

## Darwinism with the Recognition of God: Planet Earth Awesome or Awful

Vincent van Ginneken\*

\*Bluegreentechnologies, Heelsum, Netherlands

\*Corresponding author: Vincent van Ginneken PhD-1, PhD-2, MSc, Bluegreentechnologies, Heelsum Netherlands.

Email: vvvaninneken@hotmail.com

Received: June 16, 2020

Published: July 10, 2020

### Editorial

In an earlier review I stated a rather “atheistic” statement about the evolutionary forces involved during the course of human evolution (Figure 1). That evolution has no plan of a “Benevolent Supreme Being”, and that “impersonal forces” drive evolution. That “by accident” plays a large role in the selection of these traits which were under these conditions the most favorable for survival. That e.g. “the development of the human brain has not been developed by a guiding hand, but through a conclusively endless series of ‘breedings’ and ‘eatings’ out of which some traits were developed and became what we see today”. I gave some quotes of Prof. Dr. Steven Weinberg (born May 1933) who gave his wise vision on the Netherlands television in the series “Beauty and Consolation around twelve years ago (Wim Kayzer, VPRO-television) [1] which had a great impact on me when I was a student.

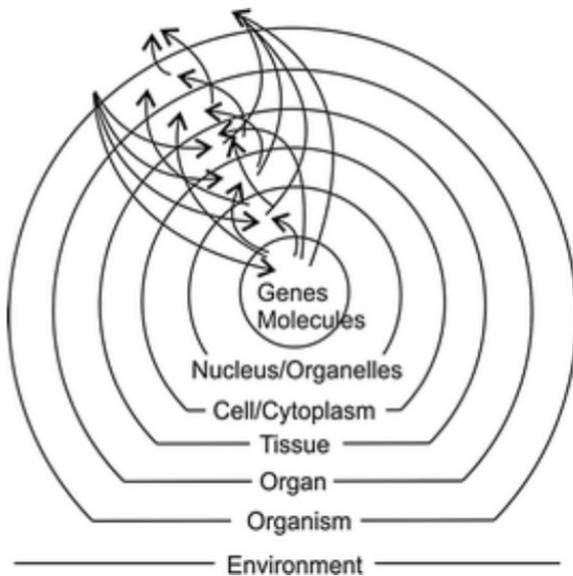
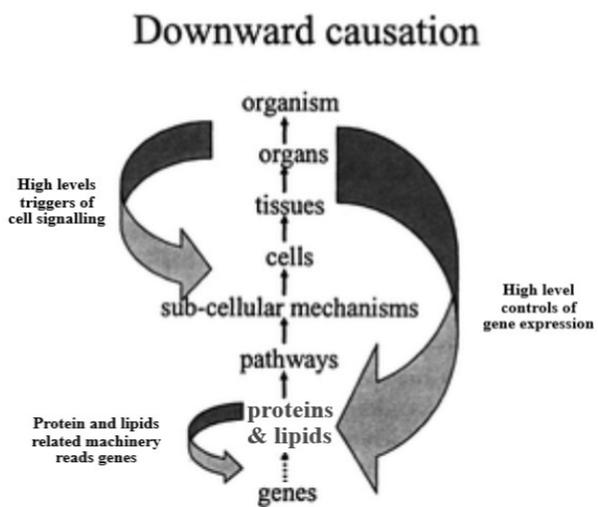


Figure 1: Example of Impersonal forces, a world in which ‘by accident’ plays a large role in the selection of these traits which were under these conditions the most favorable for survival.

I remember and could imagine what he went through as a survivor of the holocaust, lost his loved ones and family during that horribly inhumane genocide, and used the word “silly” and “shut up, I’m bigger than you are”, in the mutual relation between perpetrator and victim. It was for him still unbelievable to accept that a civilization that had produced so much beautiful works of art such as poetry (Goethe), music, composers such as Bach, Brahms, Beethoven, etcetera that such a cruel

and horrible system evolved just within such a country with such a high level of art of the emotions. And that these cruelties were a handling dictated by the “mysteries of mysteries” the omnipotent human brain with its protective mechanism of ‘mirror neurons’. Anytime you watch someone doing something, the neurons that your brain would use to do the same thing become active-as if you yourself were doing it. Like [2] stated: ‘If you see a person being poked with a needle, your pain neurons fire away as though you were poked’. And we all know what horrible conditions people had to live in concentration camps at the time. And that after such happenings with a violent impact on your life, you can no longer believe in a God who cares about the individual and that such events also determine your vision of the evolutionary laws. That evolution is just the outcome of a mathematical algorithm at the African savannah. Or according to the vision and reasoning of Richard



Dawkins in “the Blind Watchmaker:” Natural selection, the blind, unconscious, automatic process which Darwin discovered, and which we now know is the explanation for the existence and apparently purposeful form of all life, has no purpose in mind. It has no mind and no mind’s eye. It does not plan for the future. It has no vision, no foresight, no sight at all. If it can be said to play the role of watchmaker in nature, it is the blind watchmaker” [3]. And I add “the individual doesn’t count and is in the constant Darwinian “Struggle for Survival”. Such as in extremity in the holocaust which totally overrules and ‘dam-

ages' a person's character and personality when he survives. And it is in further life the struggle to continue with 'a balanced stabile mind' with a sound perception on the "Beauty and Consolation" of our evolutionary product, planet Earth "awesome or awful" with its products of evolution. But "evil" has according to religious people not to do with Gods will but with the 'free will' of human being. From a neurological perception [2] defines 'free will' in the following way: "Your left inferior parietal lobe constantly conjures up vivid images of multiple options for action that are available in any given context, and your frontal cortex suppresses all but one of them. This need to inhibit unwanted or impulsive actions may have been a major reason for the evolution of 'free will'" [2]. From an ethical and moral point of view it sounds like a rather innocent 'brain action', even when the worst crimes against humanity have been committed.

Fortunately, I don't stay in such a sad mood with such bad thoughts for long and in most cases 'Beauty and consolation' of the natural sciences brings me back to reality. In the next session, I will give a neurological example that makes me believe that 'by accident' - as mentioned earlier to avoid the image and -according to my sound perception- wrong view of the 'Supreme Being' (Adonai), because this example clearly defines the conditions for a driving force "up" the evolutionary ladder for "encephalization", (as a counterpart to the second law of thermodynamics that everything falls back into chaos (entropy)).

Recently I made a literature search what conditions a high-fat diet induced mouse model with "brain steatosis" (overgrown brain) must meet in terms of dietary conditions, age, gender and other experimental conditions before it is effective and doesn't lead to ignition of the brain tissue. The behavior / high-fat diet studies listed in table 1 are only a limited representation of the available literature. Just like a small laboratory setting how 'evolution' could work with its so mystified 'favorable traits', here clearly defined, for natural selection. It would be going too far to conduct a further more complete literature study for this editorial. For a more extensive overview of literature, see the review of [4]. However, table 1 provides sufficient experimental design information to initiate the process of evolutionary encephalization, in combination with a positive effect on behavior in some very incidental cases. Generally speaking, a high-fat diet results in inflammatory responses in the brain such as ceramide generation,  $\beta$ -amyloid accumulation, as well as neural apoptosis under the insulin resistant conditions [4,6]. This can be prevented by following the experimental design as in our mouse model (were found by happenstance) [7]

1. For example, to choose a young growing mouse model 6 weeks old [7];
2. Only take males because the estrogenic component has an inflammatory effect [8];
3. Do not expose the experimental animals to a high-fat diet for too long, for example, until this young mouse model has become insulin resistant (IR) after 40 days [7].
4. Not to take extremely high fat concentrations like [8,9] performed in their experimental design with 41% fat, but instead chose intermediate levels for example 24% fat [7]
5. Take not just a high-fat diet with any composition but more specific consisting of bovine lard with the following biochemical peculiarity with especially high levels of Arachidonic acid (C20: 4,  $\omega$ -6; ARA) and Docosapenta-

noic acid (C22: 5,  $\omega$ -3; DPA) for which the need for this is described in detail in [10]

6. Use a high-fat diet containing a high amount of unsaturated very long chain poly unsaturated fatty acids (VLCPUFAs) such as performed in our high-fat diet but also in the study of [11].
7. Adding compound like a type 2 diabetes (T2DM) drug -such as Liraglutide- that helps the lipids pass through the blood brain barrier (BBB) [12];
8. Administration of brain inflammation inhibitors such as

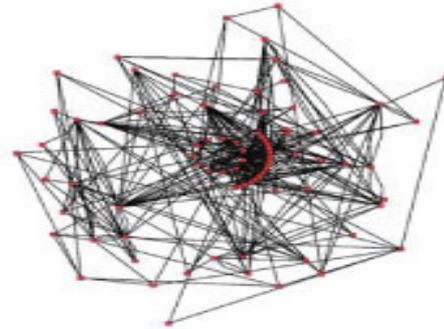


Figure 2: "Adonai" complexity behind the complexity of "evolution". Or according to Richard Dawkins "A deity capable of engineering all the organized complexity in the world, either instantaneously or by guiding evolution ... must already have been vastly complex in the first place ..." [3].

9. melatonin [11]
9. Because different saturated fatty acids (FAs) but also several mono unsaturated fatty acids (MUFAs) such as Palmitic acid (C16:0; PA), Myristic acid (C14:0; MA), and Lauric acid (C12:0; LA) have a blood plasma cholesterol raising potential [13] and cholesterol is a major constituent of the human neocortex [14], the high-fat diet should also contain cholesterol, like in our formulated diet 0.25% cholesterol [7].
10. In addition, oral intake of EPA: DHA at a ratio of 6: 1 by middle-aged rats for one week improved age-related endothelial dysfunction in both the femoral artery and vein [15] and has possibly a stimulating effect at blood vessel formation which is an important evolutionary trait for human brain encephalization.

Briefly summarized, we hypothesize all these ten research variables should result in a better memory performance and cognitive functioning in these rodent models (table 1). Reflecting it towards the evolutionary sciences, "the ladder up" or are these ten preconditions ('favorable traits') just a matter of staggering incomprehensible statistics (chances to meet each other) or more comprehensible algorithmic mathematics (a set of rules that precisely defines a sequence of operations)? Or nevertheless the hand of a "Benevolent Supreme Being"? It can be defined as the complexity ( $\approx$ Adonai) behind the complexity ( $\approx$ evolution). Bringing together all these favorable traits; I repeat, in a countless series of 'breedings' and 'eatings'. So, in summary, going back to our small evolutionary laboratory setting, review-like (table 1). Most high-fat diets based on a westernized eating profile results in brain inflammation [16,17]. Only under specific high-fat conditions which are mainly based on bovine lard given to a juvenile rodent model containing many unsaturated FAs we have indications of 'brain

Table 1: Behavioural changes in rodent models after exposure to a high-fat diet.

Study	Type HF-diet exposure	Rodent model	Behavioural Changes	Source
1)	High saturated fat diet; 20% (w/w) fat (lard)	Long-Evans rats	Learning and memory impairment	21.Greenwood & Winocur 1990
2)	Fructose & High-fat diet induced IR	Middle-aged Rats	Impaired hippocampal synaptic plasticity and cognition	22.Stranahan et al 2008
3)	High-fat diet induced obesity 3 weeks to 9-12 months	Mice C57bl/6J	Metabolic alterations and deficits of learning and hippocampal synaptic plasticity	23.Hwang et al 2010
4)	Western diet (41% fat) & very high fat lard diet (60% fat)	Aged mice, 20-month old male C57BL/6	Increased hippocampal oxidative stress and cognitive impairment implications for decreased Nrf2 signalling	8. Morrison et al 2010;
5)	Western diet (41% fat) & very high fat lard diet (60% fat)	Mice C57BL/6	Cognitive impairment associated with brain inflammation	9. Pistell et al 2010;
6)	High-fat dietary-induced obesity & IR for 20 days	Four weeks administration of Liraglutide in young Swiss TO mice	Improves memory and learning as well as glycaemic control	12. Porter et al 2010
7)	High-fat diet (45 kcal% from fat) vs. Control (10 kcal% from fat)	Mice C57BL/6J	Impair spatial learning in the radial-arm maze	24.Valladolid-Acebes et al 2011;
8)	High-fat diet; Control (10% fat) vs. HF (45% fat)	Juvenile Swiss TO Mice (6-8 weeks old)	Actions of incretin metabolites on locomotor activity, cognitive function and <i>in vivo</i> hippocampal synaptic plasticity	25. Porter et al 2012;
9)	a). Moderately high-fat (MHF) of 35%; b). Obesity resistant lean mice; c) Genetic model of obesity (MC4R).	Mice C57BL6/J (MHF) for 6 months	Olfactory ability and object memory	26. Tucker et al 2012
10)	High-fat diet-induced obesity: HF: (524 kcal per 100 g feed); Co: (346.8 kcal per 100 g feed)	Male C57BL/6J mice (6 weeks old)	Impairment of fear-conditioning responses and changes of brain neurotrophic factors (BDNF and NT-3 content in the brain and	27. Yamada-Goto et al 2012
11)	High-fat diet induced obese IR 24% bovine lard and 0.25% cholesterol	Juvenile mouse (6 weeks) C57bl6	LCMS determined "Brain steatosis" no behavioural tests performed	18.;19, van Ginneken et al 2011, 2013
12)	Very high-fat diet (60% kcal by fat) for 17 days; moderate high fat diet (HFD, 45% kcal by fat) for 8 weeks	Juvenile C57BL/6J male 8 weeks mice	Deleterious effects on synaptic integrity and cognitive behaviour	28. Arnold et al 2014
13)	High-fat diet (HFD) 25% total fat including 11% unsaturated; Control 5% total fat including 2% unsaturated	Adult male Wister rats 160-200 g	Consumption of high-fat diet (HFD) induces oxidative stress in the hippocampus that leads to memory impairment. Melatonin has antioxidant and neuroprotective effects.	29. Alzoubi et al 2018

steatosis' without brain inflammation [18,19] together with improved cognitive function [20].

If these mouse models with "brain steatosis" are a reflection of what happened to the first hominids about 2.4 million years ago in the East African savannah, then it is amazing that precisely those biochemical conditions occurred in terms of lipid composition of the meat of prey animals - especially the ancestors of the African buffalo (*Syncerus caffer*) - which enabled human brain growth [10]. And by this we can explain the exponential growth spurt of *Homo habilis* with its 800 cm<sup>3</sup> brain to that of modern man *Homo sapiens* with its 1500 cm<sup>3</sup> brain. Considering all this, I can only say with complete devotion: "God's works are wonderful and what a wonderful awesome and sometimes awful world (presently with the Corona pandemics) he created".

## References

1. Van Ginneken V. Where Darwin neglected to explain the human-brain encephalization: 1) Ecological arguments supporting the Savannah Dryland (SDL) hypothesis. *ES J Neurol.* 2020;1(1):1005.
2. Ramachandran VS. *The Tell-Tale Brain; unlocking the Mystery of Human Nature.* Published by Windmill Books; ISBN 2011;9780099537595; 357pp.
3. Dawkins R. "The Blind Watchmaker", *Evolutionary Biology*, Norton & Company, Inc; ISBN: 0-393-31570-1986;3.
4. Abbott KN, Arnott CK, Westbrook RF & Tran DMD. The effect of high fat, high sugar, and combined high fat-high sugar diets on spatial learning and memory in rodents: A me-

- ta-analysis. *Neuroscience & Biobehavioural Reviews*. 2019. doi:10.1016/j.neubiorev.2019.08.010.
5. Van Ginneken V & Schouten F. The Expected Pandemic of Mild-Alzheimer (Type 3 Diabetes). How to Combat? *Gastroenterology & Hepatology International Journal* 2019;4(2):000157. doi: 10.23880/ghij-16000157.
  6. Maciejczyk M, Żebrowska E & Chabowski A. Insulin Resistance and Oxidative Stress in the Brain: What's New? *International Journal of Molecular Sciences*, 2019;20(4):874. doi:10.3390/ijms20040874.
  7. Van Ginneken V, de Vries E, Verheij E, van der Greef J. "Brain steatosis" in an obese mouse model during cycles of Famine and Feast: the underestimated role of fat (WAT) in brain volume formation. *Integr Mol Med*. 2017;4(2):1-6.
  8. Morrison CD, Pistell PJ, Ingram DK, Johnson WD, Liu Y, Fernandez-Kim SO, Keller JN. High fat diet increases hippocampal oxidative stress and cognitive impairment in aged mice: implications for decreased Nrf2 signalling. *Journal of Neurochemistry*. 2010;114(6):1581-1589. doi:10.1111/j.1471-4159.2010.06865.x.
  9. Pistell PJ, Morrison CD, Gupta S, Knight AG, Keller JN, Ingram DK, et al. Cognitive impairment following high fat diet consumption is associated with brain inflammation. *Journal of Neuroimmunology*. 2010;219(1-2): 25-32. doi:10.1016/j.jneuroim.2009.11.010.
  10. Van Ginneken V. Where Darwin neglected to explain the human-brain encephalization: 2). The biochemical model supporting the Savannah Dryland (SDL) hypothesis. In press. 2020.
  11. Alzoubi KH, Mayyas FA, Mahafzah R & Khabour. Melatonin prevents memory impairment induced by high-fat diet: Role of oxidative stress. *Behavioural Brain Research*, 2018;336:93-98. doi:10.1016/j.bbr.2017.08.047.
  12. Porter DW, Kerr BD, Flatt PR, Holscher C & Gault VA. Four weeks administration of Liraglutide improves memory and learning as well as glycaemic control in mice with high fat dietary-induced obesity and insulin resistance. *Diabetes, Obesity and Metabolism*. 2010;12(10):891-899. doi:10.1111/j.1463-1326.2010.01259.x.
  13. Grundy SM. What is the desirable ratio of saturated, polyunsaturated, and monounsaturated fatty acids in the diet? *The American Journal of Clinical Nutrition*, 1997;66(4): 988S-990S. doi:10.1093/ajcn/66.4.988s.
  14. Zhang J & Liu Q. Cholesterol metabolism and homeostasis in the brain. *Protein & cell*. 2015;6(4):254-264. <https://doi.org/10.1007/s13238-014-0131-3>.
  15. Gaertner S, Auger C, Farooq MA, Pollet B, Khemais-Benkhiat S, Niazi ZR, et al. Oral Intake of EPA: DHA. 6: 1 by Middle-Aged Rats for One Week Improves Age-Related Endothelial Dysfunction in Both the Femoral Artery and Vein: Role of Cyclooxygenases. *International Journal of Molecular Sciences*. 2020;21(3):920. doi:10.3390/ijms21030920.
  16. Pakiet A, Jakubiak A, Czumaj A, Sledzinski T & Mika A. The effect of western diet on mice brain lipid composition. *Nutrition & metabolism*, 2019;16:81. <https://doi.org/10.1186/s12986-019-0401-4>.
  17. Melo HM, Santos LE & Ferreira ST. Diet-Derived Fatty Acids, Brain Inflammation, and Mental Health. *Frontiers in neuroscience*. 2019;13:265. <https://doi.org/10.3389/fnins.2019.00265>.
  18. Van Ginneken V, Verheij E, Hekman M, van der Greef J, Feskens E, Poelmann R. The comparison of lipid profiling in Mouse brain and liver after starvation and a high-fat diet: a Medical Systems Biology approach. Chapter 3 page 151-186. In: *Biology of Starvation in Humans and other organisms*. Editor: Todd C. Merkin, ISBN: 978-1-61122-546-4, Nova Science Publishers, Inc. 2018.
  19. Van Ginneken VJT, Verheij E, Hekman M, van der Greef J, Feskens EJM, Poelmann RE. Chapter 3: 'The comparison of lipid profiling in mouse brain and liver after starvation and a high-fat diet: A medical systems biology approach'. In: *Low and High-Fat Diets: Myths Vs. Reality*. 2013;105-140.
  20. Tan BL & Norhaizan ME (2019). Effect of High-Fat Diets on Oxidative Stress, Cellular Inflammatory Response and Cognitive Function. *Nutrients*, 11(11): 2579. <https://doi.org/10.3390/nu11112579>
  21. Greenwood CE & Winocur G. Learning and memory impairment in rats fed a high saturated fat diet. *Behavioural and Neural Biology*. 1990;53(1):74-87. doi:10.1016/0163-1047(90)90831-p.
  22. Stranahan AM, Norman ED, Lee K, Cutler RG, Telljohann RS, Egan JM. Diet-induced insulin resistance impairs hippocampal synaptic plasticity and cognition in middle-aged rats. *Hippocampus*, 2008;18(11):1085-1088. doi:10.1002/hipo.20470.
  23. Hwang L-L, Wang C-H, Li T-L, Chang S-D, Lin L-C, Chen C-P. Sex Differences in High-fat Diet-induced Obesity, Metabolic Alterations and Learning, and Synaptic Plasticity Deficits in Mice. *Obesity*. 2009;18(3):463-469. doi:10.1038/oby.2009.273.
  24. Valladolid-Acebes I, Stucchi P, Cano V, Fernández-Alfonso MS, Merino B, Gil-Ortega M. High-fat diets impair spatial learning in the radial-arm maze in mice. *Neurobiology of Learning and Memory*. 2011;95(1):80-85. doi:10.1016/j.nlm.2010.11.007.
  25. Porter D, Faivre E, Flatt PR, Holscher C & Gault VA. Actions of incretin metabolites on locomotor activity, cognitive function and in vivo hippocampal synaptic plasticity in high fat fed mice. *Peptides*. 2012;35(1):1-8. doi:10.1016/j.peptides.2012.03.014.
  26. Tucker KR, Godbey SJ, Thiebaud N & Fadool DA (2012). Olfactory ability and object memory in three mouse models of varying body weight, metabolic hormones, and adiposity. *Physiology & Behavior*. 2012; 107(3): 424-432. doi:10.1016/j.physbeh.2012.09.007.
  27. Yamada-Goto N, Katsuura G, Ochi Y, Ebihara K, Kusak-

- abe T, Hosoda K, et al. Impairment of Fear-Conditioning Responses and Changes of Brain Neurotrophic Factors in Diet-Induced Obese Mice. *Journal of Neuroendocrinology*. 2012;24(8):1120-1125. doi:10.1111/j.1365-2826.2012.02327.x.
28. Arnold SE, Lucki I, Brookshire BR, Carlson GC, Browne CA, Kazi H, et al. High fat diet produces brain insulin resistance, synapto-dendritic abnormalities and altered behaviour in mice. *Neurobiology of Disease*. 2014;67:79-87. doi:10.1016/j.nbd.2014.03.011.
29. Alzoubi KH, Mayyas FA, Mahafzah R & Khabour OF. Melatonin prevents memory impairment induced by high-fat diet: Role of oxidative stress. *Behavioural Brain Research*. 2018;336:93-98. doi:10.1016/j.bbr.2017.08.047.