Mitochondria by Exogenous UV?

Thomas Prevenslik
Researcher
QED Radiations, Berlin, Germany

Abstract: Endogenous DNA damage is thought to occur by oxidative stress in cells of internal organs in contrast to exogenous DNA damage of the skin from solar UV. However, by simple QED, endogenous UV produced in nanoscale features of cellular structures under metabolic heating directly causes DNA damage, although the UV may create oxidative stress that indirectly damages DNA. Simple QED is the consequence of the Planck law that denies atoms in nanostructures the heat capacity to conserve heat by an increase in temperature. Because of the high surface-to-volume ratio of nanostructures, metabolic heat is almost totally confined to their surfaces, and the surface heat as EM energy provides the confinement by which standing EM radiation is created inside the nanostructure. Once the surface heat is expended in creating the standing radiation, the EM confinement vanishes, and the EM radiation is free to damage DNA or create oxidative stress in surrounding tissue. Body temperature produced by metabolic heat also induces nanostructures to emit EM radiation. At UV levels, DNA is damaged, but the UV also produces ATP and excites enzymes for DNA repair, the delicate balance of which allowing the DNA mutated by UVC on the early Earth to be the ancestor of all life in Darwin's origin of the species. To illustrate the balance of UV induced ATP synthesis and DNA damage, the mitochondria or other cells are simulated by a single nanoparticle (NP) producing endogenous UVC radiation upon entering the blood stream in vaccination adjuvants.

Keywords –ATP synthesis, DNA damage, Endogenous, Nanoparticles, Planck law, UVC

I. INTRODUCTION

Mitochondrial survival depends on ATP synthesis with the DNA maintaining genomic information. However, the preservation of the DNA genome is continuously threatened by damage from exogenous and endogenous sources. Exogenous sources of DNA damage are environmental factors [1] external to the human body comprising chemicals including UV and ionizing radiation. Indeed, solar UV is the leading cause of skin cancer in humans. UV radiation is categorized into UVC (190–290 nm), UVB (290–320 nm) and UVA (320–400 nm). Maximum DNA absorption occurs in the UVC at about 260 nm. Today, the UVC does not reach the Earth today because of the ozone layer, but high UVC levels present on the early Earth were thought [2] to influence cellular evolution. DNA absorption of EUV radiation (< 260 nm) is dramatically reduced from UVC levels, and therefore the EUV may be ignored in ATP production and DNA damage. UVC more efficiently forms pyrimidine dimers compared to UVB while UVA damages DNA by strand breaks. Hence, UVC is widely used in vitro as the upper bound DNA damage agent because of maximal absorption by DNA.

In contrast, endogenous DNA damage is widely thought to be a consequence of oxidative stress induced in chemiosmosis [3] by aberrant electron chain reactions with dissolved oxygen because of the H+ proton gradient across the mitochondrial inner membrane. Oxidative stress produces reactive oxygen species (ROS) that induce a total of approximately 100 different DNA base lesions, the consequences of which are regulated by restricting respiration in mitochondria to protect cellular components. ROS attack the DNA backbone [4] causing an estimated 2300 single strand breaks per cell per hour in mammalian cells. Although ROS produced in mitochondria are thought to be the major source of DNA damage, other endogenous sources [1] of DNA damage include replication errors, spontaneous base deamination, and DNA methylation. Regardless, each ROS mechanism requires the mitochondrial to produce EM energy to initiate the respective enzyme action, but the source of EM energy forming ROS has never been identified.

DNA damage aside, ATP synthesis is explained by Mitchell's chemiosmosis theory [3] in mitochondria by oxidative phosphorylation from the energy of a proton H+ gradient,

$$ADP + Pi + H+ \rightarrow ATP + H2O$$

However, chemiosmosis has a history of controversy. Although a high-energy chemical intermediate provided a more rational explanation, chemiosmosis survived as the ATP mechanism only because the high-energy intermediate was never found. Oxidative phosphorylation in mitochondria occurs by a chain of complex redox reactions with electron transfer from donors to acceptors assisted by enzymes. The electron transfer chain is sequential and may be interrupted at any point by the absence of the necessary enzyme, or the production of ROS upon collision with oxygen. Because the sequential requirement of the electron chain is unlikely, chemiosmosis by hydrolysis is proposed superseded [5] by ATP synthesis in a dehydration reaction mediated by UV.

$$ADP + Pi + UV \rightarrow ATP + H2O$$

In mitochondria, UV is produced by simple QED converting [6] metabolic heat Q from ATP that binds to cristae surfaces or the temperature T of surrounding tissue. Simple QED is based on the Planck law that requires the heat capacity of the atom to vanishe under EM confinement in nanostructures. Heat Q therefore cannot be conserved by an increase in temperature, and instead standing EM radiation is created across the dimension d of the nanostructure having half-wavelength λ/2. Simple QED for a nanoparticle (NP) is shown in Fig. 1.

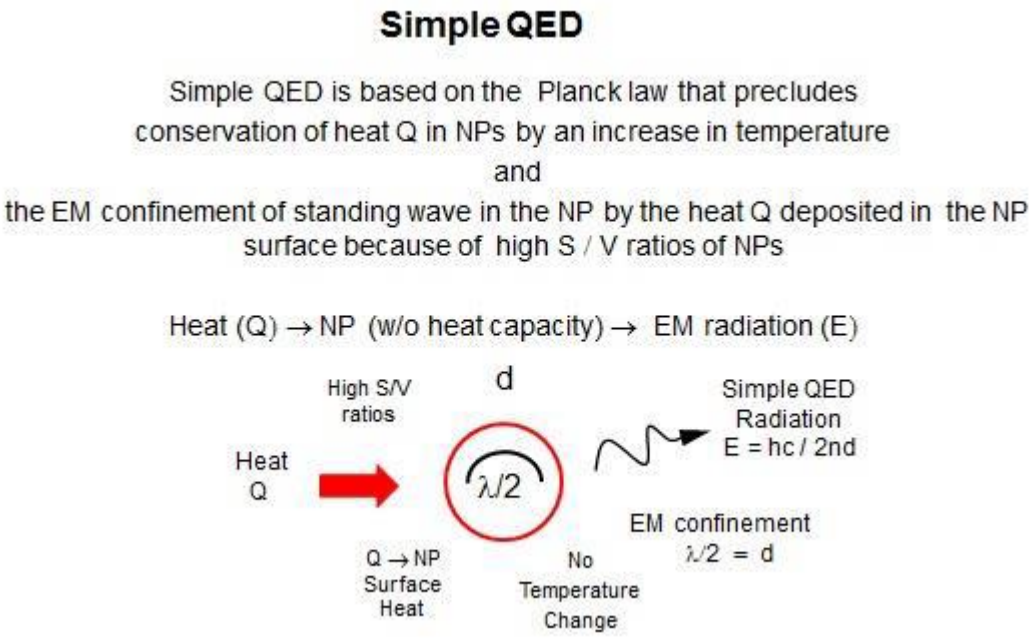

Figure 1. Simple QED conversion of heat to EM radiation

EM confinement occurs by the high surface-to-volume ratio of nanostructures that requires the heat Q to almost totally be confined in the surface, the heat as EM energy itself providing the brief EM confinement to create the EM wave standing in the nanostructure.

ATP synthesis in mitochondria is mediated by simple QED induced UV radiation standing between adjacent cristae surfaces. The standing UV is created from the heat of ATP binding to cristae and heat Q from thermal surroundings as noted in Fig. 2.

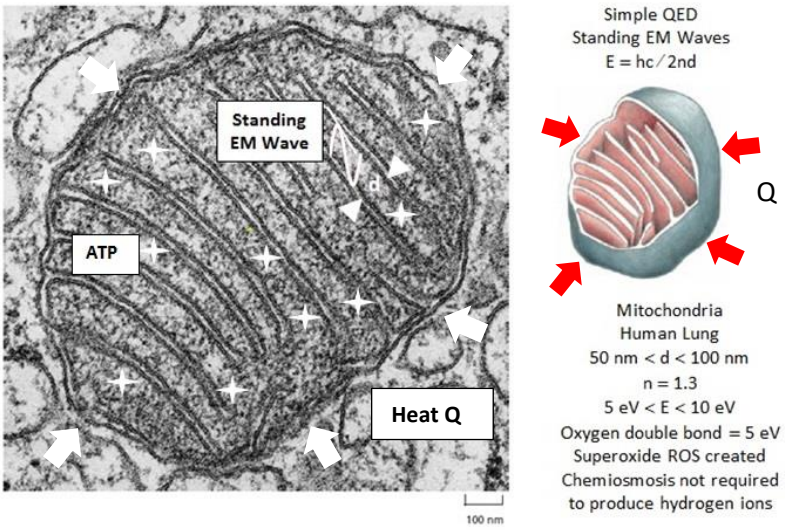

Figure 2. Human Lung Mitochondria providing Heat (ATP + Q) to EM standing waves

The EM wave standing in the mitochondria matrix with a spacing d between adjacent cristae is defined by (50 > d > 100 nm). ATP molecules – white stars – are shown to bind and heat cristae of the mitochondria in Fig. 2. But the thermal heat Q from the mitochondrial surroundings also heats the cristae as shown later for a single NP. The Planck energy E of the standing EM waves based on a refractive index n = 1.3 is beyond the UVC at 5 to 10 eV.

In UV mediated dehydrogenation, the UV produces the oxygen anion superoxide by breaking the double bond of dissolved oxygen and the H+ protons from the OH radical. The UV radiation also creates ADP from breaking down glucose from foods. The ATP is produced from UV in the presence of ADP and Pi, but the UV also forms ROS. Whether hydrolysis or dehydration, ROS induced DNA damage is a consequence of ATP synthesis, i.e., absent ATP synthesis there is no DNA damage.

Unlike chemiosmosis, simple QED does not depend on the H+ gradient across the inner membrane to produce ATP by hydrolysis. For over 50 years, chemiosmosis claimed to synthesize ATP by the unlikely sequence of electron chain reactions powered by the H+ proton gradient across the inner membrane, when in fact simple QED was producing the ATP by UV mediated dehydrogenation.

II. PURPOSE

In support of simple QED, the endogenous UV produced between adjacent cristae requires experimental verification. However, the UV produced from the heat Q produced by binding ATP to cristae surfaces is not convenient because of experimental difficulties. Indeed, measuring the metabolic heat Q from nanostructure features of any cell is problematic.

But thermal bath temperatures from metabolic heat Q avoid details of cellular features.

Experimentally, the EM radiation from a NP in a water bath simulating biological nanostructures in blood is comparatively more tractable as UV photons produced by simple QED could be measured at different temperatures. Since UV absorption of water is not significant, simple QED may be experimentally verified in a UV transparent cuvette. The creation of UV photons within the NP is very rapid with the UV photon production depending on how quickly the heat from the bath is absorbed by the NP. The purpose of this paper is to present a simple QED analysis of a single NP subject to heat flow Q from a thermal bath.

III. ANALYSIS

Simple QED predicts aluminum hydroxide NPs in vaccination [7] adjuvants damage DNA in the brain by producing UVC radiation. In this analysis, a spherical 80 nm NP in a blood or a water bath producing UVC from heat Q in the surroundings is illustrated in in Fig. 3. The Planck energy E of the UVC photon, $E = hc/\lambda = 7.82 \times 10^{-19}$J is created in the NP from surface heat Q in time $\tau = 2d / (c/n)$. For a NP having refractive index n = 1.4, τ = 0.75 fs. Hence, the heat $Q = E/\tau \sim 1$ mW. But this does not happen. The next UVC photon cannot be created until the NP surface temperature is recovered. Fourier's law valid only for atoms in blood having heat capacity kT is noted in the region R > Rs. The recovery of the initial temperature change ΔT taken from [8] depends on the thermal diffusivity α of blood, $\alpha = K_b/\rho C$, where K_b, ρ, and C are the thermal conductivity, density, and specific heat. Numerically, $\alpha = 1.24 \times 10^{-7}$ m^2/ s and K_b = 0.52 W /m-°K.

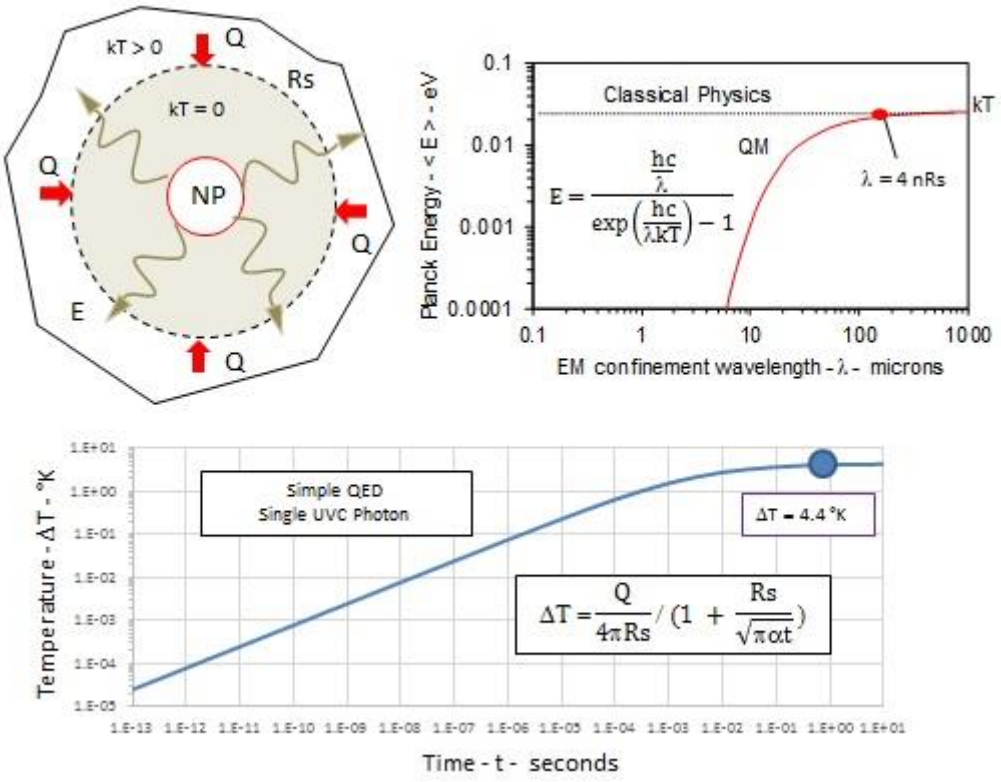

Figure 3. Recovery of NP surface temperature following single UVC emission

The radius Rs at which blood atoms have thermal kT energy is illustrated in Fig. 3. The Planck law at 300 °K shows atoms to have thermal kT energy at EM confinement wavelengths $\lambda > 200$ microns. For blood having refractive index n = 1.4, the radius $Rs = \lambda/4n \sim 36$ microns. No temperature changes occur for R < Rs including the NP. What this means is the heat flow Q from the bath for R < Rs at temperature T is converted at Rs to EM radiation in the far IR and upon being absorbed at the NP surface produce the UVC photon. For a temperature change $\Delta T \sim 5$ K, the recovery time is about 1 s, or the NP produces about 1 UVC photon per second which can easily be measured in a water bath. Daily, the single 80 nm NP presents a significant burden on DNA repair as about 86,000 UVC photons are emitted into the blood stream, the UVC photons providing both ATP synthesis and damage DNA.

IV. DISCUSSION

A. Comparisons of UV induced DNA damage

Endogenous DNA damage to mitochondria by UVC radiation from NPs is expected to severely affect replication and transcription. Since the 1950's, exogenous DNA damage from X-rays, UV, and chemicals was known [9] to produce genetic changes leading to cancer. Unlike exogenous DNA damage that can be avoided, endogenous DNA damage to mitochondria that occurs during normal metabolism is unavoidable. It is estimated [10] that endogenous DNA damage in each human cell causes approximately 70,000 lesions per day. Lesions include single-strand breaks which in part occur to ROS damage during metabolism, the double-strand breaks occurring from single-stand breaks. Unlike simple QED, endogenous DNA damage is not thought [11] caused by UVC radiation from NPs or nanoscopic features of cellular structure, but rather by chemical instability including DNA replication and repair. ROS as by-products of normal metabolism are thought to create 50,000 DNA lesions per human cell per day including base modifications, single-strand breaks, double strand breaks, and inter-strand cross-links. Except for now, a source of EM energy to create the ROS is not identified. Nevertheless, endogenous DNA damage from a single NP by simple QED of about 86.000 lesions per day is supportive of [9-11] estimates. But ROS are not a magic mechanism arising from the need of a reason to explain DNA damage, but rather are the product of simple QED induced UVC.

But NPs need not be man-made.

In 1964, after Mitchell proposed [3] chemiosmosis, electron microscopy [12] revealed numerous submicron particles (SMP) comprising spherical vesicles attached by a stalk to cristae of the mitochondria. A typical micrograph is shown in Fig. 4.

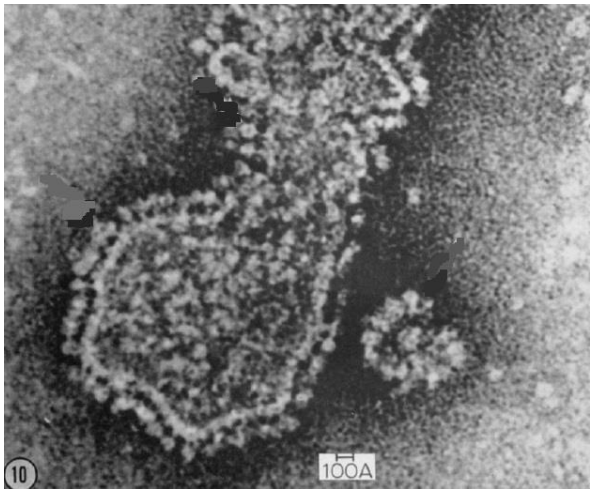

Figure 4. SMP from Ultrasonic treated Mitochondria

The SMP have diameters < 8 nm located on the surface of the cristae, but also are dispersed throughout the matrix. Like the aluminum NPs in vaccinations, the SMP convert thermal kT energy in the mitochondrial blood to EM radiation not in the UVC, but rather in the EUV at wavelength $\lambda = 2nd <$ 30 nm. Since DNA is not absorptive in the EUV, DNA damage in the EUV may be neglected. In mitochondria, the DNA damages is caused by EM radiation in the UVC standing between cristae spacings from 50 to 100 nm. as shown Fig. 2. DNA damage by biological globular NPs in the UVC requires diameters closer to 100 nm.

In this regard, reconstituted phospholipid vesicles simulating SMP with a larger diameter of about 100 nm were shown [13] to produce ATP without intact mitochondria. Consistent with chemiosmosis, the reconstituted vesicles were thought functioning as a proton H+ pump as the vesicles started to take up protons from the medium upon illumination. If ADP and inorganic phosphate was added, the vesicles generated ATP marking the first time that a reaction of oxidative phosphorylation was observed in a system which contained no intact mitochondria or SMP. Contrarily, simple QED suggests the vesicles produced UVC that removed electrons while charging the vesicle positive giving the false impression that chemiosmosis was taking up protons from the surrounding medium.

B. UV induced ATP

In mitochondria, EM radiation is produced between adjacent cristae having a frequency dependent on the spacing d between cristae surfaces. Standing EM radiation between cristae [14] is illustrated in Fig. 5A. Scale bar = 100 nm. Since the spacing varies from 50 to 120 nm, the simple QED induced endogenous EM radiation is broad band, but UVC is taken as typical because of mitochondrial evolution [2] from exogenous UVC predicted on the early Earth. The ATP synthase arrange [14] in rows of pairs along the cristae ends, a schematic cross-section through adjacent cristae is shown in Fig. 5B. The ATP synthase occur in pairs at the ends of cristae to allow each site to provide a different amount of EM energy to the standing EM wave on the nearest flat surface. The ATP synthase pair is necessary as the amount of EM energy to the respective flat surface depends on the spacing d which can vary throughout the mitochondria. The ATP synthase as an enzyme converts ADP + Pi + UV → ATP, the ATP then binding to flat cristae surfaces to generate heat which is converted by simple QED to EM energy and at UVC levels.

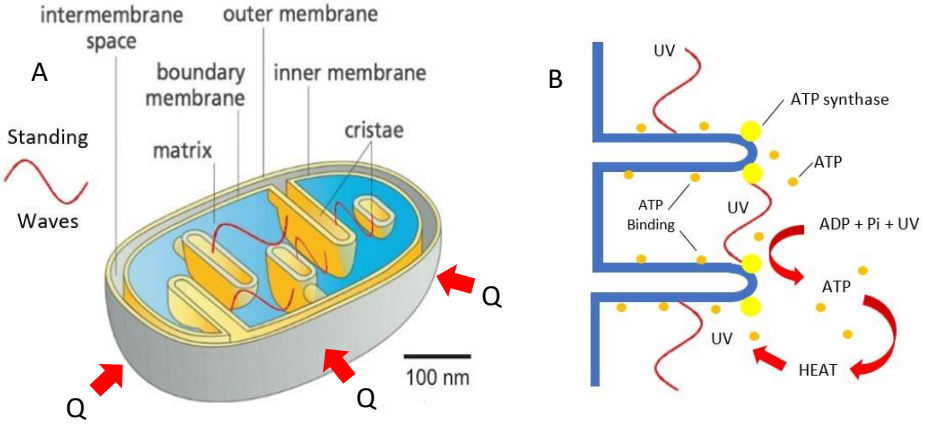

Figure 5. ATP synthase in cristae surfaces

ATP is synthesized at ATP synthase sites in Fig. 5B on the cristae ends by the UVC mediated dehydrogenation reaction, ADP + Pi → ATP. The ATP sites < 8 nm shown as SMP in Fig. 4 do emit simple QED induced EUV, but not UVC. Similar to the 80 nm NP, the UVC is produced from body heat Q at temperature T conserved by creating EM standing waves between adjacent cristae having the UVC photon energy $E = hc/\lambda = 4.88$ eV, the UVC mediating each ATP site producing an ATP having $E_{ATP} = 30$ kJ / mol = 0.311 eV.

Electron microscopy [15] showed the concentration of ATP synthase sites on the cristae of 1 per nm^2 giving the number of ATP synthase sites on a mitochondrial of about 320,000. Hence, the mitochondria energy $U = 320{,}000 * E_{ATP} = 99{,}520$ eV = 1.59 x10^{-14} J. The power P produced is, P = 1 x10^{-15} W / μm^3 of volume. For 1 μm^3, P =10^{-15} W. The time τ to produce 320,000 ATP is, $\tau = 320{,}000 * E_{ATP}/P \sim 16$ s. Hence, the average mitochondrial synthesizes about 20,000 ATP per second. However, not all of the ATP is available to power other cells as the mitochondrial sacrifices some ATP to protect the DNA from which the UVC was created.

C. Evolution of UV induced ATP synthesis

The significant endogenous UVC produced by a single NP if taken as representative of the average mitochondria means the large number of ATP produced must exceed the number of damaged DNA including their repair, as otherwise survival of cellular life cannot continue. Indeed, the UVC that damages DNA also mediates ATP synthesis. But mitochondria release ATP to protect [16] mitochondria against UV damage. The protective ability of ATP allows the dehydration reaction to proceed during UV radiation. Perhaps, the simplicity of UV oxidation of food to glucose in allowing ATP synthesis to sustain life justifies the expense of sacrificing ATP to protect the mitochondria. Chemiosmosis does not require UV radiation. Dehydrogenation differs by requiring UVC in both ATP synthesis and DNA damage, the combination of which could not have naturally occurred unless the early Earth favored the evolution of life [2] to proceed in an UVC environment, i.e., evolution favored ATP synthesis by dehydrogenation instead of chemiosmosis.

In 1963, Sagan proposed [2] synthesis of ATP on the primitive Earth to be a consequence of the absorption of UVC because the evolution of the simplest living organisms from aggregations of molecular constituents is statistically unlikely. ATP production must therefore have evolved from molecules available in the primitive environment. In this regard, a dilute solution of adenine irradiated with UV light showed the nucleotides ADP and ATP were formed. Adenine having a large UVC absorption is also the most stable of such molecules under UVC irradiation.

Today, the surface of the Earth is absent UVC because of the absorption by the ozone layer. But in the Archean era about 4 billion years ago, the early Earth lacking oxygen and ozone was exposed [17] to significant UVC that most likely affected evolution of biological systems. Estimates of DNA inactivation suggest physical and biological methods likely evolved allowing DNA survival under the high UVC levels. Indeed, sulfur-33 isotopes showed [18] chemical interaction with UVC radiation that continued until the time ozone began to form in the Earth's atmosphere about 2.45 billion years ago. In the comparison between hydrolysis and dehydrogenation, the latter is favored in evolution because the high UVC levels on the early Earth is consistent with UVC mediation of ATP synthesis. Although UVC also damaged DNA damage, DNA repair systems compatible with UVC would naturally have evolved. Hydrolysis based chemiosmosis would not have been favored in the high UVC levels on the early Earth.

VI. CONCLUSIONS

Simple QED is based on the Planck law that denies constituent atoms in nanostructures the heat capacity to conserve heat by an increase in temperature. Instead, conservation proceeds by creating standing EM radiation inside the nanostructure. The EM confinement of the standing radiation is caused by the high surface-to-volume ratios inherent in nanostructures that constrains the applied heat to the nanostructure surface, the surface heat itself providing the brief EM confinement necessary to create the EM radiation. Once the surface heat is depleted in creating the standing radiation, the EM confinement vanishes, and the EM radiation is free to move into the surroundings to create ATP or damage DNA.

In mitochondria, UVC mediated dehydrogenation reactions are proposed to supersede hydrolysis by chemiosmosis. However, the UVC also damages DNA and therefore mitochondria survival requires UVC photon have a higher probability of creating ATP than damaging DNA. Moreover, the high UVC levels on the Earth from 4 to 2.5 billion years ago favored ATP synthesis by dehydrogenation suggesting Darwin's intelligent creator of the origin of the species may be nothing more than the random scrambling of DNA genomes by UVC radiation.

In future experimental verification of simple QED in mitochondria, difficulty in measuring the heat of ATP binding on the surface of cristae is avoided by using NPs in a thermal water bath at different temperatures to simulate the creation of UVC photons in nanostructure features of mitochondria. Experimentally, the findings are self-evident as the large number of UVC photons expected from 80 nm spherical aluminum NPs should be easily measured.

The large number of UVC photons predicted from NPs in a thermal bath suggest that the current vaccination practice of adding aluminum adjuvant NPs to stimulate the immune systems should be reviewed for brain damage. Regardless, 50 – 100 nm NPs should be prohibited in vaccinations because of UVC damage to DNA. Tests are recommended to determine if NPs < 50 nm producing EUV can stimulate the immune system. If so, only < 50 nm NPs should be used as stimulants in vaccinations to avoid UVC induced brain damage.

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