Lilac Ram
Dr. Bo Kühls
16-10-1978

Loggbok:

Cancerhämmande effekter från progenitor-viruset i patient nr 1

Bilaga B:

The plantoid mutation effect in the progenitor virus

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Origins and effects of the Progenitor Virus

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1		2		9				3		6		8
	4		7		6		3		2		1
9		2		3				6		6		6

Loggbok

16 Oktober

Första experimentet ska utföras på patient nr 1 den 17 oktober. Patient nr 1 lider av en obotlig cancer i magen som sprider sig snabbare än andra cancerformer. Jag har fått tillåtelse från företagsledningen att använda Progenitor-viruset i experimenten. Detta virus ska hanteras med största försiktighet eftersom det förvaras i syra (Syran hindrar viruset från att förstöras vid eventuella transporter). För att experimenten ska nå sin fulla framgång behöver jag injicera progenitor-viruset på olika ställen av patient nr 1 kropp. Se bilaga B för tidigare forskning om progenitor-viruset.

17 Oktober

Första försöket
Patient nr 1 fick ligga ner på britsen. Injektionen av progenitor-viruset skedde via magen. Patient nr 1 klagade på kraftiga buksmärtor.
Ingen direkt reaktion på virusinjektionen inträffade. Jag får avvakta ett par dagar innan jag beslutar om ett andra försök behöver göras.

19 Oktober

Patient nr 1 uppvisade redan efter några timmar biverkningar från behandlingen. Nedsatt syn, okontrollerade kramper och feber framkom under dagen, men de verkar ha passerat nu. Troligen inget att anmärka, men framtida tester kräver vidare uppsyn timmarna efter injektion.

25 Oktober

Andra försöket
Första injektionen gav inga direkta resultat. Dock kan man se en förändring hos patient nr 1. Patienten visade upp drag av aggressivitet och visade tydliga förändringar i humöret.
På grund av detta får nu patient nr 1 från och med nu vara fastspänd, för att undvika svårigheter i behandlingen.
Progenitor-viruset injicerades denna gång via dropp. En långsam process för att se om den långsamma överföringen har en annan effekt än den direkta.

26 Oktober

Injicering via dropp visade inga spår av biverkningar. Troligtvis handlade det bara om kroppens ovana vid ämnet.

5 November

Sedan andra försöket kan man se en tydlig förändring hos patient nr 1. Patienten klagar inte på smärtor längre. Patient nr 1 ser ut att må bättre, detta märks genom att patientens ansiktsfärg börjar se normal ut, även genom klarheten i patientens ögon. Patienten väljer även att sysselsätta sig genom lek och interagera med andra människor. Injicering via dropp tycks tillåta viruset snabbare åtkomst till cancercellerna. Planerar in ytterligare ett pass med dropp innan röntgen får visa om cancercellerna påverkats.

11 November

Resultaten från försök nr 2 visar att patientens blodvärde har sjunkit. Efter en period av bitvis förbättring tyck cancern fortsatt att sprida sig och inom två veckor kommer cancern ha spridit sig till lungorna. Den snabba försämringen kan inte förklaras på annat sätt än att dropp-metoden skapar snabb immunitet mot behandlingen. Kommer höja doserna framöver på grund av den explosiva expansion cancern uppvisar. Patienten visar även på hastiga humörsvängningar och vissa mindre biverkningar, så som försämrad motorik och slöhet i sinnet. Får övertyga fadern om att hålla patienten inomhus tills vidare. Försök nr 3 måste ske snarast.

13 November

Patient nr 1 kände en extrem smärta igår. Försök nr 3 fick inledas två dagar tidigare än planerat. Patienten var aggressiv och visade detta genom fysiska handlingar.
Patient nr 1 lyckades ta sig loss från spännena, fick tag på syran och kastade den i mitt ansikte. Injektionen skedde i halspulsådern, med dubbel dos den här gången. Patient nr 1 svimmade av behandlingen och blev återförd till sitt rum. Tror det handlade om fysisk utmattning, snarare än den höga dosen men patienten får vara under observation i ett par dagar.

15 December

Patient nr 1 visar på starka förändringar i sitt beteendemönster. Patienten blir mer aggressiv och stänger in sig på sitt rum. Inga fysiska symptom syns dock längre. Patient nr 1 tycks, fysiskt sett, vara på bättringsvägen. De psykologiska aspekterna får vänta så länge, huvudsaken är att hon blir frisk. Blodprovs togs när patienten sov. Avvaktar för resultat.

12 Januari

Blodproverna visade att blodvärdena sjunkit. Cancern sprids snabbare än innan, trots uppenbara tecken på bättring. Patienten är piggare och aktivare än vid starten på behandlingen, om än väldigt aggressiv, men cancern tycks värre än innan. Progenitor-viruset tycks inte bekämpa cancercellerna, utan muterar dem istället. Osäker på om det innebär ett botemedel eller ej. Om inte rätt metod hittas snart, har patienten knappt tre månader kvar att leva av en vanlig cancerpatients mått mätt. Tror dock att mutationen av cellerna kan rendera sjukdomen icke dödlig. En undersökning av patientens hjärna kommer göras så fort som möjligt och om möjligt kommer nästa försök ske med direktinjektion till hjärnbalken. Om viruset kan komma att påverka cellerna i hjärnan kan mutationen påskyndas snabbare på det sättet.

20 Januari

Företagsledningen har pratat om positiva effekter om injicering av progenitor-viruset genom en teknik som nyttjar blodiglar. Har eftersökt möjligheten att införskaffa dem för framtida tester.

2 Februari

Patient nr 1 visade stora aggressiva tendenser, då patienten försökte anfalla med en kniv. Vidhåller ställningen att patienten borde hållas fastspänd på heltid, men fadern vägrar. Patienten blev sövd och prov togs på hjärnbalken. Beslutet togs att Progenitor-viruset skulle injiceras i hjärnan.

15 Februari

Injiceringen via hjärnbalken lyckades. Timmarna efter proceduren verkade ge positiva resultat. Patient nr 1 tycks ha minskade aggressioner och inga fysiska biverkningar framkom. Patient nr 1

sitter på sitt rum och skrattar, antar att det betyder att de psykologiska biverkningarna av viruset är överspelade.

2 Mars

Mina teorier om att cellmutationen skulle ske i mycket snabbare takt med injicering via hjärnbalken tycks stämma. Vid röntgen idag syntes inga cancerceller till alls. Patient nr 1 log mot mig under röntgen. Verkar som att experimentet blev en framgång till slut.

The plantoid mutation effect in the progenitor virus

Etnon Morieris

Lilac Ram

Introduction

I made this topic as a central hub for discussion of the biological processes involved in the Progenitor and Progenitor-derived Viruses. Having only read The Biology of Evil, Part 1, I can only comment on the information presented thus far, but hopefully I can shed some light as to the processes involved with the progenitor virus. If we're to understand how the progenitor virus work, it involves looking at the specific mechanisms through which it enacts mutations to the organism which it infects. In other words, we should observe the symptoms if we want to identify the processes involved. I believe that the virus is somehow related to hormones, specifically the release of hormones triggered by the virus. Interestingly enough, I was already pretty convinced that the virus worked by overstimulation of the hypothalamus. However, as enzymes playing any substantial role in the viruses actions, I'm less convinced, if only for the reason that the hormone-influencing theory works itself in so much more comfortably with what we see happening to the organisms infected with the virus.

Auxins and Lectins

Auxins are plant Growth Hormones that directly affect how tall a plant gets. In humans, Somatropin is released by a small part of the brain called the pituitary gland. What's weird is that this release isn't how you would imagine a release of a hormone to be. It is released in constant small bursts throughout the day, with more being released later in the day and the most being released in the first 30 minutes of when you fall asleep.
The Doctor have questioned the capability for a virus to affect both human and plant hosts. These Auxins may somehow interact with the virus, deactivating key receptors of the virus, the plant, or perhaps binding to the virus's RNA receptor sites thereby rendering it unable to perform any action on the plant itself. This would ensure the plant is not harmed by the virus or simply that the virus does not affect the plant for better or for worse.
Lectins are nasty little substances made of protein found in many common foods, but mostly in the seeds of plants. The most common sources are wheat and other gluten-containing grains, legumes, nightshade plants such as tomatoes, dairy, and all seeds and nuts. They are an evolutionary measure that protects the seed of a plant from predators. This self-defense mechanism ensures that even if the seed does get eaten, the organism that eats it suffers some consequence and, hopefully, in the future will avoid eating that plant so that it can prosper. Lectins are what scientists refer to as glycoproteins, they are not true proteins in that they are simply made of amino acids stringing together to form peptide bonds. They actually aren't sticky at all, but the stickiness refers to their uncanny ability to bond with cell walls and receptors[1] but especially the intestinal lining. Lectins also bind with minerals and can deplete the body of nutrients that we need to live and function properly, such as Zinc and Calcium. Due to their ability to bind with cells in a way that is not beneficial to the host, they indirectly have the ability to let the cell do things it wouldn't normally do. People who have been exposed to many Lectins over a long period of time literally have micro-perforations of their stomach and intestines. These small holes allow things into the blood stream that, under normal conditions, would be perfectly fine. However, once these holes exist, the Lectins actually inhibit the intestinal wall's ability to rebuild due to the aforementioned "receptor blocking."[2]. Once the intestinal wall is compromised, reactions to external stimuli is changed

1 http://www.ncbi.nlm.nih.gov/pubmed/3000517
2 http://www.ncbi.nlm.nih.gov/pubmed/9987662

drastically. Foods or biological processes such as Ketosis may cause an antibody response through a Histamine or cortisol release to something which the person is not (inherently) allergic, sensitive or intolerant of.
As mentioned earlier, due to the cellular activities of plants, it's possible that the progenitor virus had no effect on it. Do we see this anywhere else in nature? Yes, with Lectins as well. As we have established, Lectins only bind to certain sites. But what if that site is blocked already by something that is supposed to be there? Well, then the Lectin has no effect or the effect is minimized substantially. Wheat Germ Agglaglutin (the Lectin found in wheat) binds to the receptor sites in Glutamate. However, if the person was to take a supplement containing N-acetyl Glutamate, the receptor site would be blocked for the lectin and it wouldn't interact with the cell and can be excreted as waste[3].
Neurons are unable to signal cells to multiply rapidly to rebuild the intestinal lining. This, in addition with the extra Growth Hormone actually inhibits Cortisol, which would normally be a good thing, since too much Cortisol is bad, but Cortisol coincidentally also goes to the site where the body is damaged and signals the release of various processes that are involved in rebuilding and healing the body. This explains why various creatures infected with the progenitor virus look like deviants. If Cortisol is being inhibited, less wound healing and rebuilding is happening. This could explain why many organisms infected with the virus become disfigured, but more accurately why they are unable to heal from such injuries.
Almost all vegetables and herbs that are healthy for you contain things called phytoestrogens. These are healthy compounds that have antioxidant benefits and decrease androgens in the body which can cause cancer and a host of other health problems. Some of them are slightly anti-nutriental, although the effect is so minor - and the benefits outweigh the negatives - that one would be foolish to avoid certain foods for this reason alone. However, plants such as soy and some grains contain Xenoestrogens. These are a group plant estrogens, similar to phytoestrogens minus the benefits. They are surprisingly similar to human estrogen and thus have a more pronounced effect in what they do. Their effects on the organism's body could be further exacerbated by the Auxins and overstimulation of the Pituitary Gland resulting in over-secretion of Growth Hormone. When you combine several of these factors, cell proliferation and the over-division of cells/increased cell turnover (cancer-like activities, but not necessarily cancer) may happen, leading to genetic mutations, extreme physical growth including a significant increase in muscle mass. These xenoestrogens could also be the reason why subjects exposed to the progenitor virus become sterile.
As we have noted earlier, Auxins are capable of making plants grow, Lectins are capable of protecting plants and binding to receptor sites, Phytoestrogens are capable of providing antioxidant + anti-nutrient benefits (for the plant, anyway) and Xenoestrogens are capable of mimicking and provoking response by human hormones. As opposed to having several individual factors happening, which our bodies may normally be equipped to deal with, we now have a compounding effect of the virus.
Since all of these factors affect not only plants, but animals as well, we are able to apply the above information generally and indiscriminately.

Hunger

Not to be confused with Lectins, Leptin increases feelings of satiety after a meal. Leptin OB mice (mice bred to engineer smaller or virtually non-existent amounts of Leptin) grow obese very quickly and find themselves hungry more often and less satiated after every meal. Ghrelin and Leptin have an inversely proportionate relationship. When Ghrelin goes down, Leptin goes up. Sleeping actually decreases Ghrelin significantly, which increases Leptin production. The inverse is also true, if you’re not sleeping a lot you will crave food more. Cortisol is also involved in the regulation of Insulin[4] but more importantly, insulin resistance. A protein called Resistin, which functions more like a hormone, is released by adipose tissue and aids in many of the same inflammatory response

3 http://www.ncbi.nlm.nih.gov/pubmed/12504587, http://www.ionchannels.org/showabstract.php?pmid=12504587
4 http://www.ncbi.nlm.nih.gov/pubmed/7033265

and wound healing processes that Cortisol is involved in[5]. Could this mean that in times of Cortisol inhibition, Resistin takes on the role of Cortisol? Resistin is also responsible for making us less sensitive to the effects of Insulin[6]. When Insulin doesn't work at all, or doesn't work as well, a host of problems including diabetes (not related,) less blood flow and cell death can occur (both related.) Common knowledge nowadays tells us that Insulin and hunger are directly related. If an organism is not producing insulin, or is insulin resistant, unnatural hunger will occur. It's also interesting to note that Somatropin supplementation also increases Resistin[7]. Strangely enough, Ghrelin is in a group of substances and compounds known as Growth Hormone Secretagogues; they stimulate the body to secrete Growth Hormone, but are not GH themselves, even similar to GH or have the same functions of GH. Since we have established that the progenitor virus are capable of stimulating the release of somatropin, we can combine these two facts together to understand why mutations both crave lots of flesh and why they are not obese. Two ideas that on the surface should not go hand in hand, biologically speaking.

Aggression

Increased GH can increase testosterone production in males and we've all heard of roid rage. It can also increase GABA production, which affects muscle tone and is a neurotransmitter that plays a role in movement. This could also tie into some subjects superhuman speed.
If the virus is stimulating GH, it is increasing hunger and muscle mass due to:
-Increased Androgen (which turns into either Testoserone or DHT)
-Decreased Cortisol
-Increased Resistin taking on the role of inflammatory response due to decreased Cortisol.
-Less Insulin and/or Insulin resistance due to increased Resistin.
-Increased Ghrelin due to lack of sleep
-Decreased Leptin due to lack of sleep
-Increased Testosterone
-Increased Androgen
-Increased GABA
-Increased GH (which itself helps build lean muscle mass) due to a multitude of internal factors and interactions.
Although the mutations look ancient and decrepit, they prove time and time again to be quite resilient and strong. Or at least stronger than one would expect from a creature who has had several of its biological processes shut down or stop completely. I believe the increased GH is allowing the mutations in particular to function with better efficiency than they would be able to had their GH levels not risen so much. The balance between increasing hormones or proteins, combined with the opposite-pull effect of hormones and neurotransmitters which increase muscle mass would explain why mutations are right in the middle in terms of body composition. The two opposing forces find a sort of equilibrium.

Additional sources:

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5 http://www.cmi.ustc.edu.cn/3/1/29.pdf, page 30-31, Resistin and Inflammation
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Origins and effects of the Progenitor Virus

Doctor Murdoc

Lilac Ram

Classification

Group: Group VI (ssRNA-RT)

Family: Retroviridae

Genus: Unknown

The Progenitor virus (also known as the Clay virus) was a unique mutagenic retrovirus, a type of RNA virus discovered from a rare flower in West Africa. It possessed the ability to parasitize a host at a cellular level and mutate DNA and recombine genes, thereby strengthening a host's characteristics but causing detrimental mutations when cells developed abnormally. It was unknown to most of human civilization until its discovery by me. For it’s purpose, the virus became the basis for a range of medical and biotechnical virus projects.

History

Origin

It was theorized that it must have previously existed in an isolated system since its zoonotic attribute to infect almost any organism would have otherwise allowed it to become a trans-species pandemic. It is now confirmed to have been discovered in an underground cave system in West Africa, in a region home to an ancient civilization known as the "Ndipaya" who possessed advanced technological knowledge for their time and had constructed an expansive underground city deep within the caves, where their monarchy ruled.

The flower was extremely poisonous to humans and its effects were usually fatal if consumed. However, some individuals possessed a natural genetic resistance to the virus. The Ndipaya people believed that a man who could survive after ingesting the plant was destined to become king. Finding an individual who could survive ingestion of the powerful poison was a rarity, but a successful individual would gain superhuman abilities, intelligence and above all, immortality.

Search (1960s)

The search for the Progenitor virus was started by me and Dr. Etnon Morieris, a virologist. Alongside us was Dr. Frank, a biochemist and authority on viruses. In the beginning, we had been searching for a virus which could promote evolution, based on the scientific theory that viruses were a major factor in the evolutionary process and could directly promote it.

The subsequent search for the Progenitor virus began based on information about a Ndipaya ritual contained within the "Natural History Conspectus", a set of books penned by Henry Texas as an encyclopedia of Africa. I was interested in the conspectus for its ethnological side, but upon reading the description of the Ndipaya tribe ceremony for choosing their kings, which incorporated a flower that could grant a human great abilities if consumed, I became intrigued and conveyed this information to Dr. Morieris. Dr. Frank hypothesized that the flower's ability to enhance certain characteristics was a phenomenon caused by an unknown virus altering DNA.

In September 1966, we traveled to the region where the flower was described. Upon reaching the garden, the expedition group began to examine the flower. On December 4, we confirmed Dr. Morieris hypothesis and discovered that the flower did contain a new virus strain; an RNA retrovirus we named "Progenitor". With the unique mutagenic attribute of the Progenitor virus, I had proposed using the virus for the purpose of finding a cure for cancer.

Culture Attempts (1967)

On February 12, 1967, while attempting to cultivate the Progenitor flower outside of its original

habitat, we hit a dead-end in our research. We initially tried tissue culture of the virus; however the result lacked the DNA transmutation characteristic. Therefore, we decided to mass-produce the virus by cultivating the Progenitor flower. At first, it was favorable, as the flower grew quickly with strong vitality in few terms. However, the virus was not present in the resulting flowers. At another dead-end, we decided to inspect further and examine whether the cultivation environment would affect the virus' development within the flower.
By March 23, we had cultivated the flower in various conditions under many variables for soil, water, temperature, humidity and duration of sunshine. The virus did not develop in the flower even after making every condition the same as the Sun Garden.
On November 10, the first human administration experiments were secretly conducted with the Progenitor virus. At our lab in Procyon, two variant strains of the virus were produced in an attempt to successfully adapt it to the human body and remove the need for a genetic resistance. The first variant known as "TYPE-A" was injected into Patient 0, whose tissue deteriorated when the virus failed to establish itself in her body. "TYPE-B" was administered to Patient 0.2, whose tissue also deteriorated rapidly. However, the virus successfully established itself and some positive results were found in her remodeled body. She was kept under observation and became the main test subject for the research, and we kept her existence, the experiments and the results a strict secret from the outside world. Research on the Progenitor virus continued in Lilac Rams laboratories around the world, primarily for its use as the foundation upon which new virus strains could be developed.

Attributes
The primary attribute of the Progenitor virus was its DNA mutation characteristic which enabled the recombination of genes and potential for organic evolution through subjecting infected cells to mutation. When the virus was administered directly into an organism, it would take up residence in the cells of the host and self-propagate by using the synthesis properties and energy of proteins. The protein and nucleic acid synthesis program of cells infected with the virus were then rewritten based on the nucleus of the virus, resulting in growing numbers of new Progenitor virus nuclei.
When infected cells developed abnormally, the subsequent rapid cellular mutations would not only destroy the host's original tissue, but also cause the extreme deterioration of intelligence through the erosion of brain cells. In other cases, infection would simply result in a host's death. Creatures that survived infection either became brutal and ferocious or were left in a weakened and damaged state. Size increases due to an excessive secretion of growth hormone were also not uncommon in infection cases.
Progenitor Humans
The only mutation caused by the virus that could be referred to as true "evolution" was its adaptation to human hosts with an inherent genetic resistance to its otherwise fatal effects, known as a "Progenitor Human." This resistance was actually a specific genetic makeup which enabled the production of certain anti-bodies, with which mutation by the Progenitor virus did not affect the host's tissue and allowed them to gain superhuman strength, enhanced intelligence and biological immortality. Another common physical trait shared between these evolved humans was the ability for their eyes to glow red, usually when enraged. However, as adaptable individuals were extremely rare, the virus' potential for use in the evolution of humanity was also extremely low.