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Review

Alzheimer's Disease: the Hypotheses, Known and Unknown Connections between UV-Radiation, mtDNA Haplotypes and Life Span – a Review

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Abstract

Alzheimer's disease (AD) is the most common neurodegenerative disease with controversial etiology. One theory claims that AD is due to brain aging affecting mainly the functions of mitochondria, therefore, the factors leading to mitochondrial ageing should lead to the development of Alzheimer's disease. Another theory is that different mitochondrial DNA haplogroups can be predisposition for the onset of the condition. Here we focused on the possible connection between AD and UV radiation using the data on the monthly UV index in Europe, its correlation with mortality rate due to AD and mitochondrial DNA haplogroups distribution. If a link between the two theories is proved, it will mean that UV radiation is a risk factor not only for skin cancer but also for a large group of neurodegenerative diseases amongst which is the Alzheimer's disease.

Keywords

Alzheimer's disease, life-span, mtDNA haplotypes, UV-radiation

INTRODUCTION

Along with Parkinson's disease, Alzheimer's disease (AD) is the most common neurodegenerative disease. With millions of people suffering from Alzheimer's disease across the globe, the prognosis for the future development of AD is for the number of patients to increase. At the same time, little is clear about the origins and the development of this disease.

There are two theories we are going to discuss in this review. The first one looks at the Alzheimer's disease as a hereditary condition. It has been established that in a small proportion of all cases of Alzheimer's dementia, AD is hereditary. Most of the genes that cause Alzheimer's disease have already been studied. The second theory proposes that Alzheimer's disease develops due to brain aging.

Effect of oxidative stress on the brain aging

In most patients with Alzheimer's dementia, the pathogenesis is due to brain aging. Oxidative stress and mitochondrial dysfunction are the major factors behind developing Alzheimer's dementia.[1] Oxidative stress is caused by reactive oxygen species which in healthy individuals are

balanced out by antioxidants.[2] When this balance is disturbed, oxidative stress leads to neuronal loss in great numbers. The brain requires a lot of energy, but in patients with Alzheimer's disease, the neurones' glycolysis is restrained. The neurones depend on the mitochondria for the production of adenosine triphosphate (ATP) and are sensitive to the accumulation of reactive oxygen species.[3]

Oxidative damage leads to deletion in mitochondrial DNA (mtDNA), but the mechanism is under-researched. Oftentimes, point mutations and rearrangements are discovered. There are mutant copies of mtDNA within the cell and their number must reach a certain threshold before clinical symptoms and cell death become visible.[4] According to research from 2009, this threshold is between 70% and 90% of the entire cell mtDNA.[5] The mutations accumulate over time and are found in great numbers in the neurons of the brain because of their reliance on the mitochondria. The outcome is decreased efficiency of the electron transport chains, production of less ATP, and more reactive oxygen species. This represents a 'vicious cycle'.[6]

The process of cell aging was first described by Harman fifty years ago. He suggested that cell aging was due to the production of reactive oxygen species.[2] With the discovery of the enzyme superoxide dismutase, it has been established that reactive oxygen species are generated continuously.[7] Elevated levels of superoxide dismutase increase the life span of the cell and decrease the rate at which telomerase shorten.[8] Decreases in superoxide dismutase lead to the activation of p53, which induces either senescence[9] or apoptosis[10]. The most reactive oxygen species are produced by mitochondria. This is vital for apoptosis.[2,11] Harman's theory of cell aging is further supported by an earlier work by Pearl who claimed that cell death depended on the metabolic rate.[12] There is the possibility that reactive oxygen species simply correlate with cell aging but do not cause it.[13]

The main reason why mitochondria generate reactive oxygen species is the respiratory chain. In the respiratory chain, which consists of sites I to IV, energy is produced from two stable molecules – reduced nicotinamide adenine dinucleotide (NADH) and semiquinone flavin adenine dinucleotide (FADH). Site I contains flavin mononucleotide (FMN), which is responsible for the release of reactive oxygen species.[14] In fact, the production of reactive oxygen species is due to the 'slippage' of electrons at site I and site III which directly react with O_2 or another acceptor. According to some estimation, between 1% and 5% of all the oxygen that passes through site I and site III escapes as superoxide. If the redox potential at site I[15] and site III[16] is increased, we will have a greater production of reactive oxygen species. The same result is visible if we inhibit the electron transport at site I or site III.[17-19] Some inhibitors could lead to reverse electron flow and, thus, a greater number of reactive oxygen species.[18,19] They are harmful to the cell but are also a part of signaling pathways.[20]

An increase in oxygen consumption does not increase the production of reactive oxygen species but reduces it. The only instance when increased oxygen consumption increases the levels of reactive oxygen species is when there are a greater number of mitochondria.[13] About 0.2% of the total oxygen consumption is used for reactive oxygen species generation.[17,18] To increase that production, there has to be a lack of adenosine diphosphate (ADP) for oxidative phosphorylation[19] or uncoupling proteins at play[21].

With aging, changes occur in the mitochondria. An example of such alteration is the oxidation of mtDNA. Oxidative damage can be explained by the lack of histone protection, its close proximity to reactive oxygen species, and the few repair mechanisms.[22] This is part of the so-called 'vicious cycle' – the more reactive oxygen species are generated, the more mutations occur, leading to even greater production of reactive oxygen species, and so on and so forth. Many studies support the existence of connection between aging, accumulation of mtDNA mutations, mitochondrial dysfunction, and increased production of reactive oxygen species. Aging mitochondria produce more reactive oxygen species and less ATP.[23]

Mitochondrial haplogroups as a predisposition for Alzheimer's disease

Mitochondria are exceptional because they have their own DNA in the form of a circular, double-stranded molecule.[24] Mitochondrial DNA still affects the cell cycle and the lack of it can block the progression from G1 to S-phase.[21] The number of mutant copies increases with every cell cycle. The mutant copies exist simultaneously with the wild-type and this state is known as heteroplasmy. The first mutant copies are created in mid-thirties of one's life. The rearrangements most commonly involve deletions. A study by Kujoth et al.[25] contradicts the suggestion that the greater the number of mutations, the greater the generated reactive oxygen species. Research carried out on mutator mice shows that they do not exhibit defects in cell proliferation or a higher concentration of reactive oxygen species. This is also valid in humans.[26] However, the generated reactive oxygen species are a trigger for apoptosis. Mutations are vital since they can multiply in numbers by colonial expansion. This is why there is ground to believe that age is the main factor for the development of Alzheimer's disease.

In a study from 1994, Corral-Debrinski et al.[27] concluded that patients under the age of 75 with AD had 15% more deletions in their mtDNA compared to people of the same age but without the neurodegenerative disease. In 2004, Coskun et al. stated that mutations of mitochondrial DNA were, in general, more common amongst AD patients. Some of the changes in the structure of DNA seem specific to Alzheimer's dementia. They occur on sites responsible for replication and transcription. In 2010, the same team[28] proved that mitochondrial dysfunction was common amongst neurodegenerative conditions by finding mtDNA mutated copies in blood cells.

We can hypothesize as to whether some people are ge-

netically more prone to developing Alzheimer's disease because of their mtDNA. Studies have long taken interest in the mtDNA haplogroups. Initially discovered to allow us to follow human migration back to the mitochondrial Eve, today they can be connected to a number of illnesses. This hypothesis stands some ground when it comes to Alzheimer's disease. Van der Walt and colleagues[29] analyzed the European mitochondrial haplogroups and discovered that males from haplogroup U tend to develop Alzheimer's disease more often. The haplogroup UK is found more often in patients with AD compared to members of controlled groups.[30] If we accept the possibility that some haplogroups make people more susceptible to Alzheimer's dementia, then we must also test the opposite hypothesis, namely the chance that some other haplogroups may have a protective effect. This could be a way of explaining the vastly varying mortality rate of patients with Alzheimer's disease in countries across Europe **(Fig. 1)**. Some states fall into the most endangered group in the world, while others barely report any casualties. An early research from 1999 by Chagnon et al.[31] proposed that haplogroup T could have a shielding effect against Alzheimer's disease. Samples from patients who suffer from AD show that haplogroup T is rather under-represented compared to members of controlled groups.

The hypothesis that mtDNA is a risk factor for the development of Alzheimer's disease is unproven. The results from different tests and experiments are inconsistent to support an unequivocal conclusion. Mitochondrial dysfunction certainly causes the overproduction of reactive oxygen species. An experiment in which mtDNA was taken from a person with Alzheimer's disease and was then transferred to a cell with no mtDNA showed that the host cell began generating reactive oxygen species. The enzymes that should have neutralized the species appeared heavily reduced. The suggestion that mtDNA haplogroups may be a predisposition to Alzheimer's disease spurs from the knowledge that a risk factor in the pathogenesis is allele e4 of apolipoprotein E.[32] We also know that mtDNA is inherited from the mother's side and does not recombine. If proven that some of the haplogroups make the bearer vulnerable, this finding will support the theory that Alzheimer's disease is a hereditary condition. Research on mtDNA haplogroups has so far connected them to life expectancy **(Table 1)** and some other diseases.

Table 1. mtDNA haplogroups by frequency in some European states. Countries below the bold line are among the countries with the lowest mortality rate from AD in the world

	HV	H	H5	J	U
Finland	0	36.3	23	5.9	0.8
Sweden	0.5	45.8	2.2	7.7	2.8
Switzerland	0.4	47.9	4.4	11.5	0.4
Spain	0.7	44.1	2.6	6.6	1.8
France	2	44.3	3.1	7.7	1.4
UK	0	44.7	4.1	11.5	2.7
Italy	2.9	40.2	3.9	8.1	2.7
Germany	0.5	44.8	4.8	9	0.8
Poland	1	43.9	4.5	7.9	1.4
Czech Republic	1.7	40.2	4	10	0.6
Bulgaria	3.8	41.9	3.2	7.7	2.5
North Macedonia	0	45	4	7.5	2
Bosnia and Herzegovina	1.1	45.8	5.6	8.3	1.1

Research on brain tissue from a patient with Alzheimer's disease shows abnormalities in the structure of the functioning mitochondria called 'giant' mitochondria.[33,34] The depleted number of mitochondria become sausage-shaped and create a linked system. The new shape could be the result of abnormal patterns of fusion and fission. The changes reflect on the proteins enabling fusion and fission. These are two vital processes for the mitochondria. Fusion allows two mitochondria to merge into one, thus minimizing the number of mutated mtDNA copies.[35] Fission eliminates by autophagy the mitochondria that cannot be repaired.[36] Without the timely executed degradation of mitochondria, the cell faces death.[37] Fusion and fission are very sensitive to the conditions within the cell.[38,39] Fission can be inhibited by a higher concentration of glucose. This can lead over time to the overproduction of reactive oxygen species. The low concentration of enzymes, like peroxide, can affect the structure of the mitochondria because of the organelle's inability to eliminate reactive oxygen species.[40] The importance of these two processes comes from their ability to eliminate the damaged beyond repair mitochondria[41] but also to keep intact the mtDNA[44]. Any abnormality could

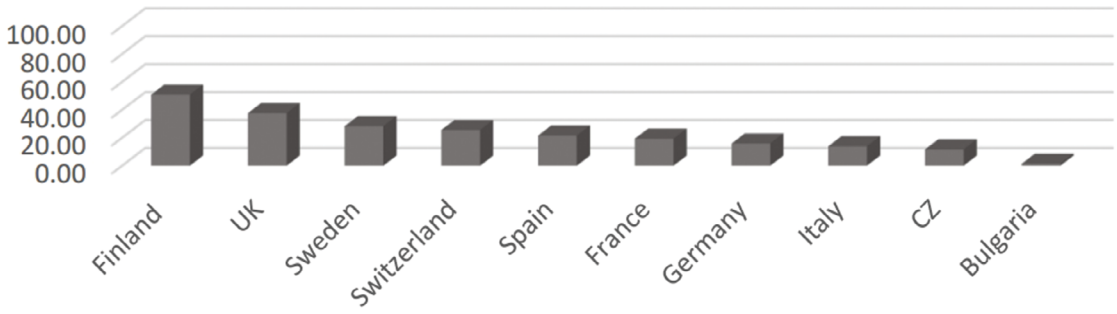

Figure 1. Comparison by mortality rate from Alzheimer's disease in some European countries. Age is standardized.

lead to swelling, dysfunction, and even fragmentation, and the latter causes an apparent increase in the production of reactive oxygen species.

In countries with a high mortality rate, haplogroup J, which is considered by some studies to be over-represented in patients with Alzheimer's disease, is indeed highly represented where it is one out of the three most common haplogroups.[31] However, in countries with few cases of death from Alzheimer's disease, haplogroup J is also well represented, where it is again one out of the three most common haplogroups. Research claims that haplogroup H5 is another risk factor for Alzheimer's disease.[43] Haplogroup H5 has a high occurrence rate in countries like Iceland, Norway, Belgium, France, United Kingdom, and Latvia. These are also countries which on a global scale report a high mortality rate as a result of Alzheimer's disease. However, there appears to be a high occurrence of haplogroup H5 also amongst countries that report one of the lowest mortality rates in the world, such as Bosnia and Herzegovina and North Macedonia. It is clear that there is great variation in the expression of haplogroup H5, hence, it cannot be concluded what its role may be as a factor in Alzheimer's disease. Another study claims that haplogroup U is under-represented in female and over-represented in male Alzheimer's disease patients.[43] As can be seen in **Table 1**, we cannot judge the gender specifications of the haplogroups since the data is disaggregated by sex for each state. However, we can see that in states with a high mortality rate, haplogroup U is usually one of the least represented haplogroups. We can also observe that in countries with low mortality rate, haplogroup U is common. In none of these countries does haplogroup U fall into the group of sub-units of mtDNA that are least represented.

There is evidence that the HV cluster is also connected to Alzheimer's disease development, regardless of sex. **Table 1** further supports this claim. The table shows that in states with a high mortality rate, the HV cluster varies from 0.2 to 8. In the countries with the lowest mortality rate, the HV cluster varies from 0 to 4.8.

A recent study of groups in the Polish population found that haplogroup H was another risk factor. It is believed that haplogroup H and, in particular, some of its subdivisions like H3, H4, H5 and H6 are associated with coupled oxidative phosphorylation. It follows that they should lead to an increased production of reactive oxygen species.

In summary, even small risk factors affecting mtDNA and haplogroups could have a pathogenic effect. It is true that some genetic abnormalities could resemble early aging[5], but there is also ample evidence from studies showing a direct link between mtDNA mutations and a shortened lifespan[44].

CONCLUSIONS

Globally, Alzheimer's disease affects about 3% of adults 60 and older, yet its etiology is still a mystery. In addition to the ones we analyzed, there may be other factors involved as well. Both of the hypotheses in the present article are still not proven. However, they pose important issues regarding the causes of Alzheimer's dementia, its risk factors, and methods for delaying or perhaps preventing the start of this degenerative disease. Studies on the causes of this illness will continue to focus on such issues.

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Competing Interests

The authors have declared that no competing interests exist.

Author contributions

M.D-F. and P.Z. conceived of the presented idea. M.D-F. and V.B. wrote the manuscript. All authors discussed and commented on the manuscript.

REFERENCES

1. Yan HM, Wang X, Zhu X. Mitochondrial defects and oxidative stress in Alzheimer disease and Parkinson disease. Free Radic Biol Med 2013; 62:90–101.
2. Harman D. The aging process. Proc Natl Acad Sci USA 1981; 78:7124–8.
3. Parihar MS, Brewer GJ. Mitoenergetic failure in Alzheimer disease. Am J Physiol Cell Physiol 2007; 292:C8–23.
4. Swerdlow RH. Does mitochondrial DNA play a role in Parkinson's disease? A review of cybrid and other supportive evidence. Antioxid Redox Signaling 2012; 16:950–64.
5. Wallace DC, Fan W. The pathophysiology of mitochondrial disease as modeled in the mouse. Genes Dev 2009; 23:1714–36.
6. Yen TC, Su JH, King KL, et al. Ageing-associated 5 kb deletion in human liver mitochondrial DNA. Biochem Biophys Res Commun 1991; 178:124–31.
7. McCord JM, Fridovich I. Superoxide dismutase. An enzymic function for erythrocuprein (hemocuprein). J Biol Chem 1969; 244(22):6049–55.
8. Serra V, von Zglinicki T, Lorenz M, et al. Extracellular superoxide dismutase is a major antioxidant in human fibroblasts and slows telomere shortening. J Biol Chem 2003; 278:6824–30.
9. Blander G, De Oliveira RM, Conboy CM, et al. Superoxide dismutase 1 knock-down induces senescence in human fibroblasts. J Biol Chem 2003; 278(40):38966–9.
10. Macip S, Igarashi M, Berggren P, et al. Influence of induced reactive oxygen species in p53-mediated cell fate decisions. Mol Cell Biol 2003; 23(23):8576–85.
11. Green DR. Apoptosis. Death deceiver. Nature 1998; 396(6712):629–30.

12. Pearl R. The Rate of Living (London: University of London Press), 1928.
13. Balaban RS, Nemoto S, Finkel T. Mitochondria, oxidants, and aging. Cell 2005; 120(4):483–95.
14. Liu Y, Fiskum G, Schubert D. Generation of reactive oxygen species by the mitochondrial electron transport chain. J Neurochem 2002; 80:780–7.
15. Kushnareva Y, Murphy AN, Andreyev A. Complex I-mediated reactive oxygen species generation: modulation by cytochrome *c* and NAD(P)+ oxidation-reduction state. Biochem J 2002; 368:545–53.
16. Chen Q, Vazquez EJ, Moghaddas S, et al. Production of reactive oxygen species by mitochondria: central role of complex III. J Biol Chem 2003; 278:36027–31.
17. Staniek K, Nohl H. Are mitochondria a permanent source of reactive oxygen species? Biochim Biophys Acta (BBA)-Bioenergetics 2000; 1460(2-3):268–75.
18. St-Pierre J, Buckingham JA, Roebuck SJ, et al. Topology of superoxide production from different sites in the mitochondrial electron transport chain. J Biol Chem 2002; 277:44784–90.
19. Loschen G, Flohe L, Chance B. Respiratory chain linked H_2O_2 production in pigeon heart mitochondria. FEBS Lett 1971; 18:261–4.
20. Nemoto S, Takeda K, Yu ZX, et al. Role for mitochondrial oxidants as regulators of cellular metabolism. Mol Cell Biol 2000; 20:7311–8.
21. Crider DG, Garcia-Rodriguez LJ, Srivastava P, et al. Rad53 is essential for a mitochondrial DNA inheritance checkpoint regulating G1 to S progression. J Cell Biol 2012; 198:793–8.
22. Wiesner RJ, Zsurka G, Kunz WS, et al. Mitochondrial DNA damage and the aging process – facts and imaginations. Free Radic Res 2006; 40(12):1284–94.
23. Shigenaga MK, Hagen TM, Ames BN. Oxidative damage and mitochondrial decay in aging. Proc Natl Acad Sci USA 1994; 91:10771–8.
24. Legros F, Malka F, Frachon P, et al. Organization and dynamics of human mitochondrial DNA. J Cell Sci 2004; 117:2653–62.
25. Kujoth GC, Hiona A, Pugh TD, et al. Mitochondrial DNA mutations, oxidative stress, and apoptosis in mammalian aging. Science 2005; 309(5733):481–4.
26. Hutter E, Skovbro M, Lener B, et al. Oxidative stress and mitochondrial impairment can be separated from lipofuscin accumulation in aged human skeletal muscle. Aging Cell 2007; 6:245–56.
27. Corral-Debrinski M, Horton T, Lott MT, et al. Marked changes in mitochondrial DNA deletion levels in Alzheimer brains. Genomics 1994; 23(2):471–6.
28. Coskun PE, Wyrembak J, Derbereva O, et al. Systemic mitochondrial dysfunction and the etiology of Alzheimer's disease and down syndrome dementia. J Alzheimers Dis 2010; 20(Suppl 2):S293–310.
29. Van der Walt JM, Dementieva YA, Martin ER, et al. Analysis of European mitochondrial haplogroups with Alzheimer disease risk. Neurosci Lett 2004; 365(1):28–32.
30. Lakatos A, Derbeneva O, Younes D, et al. Association between mitochondrial DNA variations and Alzheimer's disease in the ADNI cohort. Neurobiol Aging 2010; 31(8):1355–63.
31. Chagnon P, Gee M, Filion M, et al. Phylogenetic analysis of the mitochondrial genome indicates significant differences between patients with Alzheimer disease and controls in a French-Canadian founder population. Am J Med Genet 1999; 85:20–30.
32. Carrieri G, Bonafè M, De Luca M, et al. Mitochondrial DNA haplogroups and APOE4 allele are non-independent variables in sporadic Alzheimer's disease. Human Genetics 2001; 108(3):194–8.
33. Murakoshi M, Osamura Y, Watanabe K. Mitochondrial alterations in aged rat adrenal cortical cells. Tokai J Exp Clin Med 1985; 10:531–6.
34. Tandler B, Hoppel CL. Studies on giant mitochondria. Ann NY Acad Sci 1986; 488:65–81.
35. Seo AY, Joseph AM, Dutta D, et al. New insights into the role of mitochondria in aging: mitochondrial dynamics and more. J Cell Sci 2010; 123(15):2533–42.
36. Narendra D, Tanaka A, Suen DF, et al. Parkin is recruited selectively to impaired mitochondria and promotes their autophagy. J Cell Biol 2008; 183:795–803.
37. Wohlgemuth SE, Seo AY, Marzetti E, et al. Skeletal muscle autophagy and apoptosis during aging: Effects of calorie restriction and life-long exercise. Exp Gerontol 2010; 45:138–48.
38. Chan DC. Mitochondrial fusion and fission in mammals. Annu Rev Cell Dev Biol 2006; 22:79–99.
39. Chen H, McCaffery JM, Chan DC. Mitochondrial fusion protects against neurodegeneration in the cerebellum. Cell 2007; 130:548–62.
40. Jendrach M, Mai S, Pohl S, et al. Short- and long-term alterations of mitochondrial morphology, dynamics and mtDNA after transient oxidative stress. Mitochondrion 2008; 8:293–304.
41. Twig G, Elorza A, Molina AJ, et al. Fission and selective fusion govern mitochondrial segregation and elimination by autophagy. EMBO J 2008; 27:433–46.
42. Liesa M, Palacin M, Zorzano A. Mitochondrial dynamics in mammalian health and disease. Physiol Rev 2009; 89:799–845.
43. Santoro A, Balbi V, Balducci E, et al. Evidence for sub-haplogroup h5 of mitochondrial DNA as a risk factor for late onset Alzheimer's disease. PloS One 2010; 5(8):e12037.
44. Trifunovic A, Wredenberg A, Falkenberg M, et al. Premature ageing in mice expressing defective mitochondrial DNA polymerase. Nature 2004; 429:417–23.

Болезнь Альцгеймера: гипотезы, известные и неизвестные связи между UV-излучением, гаплотипами мтДНК и продолжительностью жизни – обзор

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Резюме

Болезнь Альцгеймера (БА) – наиболее распространённое нейродегенеративное заболевание с противоречивой этиологией. Одна из теорий утверждает, что болезнь Альцгеймера обусловлена старением мозга, затрагивающим в основном функции митохондрий, следовательно, факторы, приводящие к митохондриальному старению, должны приводить к развитию болезни Альцгеймера. Другая теория состоит в том, что различные гаплогруппы митохондриальной ДНК могут быть предрасположены к возникновению этого состояния. Здесь мы сосредоточились на возможной связи между БА и UV-излучением, используя данные о месячном UV-индексе в Европе, его корреляции со смертностью от БА и распределением гаплогрупп митохондриальной ДНК. Если связь между двумя теориями будет доказана, это будет означать, что UV-излучение является фактором риска не только рака кожи, но и большой группы нейродегенеративных заболеваний, среди которых болезнь Альцгеймера.

Ключевые слова

болезнь Альцгеймера, продолжительность жизни, гаплотипы мтДНК, UV-излучение