podfigurna A, Napierala P, Smolarczyk R, Smolarczyk K, Meczekalski B. Hormonal Effects on Hair Follicles. *Int J Mol Sci.* 2020 Jul 28;21(15):5342. doi: 10.3390/ijms21155342. PMID: 32731328; PMCID: PMC7432488.
13. Graham Kennedy GK. (2013, February 1). Positive Health Online | Article - Craniosacral Therapy, Cranial Moulding and Baby Helmets. Positive Health Online. Retrieved April 17, 2022, <http://www.positivehealth.com/article/craniosacral-therapy/craniosacral-therapy- cranial-moulding-and-baby-helmets>
14. Skin conditions by the numbers. (n.d.). American Academy of Dermatology Association. Retrieved April 17, 2022, from <https://www.aad.org/media/stats- numbers>
15. Taylor P.T. (n.d.). Why the Pattern in Male Pattern Baldness Develops. *Hair Growth SOS*. Retrieved April 17, 2022, <https://www.hairgrowthsos.com/male-pattern-baldness.html>
16. Slator W. (2021, September 14). 8 Causes of Slow Hair Growth & 13 Ways To Make Hair Grow Faster. *Hairguard*. Retrieved April 17, 2022, <https://www.hairguard.com/slow-hair-growth-causes/>
17. S. (2020, March 29). Does Skull Shape Change As We Age? *Reference.Com*. Retrieved April 17, 2022, <https://www.reference.com/science/skull-shape- change-age-db1b71d3d1026b18>
18. Kenkre JS, Bassett J. The bone remodeling cycle. *Ann Clin Biochem.* 2018 May;55(3):308-327. DOI 10.1177/0004563218759371. Epub 2018 Mar 4. PMID: 29368538.
19. Kenny AM, Raisz LG. Mechanisms of bone remodeling: implications for clinical practice. *J Reprod Med.* 2002 Jan;47(1 Suppl):63-70. PMID: 11829079.
20. Kiesewetter F, Schell H, Seidel C. Proliferationskinetik des menschlichen Anagenhaares bei Hyper- und Hypothyreose: DNS-impulszytometrische Untersuchungen [Proliferation kinetics of human anagen hair in hyper- and hypothyroidism: DNA flow cytometry studies]. *Z Hautkr.* 1990 Dec;65(12):1120- 2. German. PMID: 2087840.
21. Matsuo K, Ban R. Surgical desensitisation of the mechanoreceptors in Müller's muscle relieves chronic tension-type headache caused by tonic reflexive contraction of the occipitofrontalis muscle in patients with aponeurotic blepharoptosis. *J Plast Surg Hand Surg.* 2013 Feb;47(1):21-9. DOI 10.3109/2000656X.2012.717896.
22. Ndiaye A, Touré S, Diop FM, Ndiaye AS, Diallo BK, Sow ML. Artère temporale superficielle: typologie et application au lambeau de fascia temporal superficialis [The superficial temporal artery: anatomical type and clinical application to the flap of the fascia temporal superficialis]. *Morphologie.* 2004 Oct;88(282):155-9. French. DOI 10.1016/s1286-0115(04)98140-4. PMID: 15641654.
23. Price VH. Androgenetic alopecia in women. *J Investig Dermatol Symp Proc.* 2003 Jun;8(1):24-7. DOI 10.1046/j.1523-1747.2003.12168.x. PMID: 12894991.
24. Sudduth SL, Koronowski MJ. Finasteride: the first 5 alpha-reductase inhibitor. *Pharmacotherapy.* 1993 Jul-Aug;13(4):309-25; discussion 325-9. PMID: 7689728.
25. Taylor PJ. Big head? Bald head! Skull expansion: alternative model for the primary mechanism of AGA. *Med Hypotheses.* 2009 Jan;72(1):23-8. DOI 10.1016/j.mehy.2008.07.048. Epub 2008 Sep 11. PMID: 18789604.
26. Goodheart G. The Respiratory Function of The skull - collected reprints
27. Thom E. Stress and the Hair Growth Cycle: Cortisol-Induced Hair Growth Disruption. *J Drugs Dermatol.* 2016 Aug 1;15(8):1001-4. PMID: 27538002.
28. Toufexis DJ, Wilson ME. Dihydrotestosterone differentially modulates the cortisol response of the hypothalamic-pituitary-adrenal axis in male and female rhesus macaques, and restores circadian secretion of cortisol in females. *Brain Res.* 2012 Jan 6;1429:43-51. DOI 10.1016/j.brainres.2011.10.024.

29. Iland PM, McCann A, Midttun Ø, Ulvik A. Inflammation, vitamin B6 and related pathways. *Mol Aspects Med.* 2017 Feb;53:10-27. DOI 10.1016/j.mam.2016.08.001.
30. Urysiak-Czubatka I et al. Assessment of the usefulness of dihydrotestosterone in the diagnostics of patients with androgenetic alopecia. *Postepy Dermatol Alergol.* 2014 Aug;31(4):207-15. DOI 10.5114/pdia.2014.40925.
31. Wolff J. *The Law of Bone Remodeling.* Berlin Heidelberg New York: Springer, 1986 (translation of the German 1892 edition).
32. Ustuner ET. Baldness may be caused by the weight of the scalp: gravity as a proposed mechanism for hair loss. *Med Hypotheses.* 2008 Oct;71(4):505-14. DOI 10.1016/j.mehy.2008.05.030.
33. Walther DS. *Applied Kinesiology, Synopsis 2nd Edition,* ICAK-U.S.A., Shawnee Mission, KS, 2000, p. 494.
34. Walther. DS. *Applied Kinesiology, Synopsis 2nd Edition,* ICAK-U.S.A., Shawnee Mission, KS, 2000, p. 221
35. Whitney ZB, Jain M, Zito PM. *Anatomy, Skin, Superficial Musculoaponeurotic System (SMAS) Fascia.* [Updated 2021 Nov 21]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. Available from <https://www.ncbi.nlm.nih.gov/books/NBK519014/>
36. www.bartleby.com. 29 January 2011. <http://www.bartleby.com/107/249.html>