Journal of Tissue Science & Engineering

Foote, J Tissue Sci Eng 2018, 9:1
DOI: 10.4172/2157-7552.1000217

Perspective Open Access

A Question for Scientists in Tissue Physiology

Stephen Foote*

Independent Researcher, UK

***Corresponding author:** S Foote, Independent Researcher, UK, Tel: 07972537513; E-mail: stephen.foote@live.co.uk

Received date: March 06, 2018; **Accepted date:** April 11, 2018; **Published date:** April 18, 2018

Perspective

This short informal article is intended to raise the issue of evidence for a significant oversight, in the published studies about a particular tissue growth restriction. These studies currently fail to consider a basic *in vivo* growth control that according to the accepted science must play a central role. So here I would like to ask scientists if there is any evidence that this particular tissue is a special case and so not subject to this widely accepted fundamental *in vivo* growth control.

According to the accepted science, all normal (non-cancerous) tissue growth in vivo, is ultimately restricted by the external resistance based growth control [1].

According to this, external pressure based growth controls have an overruling action upon all normal (non-cancerous) tissue growth *in vivo*. It does not matter what is driving new tissue growth *in vivo*, a certain degree of external resistance will switch this off.

As demonstrated in the study linked above, increased external pressure restricts new growth, and reduced external pressure allows increased new growth.

Hair follicles regularly go through a cycle of regression then re-enlargement "within" the dermal tissue. Research in this field seeks to understand why the size of the hair follicles created by this process can vary greatly and in particular why the new follicle growth is often severely restricted. Yet nowhere in the published studies is there any consideration of the normal pressure based spatial growth controls that according to the accepted science must apply in this situation (Figure 1).

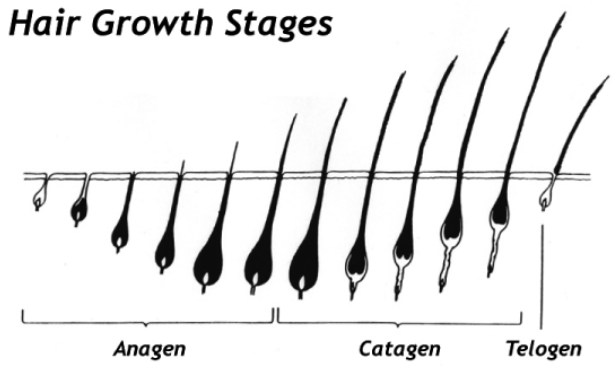

Figure 1: Hair growth stages.

Hair production is closely linked to the size enlarging anagen follicles ultimately achieve, and the often restricted growth of follicles is known to be responsible for the common cases of hair loss (Figures 2 and 3).

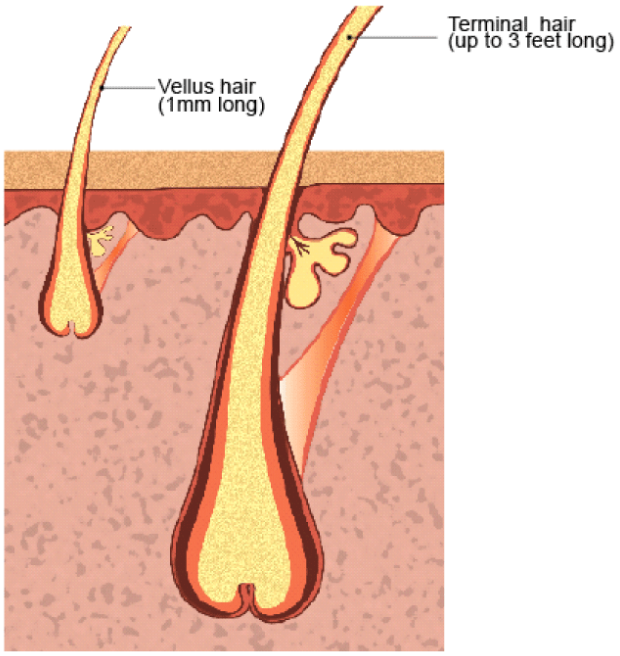

Figure 2: Scalp hair types.

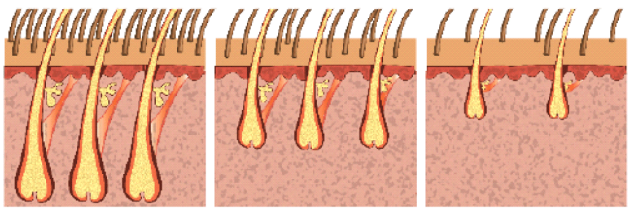

Figure 3: Time-lapse: Miniaturization of hair follicles in baldness.

Once the influence of pressure based external growth controls are considered in changes in follicle size and hair growth, a clear common factor is identified in the recognised data in the field. I discuss this and its wider implications in my main article with further references [2].

To sum this up, the indication is that hair follicles have evolved in mammals, to have their maximum size potential adjusted by small pressure changes in the dermal tissue. This has important purpose in evolution as I discuss in my article.

In the modern Human condition, all the factors known to increase hair follicle size and hair production, all reduce external tissue pressure by one action or another. This includes Minoxidil, anti-inflammatories, low level lasers and lately Latanoprost. This drug was actually developed to reduce tissue pressure in cases of Glaucoma [3].

All the known changes in the surrounding dermal tissue in cases of hair follicle miniaturisation are fully consistent with increased tissue fluid pressure, as in lymphedema. These include significant inflammatory changes, hypoxia, increased fibrotic tissue formation, and significantly increased sweating capacity [4-7].

This external influence also explains why certain transplantation procedures succeed, whilst others fail. In my opinion assumptions have been made about the results of hair transplantation, as I argue with references in my main article. Most of the current cell based research is based upon one of these assumptions. It is thought that because large so called androgen resistant follicles survive in bald areas when transplanted, cells from these will also grow large new follicles in bald areas.

The big difference here is that in transplantation the follicles are "already" large; they are not required to grow in the bald scalp conditions. Any cell based treatment requires the follicles produced to enlarge in the higher pressure conditions of the bald scalp, and these follicles will not be immune to the normal pressure based spatial controls.

Spatial growth controls are overruling. It does not matter if hair follicles are created or modified to initiate growth, or increase their internal growth potential, if external pressure conditions dictate otherwise, the expected growth will not happen.

This factor impacts negatively upon all the current research intended to create new hair follicles, initiate existing follicle enlargement, or boost the amount of follicle enlargement in Humans. This indicated scalp condition in humans, also explains why the results of *in vitro* and mouse studies in this field, and are just not relevant to the Human condition.

In my opinion this currently overlooked dermal interaction, also offers an important insight into mammalian and modern Human evolution and disease. This includes the indication for a significant role of the male hormone Dihydrotestosterone (DHT), in lymphatic function and Female susceptibility to autoimmune diseases. This indication could be easily tested by those in the position to do so, as I describe in my main article.

Scientists in tissue engineering are aware of the influence of spatial considerations in the development and guidance of new tissue growth *in vivo*. Hence the use of scaffolds to influence this process [8].

The indication here that hair follicles have evolved to use variable spatial conditions in their growth cycle, suggests they may be convenient as an *in vivo* model for further study in tissue engineering.

So given the important implications of this growth control in this context, I would repeat my basic question to scientists. Is there any evidence that hair follicle enlargement is not significantly influenced by the normal pressure based spatial growth controls?

References

1. http://phys.org/news/2014-04-room-tissue-growth-cell-response.html
2. https://www.academia.edu/17570665/A_Review_of_the_issues_in_Historic_and_Current_Hair_Research_and_an_Overlooked_Connection.
3. Blume-Peytavi U, Lönnfors S, Hillmann K, Garcia-Bartels N (2012) A randomized double-blind placebo-controlled pilot study to assess the efficacy of a 24 week topical treatment by latanoprost 0.1% on hair growth and pigmentation in healthy volunteers with androgenetic alopecia. J Am Acad Dermatol 66: 794-800.
4. Swezey L (2011) Lymphedema – Overview and etiology.
5. Zampell JC, Yan A, Elhadad S, Avraham T, Weitman E, et al. (2012) CD4(+) cells regulate fibrosis and lymphangiogenesis in response to lymphatic fluid stasis. PLoS One 7: e49940.
6. Goldman BE, Fisher DM, Ringler SL (1996) Transcutaneous PO2 of the scalp in male pattern baldness: A new piece to the puzzle. Plast Reconstr Surg 97: 1109-1116.
7. Cabanac M, Brinnel H (1988) Beards, baldness and sweat secretion. Eur J Appl Physiol Occup Physiol 58: 39-46.
8. Chan BP, Leong KW (2008) Scaffolding in tissue engineering: General approaches and tissue-specific considerations. Eur Spine J 17: 467-479.