Subject: Ultimative Lösung für Anti Aging und Haarwachstum?
Posted by Haar_Challange_2021 on Sun, 09 Nov 2014 15:31:32 GMT
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Es handel sich dabei um das Blockieren von GSK3-Beta.

Lest mal diesen Artikel von Vincent in seinem Anti Aging Blog

Es ist Unglaublich die Zusammenhänge und die möglichkeit eines selektiven GSK3-Beta Hemmers.
Lithium ist von den Metalen am Potentesten und habe schon mal gelesen das mit DMSO einer im Ami Forum unglaubliche Ergebnisse erzielt haben soll.
Wie ihr im Blog lesen könnt ist auch Kupfer, VPA etc GSK3-Beta hemmer aber nicht so Potent.

Wenn man nun aber Lithium und Hairregrowth sucht Stösst man haufenweise auf Artikel wovon Haarausfall berichtet wird.
Mann muss verstehen das Lithium bei Psychischen Erkrankungen genutzt wird mit mehreren 100 Milligram. Leider hemmt Lithium auch eine andere Kinase nämlich die für die Bildung von Inostiol was die Ursache für Haarausfall ist (In laboratory animals, a diet lacking Inositol produced baldness)

Dies könnte die Ultimatetive Lösung sein:
Eine Orale Dosis von 5 bis 15mg Lithium Orothate zusammen mit Inositol und NRF2 Aktivator (Larrea tridentata) welche nordihydroguaiaretic acid enthält.

Alternativ Topisch:
DMSO + Calbiochem
http://www.merckmillipore.com/CH/de/product/GSK-3%CE%B2-Inhibitor-I---CAS-327036-89-5---Calbiochem,EMD_BIO-361540#anchor_orderingcomp
Leider weis ich nicht ob das Molekül durch die Kopfhaut geht aber die Chance mit DMSO als Carrier stehen die Chancen sehr gut.

Wichtig ist auf jedenfall die Auflösung der Fibrose (Kollagenverhärtung) Um dies zu erreichen sollte 1x pro Monat der Dermaroller oder noch besser ein Dermastamp verwendet werden. (Wegen dem Einstich Winkel)
Zitat:Wounding is COUNTERPRODUCTIVE when done more than once every 30-40 days. When a wound occurs on the scalp, Lhx2 is activated. Lhx2 inhibits Lgr5 which regulates bulge and secondary germ stem cells. Inhibiting Lgr5 will keep hair in anagen by inhibiting stem cell activation. So, creating a new hair in anagen in the follicle will not be possible. Lhx2 stimulates sox9 and tcf4, which is needed for hair follicle progenitor cells. So in short, wounding regenerates stem cells but also inhibits their activation for new growth in a certain time period.

Zitat:1)))Androgen receptors are the main key. The factors that act on them, such as DHT and other androgens kick starts the alopecia pathway.

GSK3-beta is involved in androgen receptor nuclear binding. It is also involved with stem cell differentiation (causes it), inhibits stem cell proliferation and the canonical wnt/beta catenin

pathway. It has been shown in prostate cells that LITHIUM CHLORIDE a gsk3-b inhibitor, inhibits the nuclear binding of the androgen receptor even if it is bound to DHT. So, this means no transcription or translation of the androgen receptor= cessation of androgenetic alopecia.

Creating a topical gsk3-b is key. Lithium chloride, lithium succinate (used in seborrheic dermatitis), and lithium orotate are options. However, be careful as not all gsk3-b inhibitors have to same action on the androgen receptor.

2))) Androgen receptors create a cascade of growth inhibitors like TGF-beta to cause cell death and IL-6 expression in the hair follicle.

IL-6, Stem Cell factor (in follicle), and CD34+ progenitor cells(follicle) cause the differentiation of CD34 cells into mast cells. This is my theory as to what is happening to the CD34+ cells and why there is a depletion of them. Mast Cells are being produced and the types being produced are large, filled with ILs, chymase (involed with atherosclerosis/fibrosis), and histamine. This is important to understand the itching, tingling, burning, and inflammation on the scalp. The yeast on the scalp is reacting to the change of fatty acids produced by sebaceous gland hyperplasia (I will explain this later). This causes the allergen reaction, IgE activation of these mast cells, and the eventual cascade of microinflammation involving lymphocytes(th2), basophiles, and eosinophils. This will a create a vicious PGD2 cycle, miniaturization, and fibrosis.

3))) Mast cells and lymphocyte created PGD2. This is due to the DHT/androgen receptor IL-6 production. These cells also produce various ILs. IL-6 is one of them that cause the microinflammation around the follicle which leads to eventual miniaturization and possible death. Hematopoietic PGD2 is produced by these cells. H-PGD2 causes a gene called NRF2 to activate. NRF2 causes sebaceous gland hyperplasia and hair follicle regression via hyperkeratosis (why I believe the hair follicle miniaturizes and can, eventually, die). TGF-beta via androgen receptor/DHT causes NRF2 activation.

NRF2 is extremely important. It is an anti-inflammatory activator and its product is LIPOCALIN PGD2 SYNTHASE (L-PGD2s). Whenever nrf2 is exposed to any type of pgd2, it will keep producing L-PGD2S, hence the vicious PGD2 cycle. What happens when Prostaglandin H2 from COX2 is converted to L-PGD2? It eventually produces 15D-PGJ2.

15D-PGJ2 is a PPAR GAMMA receptor agonist. What does PPAR GAMMA do? It is for ANTI TUMOR protection. Interestingly it can cause anti inflammation or inflammation. PPAR GAMMA still needs to be studied more, however from research I found out that 15D-PGJ2 inhibits the proliferation of CD200.

CD200 is one the the two type of progenitor cells that are lacking in balding hairs. It is involved with self protection, causing production of IL10 and IL12 to down regulate inflammation against the host (hair). follicle) and involved with melanocytes (low cd200 positively correlated with graying hair). My theory is that PPAR gamma has anti tumor effects and that the hair follicle is being viewed as a tumor, hence the depletion of CD200 in order to removed the tumor(hair). PPAR gamma is needed in order to prevent scarring alopecia as it maintains functional stem cell compartment. Like I said, PPAR gamma in studies have been shown to causes inflammation or the opposite probably due to which ligand /receptor is activated and needs to be studied more.

Stem cell factor is decreased in androgenetic alopecia, so a decrease in all progenitor cells and loss of CD34 (mast cells) and CD200 (15D PGJ/ppar gamma)

Inflammation is not all bad. Certain types of inflammatory processes are needed for hair growth, but they need to be done in a certain time and manner which is not being allowed in androgenentic alopecia. This topical should be discussed further.

Here is some information of products used for hair loss:

Lithium: This should be the number one used topical for all hair loss suffers. It inhibits GSK3 beta, so it should totally inhibit the androgen receptor; EVEN IF DHT IS ATTACHED! It increases B-catenin and the wnt canonical pathway by inhibiting gsk3 beta, so it can increase anagen in hairs. It also inhibits TGF-beta which causes hair loss.

Minoxidil: It is a PGH1 synthase activator. This means more prostaglandins production. It will cause an onset of inflammation due PGE2 causing enhanced function of IgE on mast cells and increase IL-6 in those cells. Also, don't forget PGD2 and other products from cox2 that cause inflammation. Hence, the initial shed using minoxidil and it can cause thinning of existing hair (inflammation,pgd2 increase). However, due to its ability to create capillaries in dermal papilla and E2 increase, it causes regrowth of hairs and minoxidil dependent growth of hair by being a canonical wnt pathway weak agonist.

Azelaic Acid: It decreases p53 and p21 causing a decrease in apoptosis. It decreases nf kappab which is overexpressed in hair loss but is needed for anagen. It also is an anti inflammatory by apparently activating ppar gamma which explains why it is used for skin lightening (decrease melanocyes due to a decrease of CD200). PPAR gamma plus p53 causes apoptosis, hence Azelaic acid is anti-apoptotic. Also, combined with high levels of vitamin b6, it decreases DHT production.

Retinoic Acid (tretinoin, all trans retinoic acid): It can possibly cause skin cancer and this is good for hair generation. Remember that hair is being targeted as a tumor/cancer even though it is not, so we need something that can reverse/balance that anti tumor effect and RA is that something. RA inhibits canonical wnt pathway(bad for hair anagen) via tcf2 but upregulated noncanonical wnt pathway. This is good for stem cell generation. RA also inhibits NRF2 (Decrease L-PGD2s and L-PGD2). RA also activates the ERK2 pathway which is different than the ERK1 pathway. ERK2 is involved with cell angiogenesis and ERK1 is involved with apoptosis.
Fenugreek contains Trigonelline and it inhibits NRF2 to downregulated lipocalin pgd2s and l pgd2 and does not effect wnt pathway.

Luetolin inhibits NRF2 but also inhibits the canonical wnt pathway.

Hypoxia causes NRF2 inhibition: Neogenic and ciclopirox

Some information about signaling:

The JAK STAT pathway is key in regulating hair loss. There is a report that a JAK STAT inhibitor was used on a patient with alopecia totalis and all his hair grew back. We know that Androgenetic Alopecia and Alopecia Areata/Totalis are different, but are similar in their pathways.

IL-6 is the problem with Androgentic Alopecia that causes inflammation, hair thinning, fibrosis, and a pathway that leads to NRF2/PGD2. IL-6 is involved with the jak stat pathway, particularly with stat3. If we can inhibit STAT3, we can solve the IL-6 problem. This should shut down the IL-6 mast cell instigation of microinflammation around the hair follicle. There are natural and synthetic JAK STAT inhibitors available. I found one topical that should contain a stat3 inhibitor, but I will not dicuss the product so that we can focus on other important matter written here and I don't want to have to defend myself from being called a shill.

http://www.apotheken.de/news/article/hohes-alter-dank-lithium/