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## Introduction

Hi,

Thanks for downloading Skull Expansion – True Cause of Genetic Hair Loss.

I'm Paul Taylor and I wrote this ebook for anyone who's suffering from androgenetic alopecia.

You're about to read a very quick and easy guide to genetic hair loss, how I managed to completely reverse this alarming condition (as you can see from the photo) and **how you can do the same**.



In the short time it takes to read, you'll very quickly learn about:

- The **true** cause of androgenetic alopecia and how the hair loss industry has got it **wrong!**
- How techniques have been developed that can now restore strong, healthy scalp hair growth.
- How you can discover these very special techniques for yourself and finally put an end to your own hair loss.

You'll also discover many fascinating facts about hair and hair growth along the way.

I hope you enjoy reading "Skull Expansion – True Cause of Genetic Hair Loss".

**Feel free to send it to anyone you want - simply attach the ebook to your emails.**

(Help other people - let them know how skull expansion causes hair loss and how the hair loss industry got it wrong! Pass it on!)

Ok, let's start.

# 1. Androgenetic Alopecia

As its name suggests, androgenetic alopecia is a genetic form of hair loss that involves androgens (male sex hormones). However, it's also known as androgenic alopecia, AGA, male pattern baldness (MPB) and female pattern baldness. In this ebook, AGA is used. I also use the term "MPB region" - this refers to both male and female pattern baldness because each involves the same region of the scalp.

Whatever you call it (I used to call it "the bane of my life") AGA is a condition, not a disease - even though it might feel like one!

This chapter examines the current theory for AGA and explains why it's wrong!

[1.1 DHT, androgen receptor sites and 5-alpha reductase](#)

[1.2 Female pattern baldness](#)

[1.3 Problems, problems, problems](#)

## 1.1 DHT, androgen receptor sites and 5-alpha reductase

There's been much debate about the true cause of AGA. Several factors have been linked to it (genetics, hormones, nutrition, etc) and many theories exist that try to explain exactly what mechanism is taking place.

There's no doubt that androgens are the main blame - it's long been known that a derivative of testosterone called dihydrotestosterone (DHT) is directly related to this condition. But, exactly how DHT causes AGA is still unclear to the hair loss industry.

Most hair loss professionals believe that DHT chokes the blood supply to the follicles. (A follicle is basically a pouch through which the hair shaft grows).

Testosterone first gets converted into DHT by 5-alpha reductase (an enzyme produced within the scalp). DHT then attaches to androgen receptor sites in the follicles (a receptor site is an arrangement of molecules that binds to other molecules with a complimentary shape).

If DHT is produced in excess, and attaches to a large number of androgen receptor sites, it may accumulate within a follicle and block its blood supply. This forces it to prematurely enter the resting stage of the hair growth cycle.\*

\* The hair growth cycle - Anagen (the growth stage) normally lasts 3 to 5 years. Catagen (a 2 week shedding stage) is followed by telogen (the resting stage) lasting 3 to 4 months. Anagen then restarts.

If a follicle enters the resting stage earlier than usual, the time spent in its growth stage will obviously be reduced. This results in hair that becomes both shorter and thinner with each successive growth cycle, and also causes hair follicle miniaturisation. Eventually the follicle shuts down - it becomes dormant and hair growth stops.

If you ask almost any hair loss professional (trichologist, dermatologist, etc) for the mechanism behind AGA, that's what they'll tell you.

Convinced?

You shouldn't be - this theory has its problems!

I'll reveal all these problems shortly, but first of all, you might be wondering how hair loss involving **male** sex hormones can also affect women.

## 1.2 Female pattern baldness

Up to 50% of women will experience AGA to some extent during their lives.

The reason for this is simply because women produce a small amount of testosterone (mostly from their adrenal glands) <sup>1</sup>. Both men and women produce a small quantity of each others' hormones in this way.

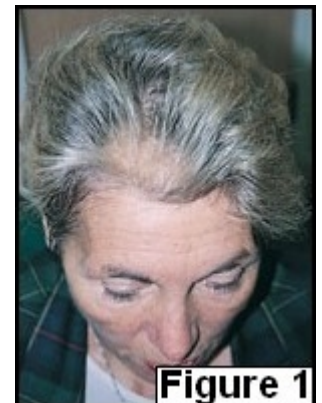
For most women, their oestrogen levels are usually high enough to completely overwhelm the quantity of testosterone they produce. And the opposite is true for men.

It's when oestrogen levels become low (e.g., due to the menopause) or there's an increase in androgen production (e.g., by stress, weight training, etc) that AGA can start to show itself on the female body.

In women, AGA usually appears as diffuse thinning (this is evenly distributed hair loss) on the crown of the head (see Figure 1).

The subject of AGA specific to women is further addressed in chapter 2.

Now, let's get back to the current theory for AGA.



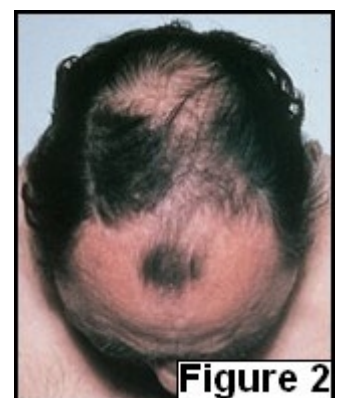
## 1.3 Problems, problems, problems!

To explain all the problems this current theory has, take a good look at Figure 2.

This photo shows a typical example of a man with severe AGA, and you can clearly see that the familiar MPB region of hair loss has almost fully developed.

This MPB region needs closer examination:

Ok, first of all, notice how his hairline has receded from the left and right temples, but **not entirely** from the centre front region. In some cases, this area keep growing reasonably strong, healthy hair throughout life despite extensive hair loss all around it. I call this, "remnant hair".



You can see more remnant hair trying very hard to grow within the centre crown area. And behind it, another significant area of hair loss has developed into a bald patch at the back of the head.

His baldness seems to have developed from the front (both left and right temples) and back of his head **independently** (i.e., two separate areas of hair loss). And, where these two areas of loss meet in the middle, some remnant hair continues to grow.

Notice how hair continues to grow normally on the sides and lower back of this head. This, of course, gives him that familiar “male pattern” profile, or horseshoe shape, synonymous with this type of hair loss.

Have you ever wondered why this pattern of hair loss always seems to emerge in nearly every severe case of AGA?

It's been observed that both the androgen receptor gene <sup>2</sup> and 5-alpha reductase <sup>3</sup> appear to be more active within the MPB region in those who suffer AGA. According to the current theory, this observation indicates that hair follicles within **just the MPB region** must be genetically programmed <sup>4</sup> for this to occur.

But this still doesn't explain why only hair follicles in this region should carry the AGA gene(s). In other words, it still doesn't give a reason why hair loss should be confined to the MPB region.

It's been acknowledged by the hair loss profession that the actual mechanism causing hair follicle miniaturisation within just the MPB region is still unclear. Furthermore, they accept that several genes may be involved and that some other mechanism must also be taking place <sup>5</sup>.

Another problem with this theory is that androgens are hair growth stimulators <sup>6</sup>.

That's right, androgens cause hair to **grow!**

The most obvious example of this is when pubic, facial and body hair starts growing during puberty, all of which is caused by androgens (especially DHT).

This fact has been pestering trichologists and dermatologists for years – DHT should help hair to grow, not hinder it. So there must be something else going on that's causing the follicles to shrivel.

No explanation has been given by the hair loss industry as to how DHT can cause both hair loss and hair growth.

Finally, DHT will **not** cause AGA in someone **without** the genetic tendency towards it (even in high concentrations).

All of this now raises several questions concerning AGA. Questions that **must** be answered before **any** theory can be recognised as the true underlying mechanism for this type of hair loss. The current theory **cannot answer** these very important questions concerning the hair loss process:

**Q1.** Why does remnant hair sometimes continue to grow within the MPB region despite extensive hair loss all around it?

**Q2.** What causes the same male pattern profile (horseshoe shape) to develop in almost all severe cases of AGA?

**Q3.** How can DHT be linked to both hair loss and hair growth?

**Q4.** What is the genetic connection to AGA?

**Q5.** Why does the rate of hair loss vary from person to person? (Up to 20% of men can suffer rapid hair loss starting as early as puberty. But most don't experience AGA until later on in life, and for them, this can be a much more gradual process).

**Q6.** Why does the location of hair loss vary within the MPB region? (Some people only lose hair from the front (temple recession) or back of the scalp (a bald patch), whilst others lose hair from both these regions simultaneously).

In chapter 2, I'll explain how skull expansion causes AGA, and provides answers to all these questions.



## 2. Skull Expansion Causes AGA

This chapter explains how skull expansion causes AGA, and by doing so, provides answers to all questions from chapter 1.

[2.1 Bone resorption and remodelling](#)

[2.2 Scalp capillary network](#)

[2.3 Remnant hair and development of the male pattern profile](#)

[2.4 Hormones and hair loss](#)

[2.5 Genetic link to AGA](#)

[2.6 Rate and location of hair loss](#)

[2.7 Re-evaluation of the current theory](#)

### 2.1 Bone resorption and remodelling

When you're growing up, your skeleton obviously gets bigger and bigger. This, of course, includes your skull, which not only grows in size, but also changes shape.

Once you reach adulthood, bone resorption and remodelling will continue to maintain skeletal integrity.

Together, these two essential processes constantly renew the entire skeleton throughout life. For some, this simply means maintaining the bones – keeping them strong and healthy. But, for those who suffer AGA, certain bones of the skull will continue to grow.

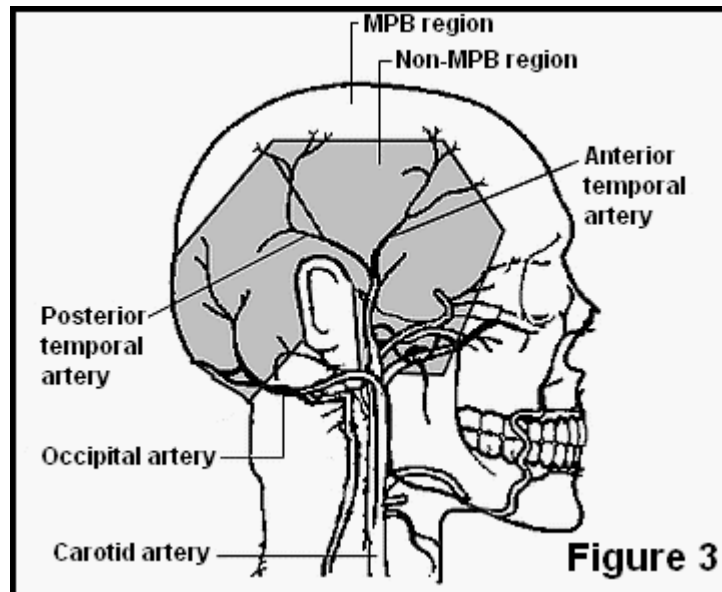
This is skull expansion, and is the direct cause of AGA within the MPB region of the scalp for both men **and** women. It can also be considered as an exaggeration of the bone resorption and remodelling processes.

### 2.2 Scalp capillary network

For all those with the genetic predisposition towards skull expansion/AGA, this process will progressively stretch and pull tight the scalp tissue that overlies the skull. This constricts the blood vessels which then reduces the blood supply.

Figure 3 shows how the **main arterial network** of the scalp will provide a strong blood supply to the back and sides. But, within the MPB region, the follicles are only served by a much **weaker capillary network** (not shown in the diagram). This illustrates how the MPB region is the most likely area of the scalp to suffer a reduction in blood supply.

Skull expansion will, within the MPB region, reduce the flow of blood and so decrease the supply of nutrients required by follicles to grow hair. In time, hair follicle miniaturisation and hair loss will result.



## 2.3 Remnant hair and development of the male pattern profile

If you compare [Figure 2](#) from chapter 1 (page 6) with Figure 4 below, you can see that the bones of the skull that underlie the MPB region are the frontal and parietal bones.

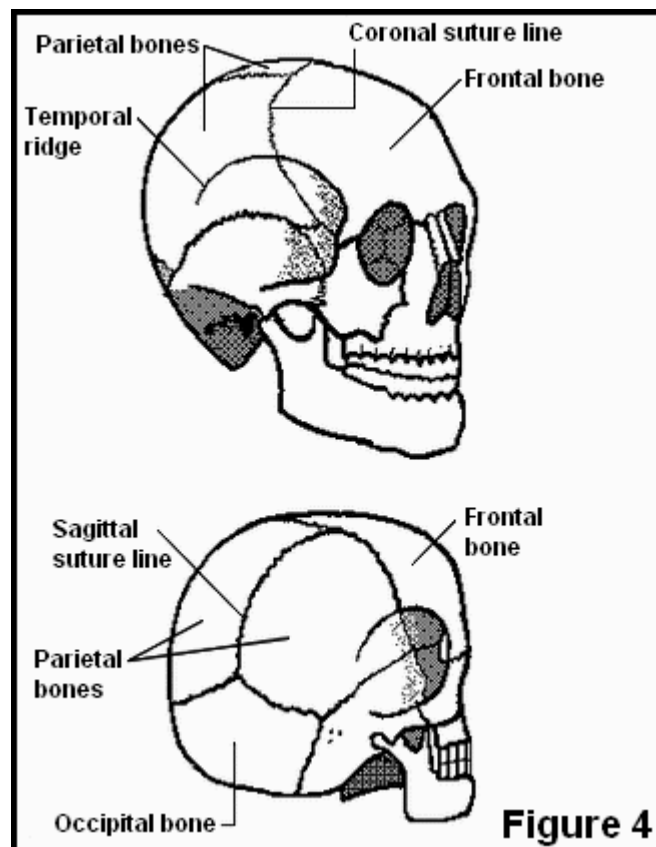
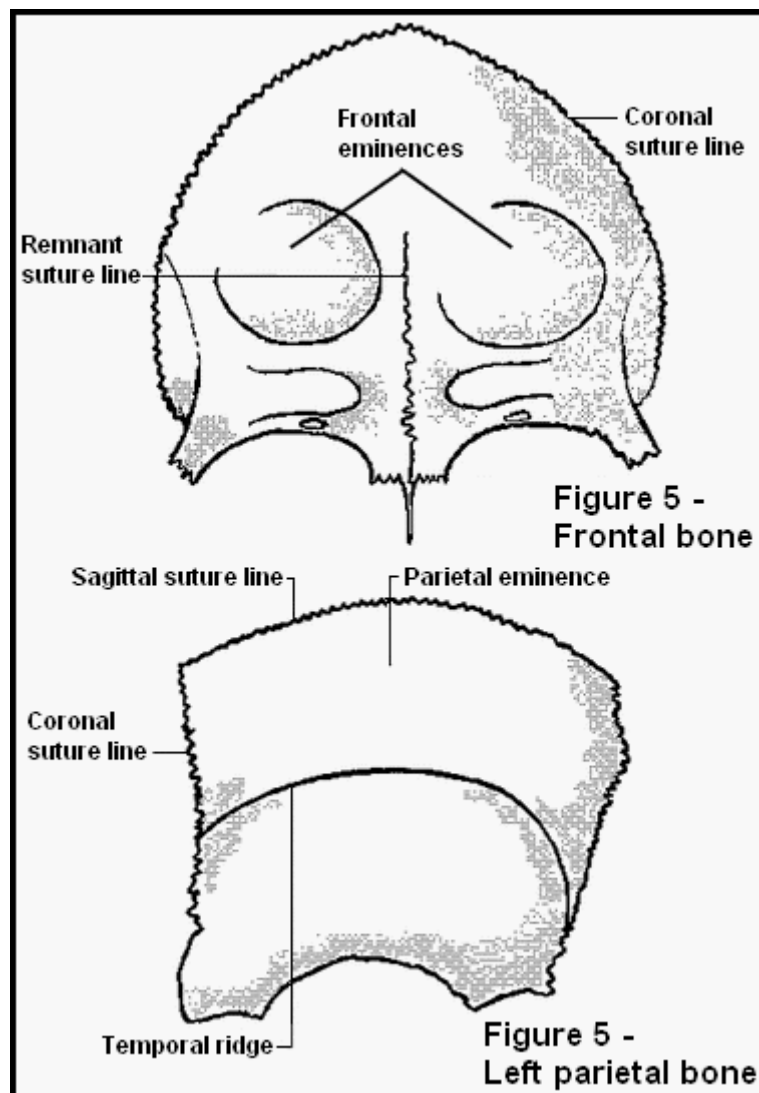


Figure 5 details these two bones. The frontal and parietal eminences represent the areas where ossification commenced (in other words, where bone initially began growing as a foetus within the womb).

From these areas, bone grows outwards, pushing against the surrounding bone tissue and so causing the frontal and parietal bones to grow larger in surface area. It's through this linear growth that the full adult skull size is eventually reached.



There are two parietal bones (left and right) so each one obviously has its own parietal eminence. But, you can also see that there are two frontal eminences as well. That's because the frontal bone actually starts out as two bones within the womb.

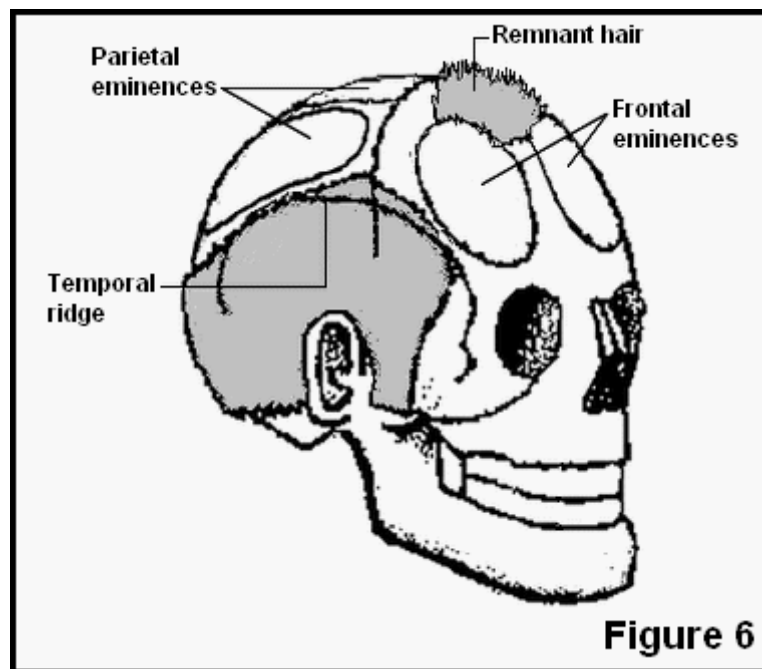
As these develop, they gradually fuse together and can leave a remnant suture line\* (as shown in Figure 5). This will usually disappear as a baby grows into a child, but the two frontal eminences remain.

\* Suture lines mark the connection between the bones of the skull.

Skull expansion of these two frontal eminences (left and right) will cause hair loss at the front hairline. This explains why, in most cases, hair recedes at the left and right temples and **not** across the entire front hairline.

It also explains why some remnant hair often continues to grow at the front - you can see this in Figure 2 and Figure 6 below. (Basically, remnant hair will continue to grow wherever there is no, or little, skull expansion).

This provides the answer to [question 1](#) in chapter 1.



In Figure 6 you can see how the frontal and parietal bones of the skull relate to the pattern of hair loss for someone with severe AGA.

Now, can you see how the temporal ridge seems to coincide with hair loss along the sides of the head?

Well, this is **no** coincidence!

Within each parietal bone, the parietal eminence (that causes skull expansion) only lies **above** the temporal ridge. So, it logically follows that hair loss can also only occur above this line. And, as a result, the temporal ridge marks an approximate boundary between hair loss and hair growth on the sides of the head.

Hair will always continue to grow at the lower back of the head because here, the occipital bone largely remains unaffected by parietal bone expansion. You can clearly see the occipital bone in [Figure 4](#).

By analysing of the structure of the skull (specifically the frontal eminences, temporal ridge and occipital bone), the familiar male pattern profile of hair loss can be explained.

This provides the answer to [question 2](#) in chapter 1.

## 2.4 Hormones and hair loss

Androgens, like DHT, can be linked to both hair loss **and** hair growth. DHT causes facial, body and pubic hair to grow in men during puberty, but it also has a direct connection with AGA.

How can this be?

DHT is a steroid hormone which means that, whilst it stimulates new hair growth, it also has an anabolic effect on bone formation <sup>7</sup>. In other words, it makes bone grow! And it's through this continued bone growth (skull expansion) that overwhelms the hair growth promoting effects of DHT.

This provides the answer to [question 3](#) in chapter 1.

For men at puberty, steroid hormones are responsible for the rapid increase in bone growth (and muscle development) they experience. This also explains the connection that exists between bodybuilding and AGA - bodybuilders often suffer hair loss because intense weight training will increase testosterone and DHT levels (as do anabolic steroids).

### 2.4.1 Sebum

Sebum is a waxy, oily substance secreted by the sebaceous glands within hair follicles. Its production is governed by androgens (including DHT) so it's easy to see the connection between DHT and sebum. As well as stimulating the skull expansion process, DHT can also contribute to hair loss locally (i.e., within hair follicles) through excessive sebum production. However, this is very much secondary to the skull expansion process.

### 2.4.2 Female hormones

Women generally experience AGA to a much lesser extent than men due to their much higher oestrogen and lower testosterone levels.

Whereas steroid hormones like testosterone and its derivatives have an anabolic effect on bone growth, oestrogen decreases the bone resorption process <sup>8</sup>. Since the oestrogen levels in a healthy pre-menopausal woman's body greatly exceed those of testosterone, there will be a reduced tendency towards skull expansion.

However, low (post-menopausal) oestrogen levels can often lead to AGA. That's because testosterone levels will be higher (relative to the lower oestrogen levels) and so the bone resorption, remodelling and skull expansion processes will all increase for those women with the genetic predisposition towards AGA.

So, for all those men and women with this genetic predisposition, DHT will cause the frontal and parietal eminences to grow during adulthood. This is the true underlying mechanism behind AGA.

But, why do some have the genetic predisposition towards skull expansion/AGA whilst others do not?

## 2.5 Genetic link to AGA

It's long been known that a genetic link to AGA exists.

Most research currently places focus on the 5-alpha reductase and androgen receptor genes. However, it's the genes that determine skull **shape** and **size** that are responsible for AGA.

### 2.5.1 Sexual dimorphism

Sexual dimorphism <sup>9</sup> means that the form (shape, size, etc) can vary between the two sexes. For example, women usually have wider hips, whilst men are generally taller and heavier (due to bigger bones and greater muscle development).

Skull expansion is a largely sexually dimorphic characteristic, affecting men much more so than women. Men will, quite simply, grow a bigger skull than most women. (This reflects the higher androgen levels men have, as well as differences in genetic inheritance). More specifically to AGA, this means that, for most women, the frontal and parietal bones will be proportionately smaller than in the majority of men.

### 2.5.2 Growth potential

Associated with skull shape and size is its growth potential. This simply means that some skull shapes are more likely to grow than others.

For example, someone whose skull shape has a high growth potential will be especially prone to skull expansion, and so invariably develop severe AGA. And it follows that, anyone else with a **very similar** skull shape and growth potential will most likely develop AGA to the same extent.

Of course, very similar skull shapes often run in families, and you may already be aware that, if your mother or father lost their hair, you too have a very high chance of losing yours as well. (This explains the strong genetic connection that AGA can have within a family, especially between fathers and their sons).

You now know that the genetically determined characteristics of skull shape and size form the genetic link to AGA.

This provides the answer to [question 4](#) in chapter1.

## 2.6 Rate and location of hair loss

Skull shape and its growth potential can account for the different rates of hair loss, and the location (within the MPB region) in which it occurs.

### 2.6.1 Rate

The rate at which you lose hair is directly related to the extent of your skull expansion. This, quite obviously means that, the more your skull expands, the more hair you're likely to lose.

For the 20% of men whose skull shape has a **high** growth potential, this explains why AGA will rapidly start developing from puberty and can lead to extensive hair loss by the age of thirty. However, in most cases, AGA won't start until later on in life and will be a much more gradual process - these skull shapes have a **low** growth potential.

Growth potential can, therefore, account for the variations that exist in the rate of hair loss. This answers [question 5](#) in chapter 1.

## 2.6.2 Location

Within the MPB region, the location in which AGA develops can vary.

For some, hair loss develops at the front (temple recession) or back of the scalp (a bald patch). For others, AGA will affect both these areas simultaneously and will cause either diffuse thinning throughout the MPB region, or more concentrated hair loss (a bald patch at the back together with receding temples at the front).

Skull expansion of the frontal bone will form a receding hairline from the temples. Skull expansion of the parietal bones will cause hair loss at the back of the scalp. And skull expansion of the frontal and parietal bones simultaneously will create hair loss in the front and back of the scalp at the same time.

This answers [question 6](#) from chapter 1.

Convinced?

You should be!

Skull expansion is the true underlying mechanism behind AGA. It has explained how, for those with the genetically determined characteristics of skull shape and size, DHT will cause the frontal and parietal eminences to grow during adulthood. It has also explained how DHT can be associated with both hair loss and hair growth, and provided answers to all six questions from chapter 1 (something the current theory cannot do).

## 2.7 Re-evaluation of the current theory

Although the current theory for AGA could not answer those questions, it did nevertheless, raise some issues that now need to be addressed.

Referring back to chapter 1, these involve the following –

1. Androgen receptor sites, 5-alpha reductase and DHT all **appear to be more abundant in the MPB region** of the scalp than in non-MPB regions for those with the genetic predisposition towards AGA.
2. A gene (or possibly several) may make **follicles genetically programmed towards hair loss** (but only in the MPB region).



Ok, we've already looked at this before, but let's examine these two fundamental parts to this theory in more detail –

**1.** First of all, let me make this perfectly clear: 5-alpha reductase, androgen receptor sites and DHT all exist in hair follicles for hair growth, not hair loss – your body does not want to lose something that insulates and protects your scalp from the sun, rain, cold, bugs, etc.

The current theory does not explain how and why any such proliferation of androgen receptor sites, 5-alpha reductase and DHT should occur within just the MPB region and nowhere else. In my view, any proliferation of androgen receptor sites is due to hyperandrogenicity <sup>10</sup>.

What this basically means is that the body tries (unsuccessfully) to grow more hair (i.e., to offset skull expansion) by producing more DHT. If there's an increased expression of the androgen receptor gene within the area of weak hair growth, receptor site proliferation will occur. (Again, remember, that's what androgens like DHT do - they stimulate bone, muscle and hair growth, not hair loss).

Furthermore, 5-alpha reductase, androgen receptor sites and DHT also occur in bone tissue <sup>7</sup>.

Coincidence?

**No way!**

To me, it seems crystal clear that DHT accumulation within the frontal and parietal bones cause these bones of the skull to continue growing – i.e., skull expansion.

**2.** The current theory has identified a number of genes that may be involved in AGA. These include: the androgen receptor or AR gene (STU1), 5a-reductase genes (SRD5A1 and SRD5A2), CYP17, etc. But, once again, no reason has been given why follicles in just one (MPB) region of the scalp should suffer hair loss, but not in any other. This theory simply states that each follicle must be genetically programmed for hair loss and that they appear to have a greater number of androgen receptor sites <sup>11</sup>.

As you now know, it's the genes responsible for skull growth (i.e., shape and size) that cause AGA. And, it's this genetic connection that explains how AGA occurs within just the MPB region.

(I believe this current theory should now be reassessed and classed as an adjunct to the skull expansion process).

Now you know that skull expansion is the true cause of AGA, the next question you need to ask is:

What skull shapes will lead to skull expansion?

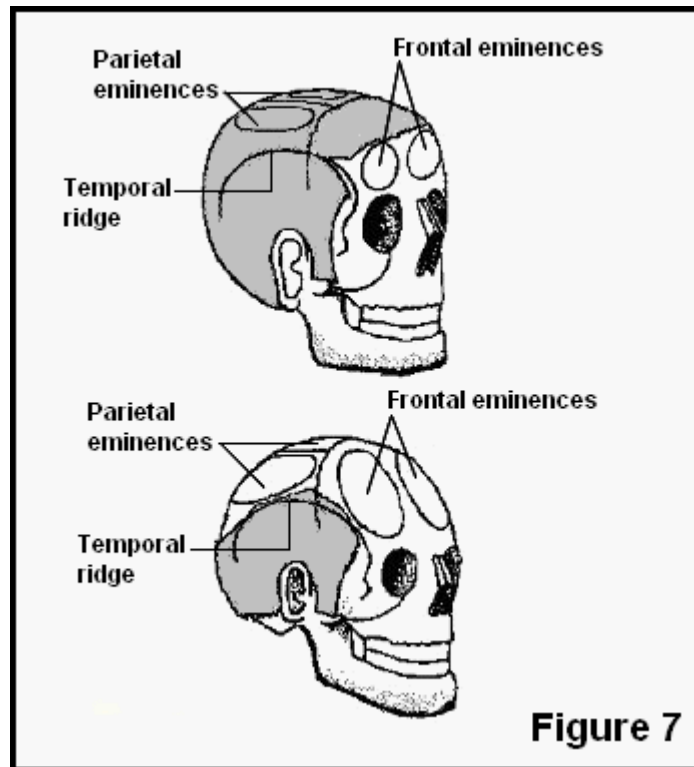
Figure 7 (below) shows two different skull shapes: one with no hair loss at all (i.e., a skull shape that did **not** experience **any** skull expansion), and one with severe hair loss (i.e., the eventual skull shape reached after, perhaps, many years of skull expansion).

You can see that, for those who suffer no hair loss at all, they'll generally have a slightly more square shape to the skull. But, for those who suffer severe AGA, the skull expansion process



will often create a somewhat rounded skull shape. (This rounded shape can also appear in both front and side profiles).

This is only a very simple explanation, but it does nevertheless reveal the skull shapes that generally correlate to the two extreme examples shown in Figure 7.



Other skull shape variations exist, and these will determine **where** within the MPB region skull expansion and AGA develops (front, back or both), and the **rate** at which this will happen. There are also several skull shape characteristics that can be recognised and used to accurately predict whether someone is likely to experience future hair loss, and to what **extent**.

In chapter 3 you can find out how I overcame my own horrible experience of AGA.

### 3. My Story

This chapter reveals my own hair loss experience, how I managed to completely reverse this condition, and **how you can do the same!**

When I first realised I was suffering hair loss, it frustrated and annoyed me. But, before long, it really started worrying me! So much so in fact that, before I realised skull expansion caused AGA, my typical day would look like this:

Get up, check the pillow for hair (and maybe even count them!) Look in the mirror – “How’s my hair looking?”

So, in other words, as soon as I woke up, the **first thing** I thought about was my hair!

I would shower and wash my hair with some kind of fancy “this-will-definitely-grow-your-hair-back” type of shampoo. I tried all sorts of different brands and formulations and also used the sink so that I could check the plug hole for hairs. If you wash your hair as you shower, it may be quicker and more convenient, but your body hair (and I have a **lot** of body hair) will mix in with your scalp hair. This means that you won’t really know how much hair you’ve just washed away.\*

\* Note: you can’t actually wash your hair away. The truth is, only hair that’s weak and ready to fall out anyway will do so.

Anyway, I had one of those plug hole protectors that collect all the hair and stop it from clogging up (a pretty neat gadget by the way). As my hair loss developed, I began losing dozens of hairs every day when I washed it. And when it was at its worst, I lost as many as **300** hairs in a single wash!

**This was something that put me into a state of shock!**

My day would then continue in much the same, almost neurotic way - that’s how much it tormented me!

Frankly, I’m embarrassed to drag you through the entire day I used to have. But you get the point, right? I had become obsessed by this insidious “disease”. Pretty sad, I know, but that’s just how much it affected me (and I’m quite sure hair loss affects many others in much the same way).

Fortunately, hair loss caused by skull expansion/AGA **can now be stopped** - and I’m living proof!

Frustrated by the lack of success I had with conventional hair loss products, I spent seven years studying the skull expansion process in the hope that I might find a possible solution. During this time my own hair loss became more and more severe. But, eventually, I managed to find a way to counteract the hair loss mechanism, and developed a series of simple techniques that anyone can do to actually **REVERSE** the effects of skull expansion.

After just a few months of using these techniques, I had not only **STOPPED** my hair loss, but was also starting to **REGROW** the hair I had lost.

That's when my day changed to look like this:

I would shower (and wash my hair at the same time), towel dry my hair, flick a comb through it, and that's it! **All done!** I then simply got on with my life, secure in the knowledge that I was doing something extremely positive about my hair loss at last.

I could even concentrate better! Whilst the thought of losing all my hair used to pester me throughout each and every day, suddenly I could start living my life like I used to before hair loss had first "infected" me. I used to spend hours dwelling on my hair loss, but once I started using these techniques, I didn't need to give it another thought!

Today I enjoy **a thick, healthy head of hair once again**, and I'm also free from the distress that the skull expansion process had, quite obviously, caused me.

The great thing about these techniques is that you can perform them in the privacy of your own home (you might even find them relaxing after a busy day at work). And, I achieved the results you can see from the photo without needing to buy **ANY** expensive hair loss drugs, bad-smelling creams or ointments, or have any kind of medical intervention whatsoever.

Best of all, these exercises will work for **ANYONE** (men and women), and the results are **PERMANENT** - once you counteract the skull expansion process, there's no reason why you shouldn't continue growing strong scalp hair for life!



To find out more about how these simple yet amazing exercises can stop your hair loss within 3 months, just [click here now!](#)

Note: whilst these techniques were developed specifically for AGA, they can also be effective against other types of hair loss too.

In chapter 4 we review everything you should now know about AGA.

## 4. Summary

Having read this ebook, you should now know the following:

1. AGA is caused by skull expansion of the left and right parietal bones, and the frontal bone.
2. For each parietal bone, expansion originates from the parietal eminence, and causes hair loss at the back and crown of the head. The parietal eminence is located only above the temporal ridge. This explains why hair growth below the temporal ridge (on the sides of the head) is largely unaffected by AGA.
3. The occipital bone lies at the lower rear part of the skull, and is less likely to be affected by parietal bone expansion. This explains why hair continues to grow low down at the back of the head.
4. Frontal bone expansion causes hair loss at the front (receding temples) and crown of the head. Frontal bone expansion originates from the frontal eminences. There are two frontal eminences located on the left and right sides of the frontal bone. This explains why both temples will recede but the centre front hairline can often continue to grow hair.
5. The skull shape, and associated growth potential, that you inherit determines whether or not your skull will expand, so causing AGA. This explains the genetic link to AGA and why some people suffer AGA whilst others do not.
6. Skull shape and size is a largely sexually dimorphic characteristic. This, alongside DHT levels, explains why men experience AGA much more so than women.
7. Your skull shape also determines where (within the MPB region) and extent to which you'll lose hair, as well as what stage in your life skull expansion/AGA is likely to start, and the rate at which it happens. This explains why some people suffer AGA more so than others.
8. DHT causes facial, body and pubic hair to grow at puberty, and skull expansion/AGA (in those with the genetic predisposition towards it). DHT is a steroid hormone which causes bone growth. This explains why DHT has been associated with both hair loss and hair growth, and, alongside sexual dimorphism, why men experience AGA much more so than women.
9. DHT production increases with weight training. This explains why bodybuilding has been linked to AGA.
10. To learn about the techniques developed from the skull expansion process that will restore normal, healthy scalp hair growth, simply [click here](#).

I've raised and answered many questions in this ebook - you now know more about the **true** cause of AGA than most people on the planet! And so, if you're one of the millions of men and women suffering hair loss, the only question that remains for you is this:

**What are you going to do about it?**

Thanks for reading.

Best wishes,

A handwritten signature in dark blue ink that reads "Paul Taylor". The signature is written in a cursive, flowing style.

**Feel free to send this ebook to anyone you want - simply attach it to your emails.**

(You can help other people by letting them know how skull expansion causes hair loss, and how the hair loss industry has got it wrong!)

## References

Various sources of reference material were used in writing this ebook. Only those that are most relevant to the current theory for AGA are listed below.

All references are to online sources of information only. This means that, to examine any reference further, all you need to do is click on the link. This will take you straight to the relevant website page. You should have no problems doing this. However, since these are all external links, it's possible that some links may change or become unavailable through server/technical issues over time.

1. Section 1.2 - [http://www.cytochemistry.net/Endocrine\\_System/adrenal.htm](http://www.cytochemistry.net/Endocrine_System/adrenal.htm)
2. Section 1.3 - <http://www.nature.com/jid/journal/v116/n3/full/5601004a.html>
3. Section 1.3 - <http://www.inhousepharmacy-europe.com/hair-loss/androgenetic-alopecia.html>
4. Section 1.3 - <http://www.hairlosslearningcenter.org/content/causes/cause-of-hair-loss.asp>
5. Section 1.3 - <http://ghr.nlm.nih.gov/condition=androgeneticalopecia>
6. Section 1.3 - [http://en.wikipedia.org/wiki/Baldness#Background.2C\\_cause\\_and\\_incidence](http://en.wikipedia.org/wiki/Baldness#Background.2C_cause_and_incidence)
7. Sections 2.4 and 2.7 - <http://jcem.endojournals.org/cgi/reprint/82/10/3493.pdf>
8. Section 2.4 - <http://joe.endocrinology-journals.org/cgi/reprint/185/2/223.pdf>
9. Section 2.5.1 - <http://endo.endojournals.org/cgi/reprint/140/12/5579.pdf>
10. Section 2.7 - <http://www.hairlosstalk.com/faq/#26>
11. Section 2.7 - [http://www.hairlosshelp.com/hair\\_loss\\_research/hairloss\\_causes.cfm](http://www.hairlosshelp.com/hair_loss_research/hairloss_causes.cfm)