Nanoparticle Induced DNA Damage

Thomas Prevenslik

Discovery Bay, Hong Kong, China

***Abstract* — DNA damage induced by nanoparticles (NPs) is now considered to mimic that by conventional ionizing radiation, and therefore it is reasonable to hypothesize the NPs somehow produce electromagnetic (EM) radiations, at least at ultraviolet (UV) levels. In fact, ionizing radiation from NPs at UV levels is consistent with the theory of QED induced EM radiation. QED stands for quantum electrodynamics. By this theory, fine NPs (< 100 nm) absorb low frequency thermal kT energy in collisions with solution molecules only to be induced by QED to be frequency up-converted to the EM frequency of the NP, usually beyond the UV. But the quasi-bound EM confinement allows the UV to leak from the NP, thereby providing a significant antibacterial agent in food processing, reducing infections in burn treatment, sunscreen skin lotions, and treating cancer tumors. However, there is a darkside. Over the past decade, experiments have shown NPs to produce the reactive oxygen species (ROS) of hydroxyl radicals that cause apoptosis/cell death and single and double strand breaks in the DNA. What enables the NPs to function as an antibacterial agent while posing a health risk is the remarkable fact NPs provide a low level source of continuous UV radiation. DNA damage leads to the increased risk of producing cancer, the health risk of which suggests the regulation of NPs in commercial applications.**

Keywords — cancer, DNA, nanoparticles, QED

I. INTRODUCTION

DNA damage by <100 nm NPs is now [1] considered to mimic the same pathways as by conventional sources of ionizing radiation. The most reasonable hypothesis is the NPs are somehow producing their own ionizing radiation at least at UV levels, albeit at low intensity.

NPs producing low level ionizing radiation is consistent with the theory of QED induced EM radiation [2]. By this theory, NPs nearby the walls of a biological cell produce at least UV radiation upon absorbing kT energy of colliding extra cellular solution molecules. Even though the UV intensity is low, DNA damage by single strand (SS) and double strand (DS) breaks may occur directly by photolysis or indirectly by forming the hydroxyl radical.

Currently, the NP oxidative stress mechanism based on the surface area of < 100 nm NPs is thought to govern DNA damage. However, experimental data [1, 3-15] over the past few decades has placed this paradigm in question.

II. PURPOSE

The purpose of this paper is to show QED induced EM radiation in NPs produces EM radiation beyond UV levels that directly or indirectly cause SS and DS breaks in the DNA culminating with the conjecture that natural and man-made NPs are indeed the most likely source of ALL cancers.

III. BACKGROUND

DNA damage caused by ionizing radiation from NPs has been cited in numerous publications. Only limited background relative to the DNA damage hypothesis by NPs is presented.

A. EM Energies

EM energies necessary to directly damage the DNA require at least photolysis at UV levels. The DNA ionization potential [3] varies from 7.5 to 10 eV. Breaking SS and DS in dry DNA requires EM radiation [4] having energies above a threshold of 7 eV. The number of DS breaks then increases monotonically to about 12 eV and then remains constant.

The indirect ionizing radiation pathway relies on photolysis to form hydroxyl radicals in the extra and intracellular water [5] that causes SS and DS breaks by chemical reaction. The EM radiation need only exceed 5.2 eV to break the H-OH bond, and therefore the indirect pathway is more likely to cause DNA damage than by the direct path. The hydroxyl radical is a significant oxidative mechanism suggesting the NP oxidative stress paradigm in part finds basis in the hydroxyl radical.

B. NP Induced Oxidative Stress Paradigm and Problems

In the 1990's, evidence that α-quartz particles (Min-U-Sil) having a mean diameter of 5 microns were capable [6] of inducing oxidation damage of biological systems. However, it is likely that some <100 nm NPs were included with the Min-U-Sil particles. In silicosis, the induced hemolysis from ROS upon the interaction of silica particles with red blood cell membranes was attributed to the formation of hydrogen peroxide on the particle surface that upon reaction with metal ions by the Fenton reaction produced the hydroxyl radical. Indeed, hydrogen peroxide was detected [7] by ESR in aqueous suspensions of quartz particles. However, the source of hydrogen peroxide that produced the hydroxyl radical has never been conclusively identified.

In 2003, the NP oxidative stress paradigm as a measure of forming ROS was [8] correlated with the surface area of <100 nm NPs, although the mechanism by which the hydroxyl radicals and hydrogen peroxide form was not defined. Instead, the toxicology of air pollution was based on polycyclic aromatic hydrocarbons (PAH) particles as the most damaging to the DNA. However, there was difficulty with this paradigm because ESR comparisons [9] of the coarse $PM_{2.3\text{-}10}$ particulate produced a greater number of hydroxyl radicals than the fine $PM_{<2.5}$ particulate.

Similar problems were found [10] with the NP oxidative stress paradigm in 2006. Ambient and manufactured NPs were investigated with regard to the biological consequences of ROS production. Ambient particulate collected from the Los

Thomas Prevenslik is a retired American living in Hong Kong and Berlin.
Contact author: nanoqed@gmail.com.
URL Reference: www.nanoqed.org

Angeles basin having diameters about 1500 nm and NH2–PS spheres 1000 nm in diameter showed the clearest evidence of toxicity compared to 100 to 300 nm NPs. Also in 2006, pulmonary studies were conducted on rats using a wide range of α-quartz NPs [11] that showed about the same toxicity for 10-20 nm synthetic and 300-700 nm (Min-U-Sil) NPs. DNA damage was attributed to surface activity.

In 2008, DNA damage [12] by silver NPs widely used as antimicrobial agents was studied. Bare 25 nm silver NPs while were coated with polysaccharide to an overall diameter of 80 nm. More severe DNA damage comprising DS breaks and apoptosis/cell death was found with the larger coated NPs. Similarly, otherwise inert gold NPs were not only found [13] to generate free radicals, but also scavenge the NP)s. Absent NPs other than naturally present [14] in ex vivo human skin, the free radical production with VIS (400-700 nm) light and NIR (700-1600 nm) radiation is difficult to explain as neither VIS and NIR light are not ionizing radiations.

C. Modified NPInducedOxidativeStress Paradigm

Observations [9-12] suggest the NP oxidative stress paradigm that correlates DNA damage with the area of <100 nm NPs should be modified to account for the greater DNA damage from larger 300-1400 nm NPs. By QED induced EM radiation, the NPs < 100 are the source of ionizing radiation induced DNA damage.

But if so, how is the greater DNA damage from larger NPs reconciled?

In this paper, the NP induced oxidative stress paradigm is modified to consider only the <100 nm NPs that accompany the large 300-1400 nm particulate. The larger NPs should not be viewed as the correlation to DNA damage, but rather as means of converting the kT energy in collisions from surrounding molecules to NIR that is subsequently enhances the collision induced UV radiation from the <100 nm NPs

D. QED Induced Radiations

Ionizing radiation from NPs based on QED induced EM radiation [2] was proposed [15, 16] as an alternative to the heating mechanism thought to cause cancer necrosis in photodynamic therapy (PDT). Previously, gold NPs attached to cancer tumors were thought destroyed by high temperatures upon the absorption of NIR laser irradiation However, conservation of the absorbed laser photon does not proceed by temperature increase of the NP, but rather by the emission of EM radiation at its EM confinement frequency, typically beyond the UV. By this theory, the UV radiation causes cancer necrosis – not high temperature.

Similar to the necrosis of cancer cells with NPs irradiated with NIR lasers, conservation of absorbed EM energy in nanostructures proceeds by UV or higher EM radiation emission by QED induced radiations. The nanostructure need not be a NP, but may be a thin film under Joule heating, or the excitation of the higher quantum states of a molecule irradiated by multiphoton infrared photons, or nanocatalysts inducing chemical reactions from the kT energy of colliding molecules in the surroundings.

QED induced EM radiation is applicable anytime nanostructures absorb EM radiation from lasers, Joule heating, or the kT thermal energy in collisions from surrounding molecules – none of which may be conserved by an increase in temperature as illustrated for NPs in Fig. 1.

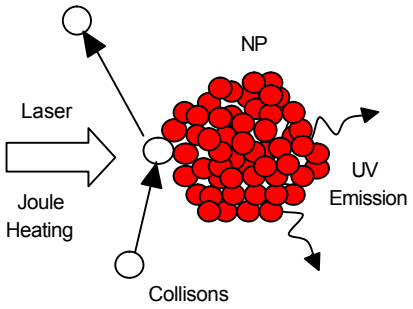

Figure 1. NP emitting QED induced EM radiation

In these and many other applications, QED induced EM radiation finds basis in simple physics – that photons of wavelength λ are created if EM energy is supplied to a quantum mechanical box having sides separated by λ/2. However, the QED induced photons only have significant EM energy if created in submicron structures. Conversely, macroscopic structures create far infrared (FIR) photons that by classical heat transfer are conserved by increase in the temperature of the structure.

IV. THEORY

The DNA in a biological cell may be damaged by NP in the extra or intra-cellular water. A NP that has entered a cell is depicted in Fig. 2. However, NPs in the extra cellular water may also damage the DNA by emitting UV radiation that penetrates the membrane. Regardless, the water molecules continuosly collide with and transfer their thermal kT energy to the NP. Since the water molecules are small compared to the NP, the collisions are inelastic and the transfer of kT energy to the NP is very efficient. Conservation of absorbed kT energy proceeds by the emission of EM radiation at the EM confinement frequency of the NP, usually beyond the UV that is sufficient to induce DNA damage

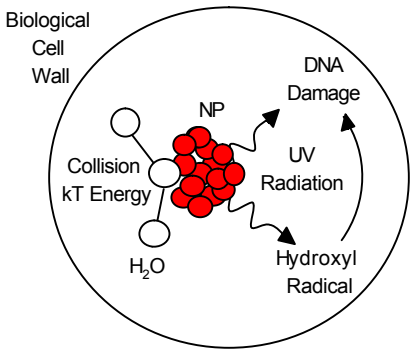

Figure 2 NP emitting UV Radiation inside Biological Cell

By photolysis, EM radiation beyond the UV may directly damage the DNA, or indirectly by forming hydroxyl radicals that chemically damage the DNA. The chemical path is more likely as only 5.2 eV is required [5] to form the hydroxyl radical compared to 7-10 eV necessary [4] for SS and DS breaks in dry DNA.

A. EM Confinement Frequencies

The EM resonant wavelength λ and Planck energy E_p in the NP upon the absorption [2] of the kT energy of the colliding molecules,

$$\lambda = 2n_r D \text{ and } E_P = \frac{hc}{2n_r D} \quad (1)$$

where, n_r is the refractive index and D is the NP diameter. The index $n_r > 1$ corrects for the retardation of the speed of light c in the solid NP.

B. QM Restrictions

The Einstein-Hopf relation for the harmonic oscillator [17] showing the dispersion of average Planck energy E_{avg} with wavelength λ at T ~ 300 K is given in Fig. 3.

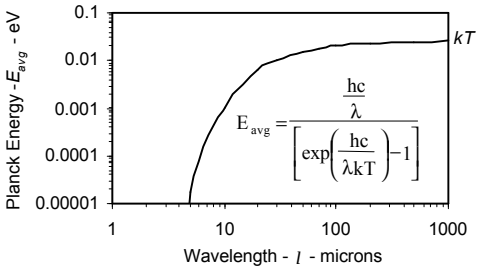

Figure 3 Harmonic Oscillator at T ~ 300K
In the inset, h and k are Planck and Boltzmann constants, and c is the speed of light in vacuum

NPs with D < 100 nm have EM wavelengths λ < 0.4 microns for an upper bound $n_r < 2$. Fig. 3 shows that for an atom confined in a NP, the average Planck energy << $1x10^{-5}$ eV. In contrast, a free atom absent EM confinement has full kT energy ~ 0.0258 eV. Hence, NPs under EM confinement at UV wavelengths λ < 0.050 microns have vanishing small kT energy << $1x10^{-5}$ eV.

C. Vanishing Specific Heat

Classical heat transfer conserves absorbed EM energy by an increase in temperature, but is not applicable to NPs because of QM restrictions on thermal kT energy. To show this, consider the specific heat C = ∂U/∂T from the Einstein-Hopf relation to give the dimensionless specific heat C*,

$$C^* = \frac{C}{3Nk} = \frac{\left(\frac{hc}{\lambda kT}\right)^2 \exp\left[\frac{hc}{\lambda kT}\right]}{\left[\exp\left(\frac{hc}{\lambda kT}\right) - 1\right]^2} \quad (2)$$

At 300 K, Fig. 4 shows C* vanishes for $\lambda = 2n_r D < 5$ microns. For n_r =1.2, the absorbed EM energy for D > 2 microns is conserved by a temperature increase while EM emission occurs for D < 2 microns.

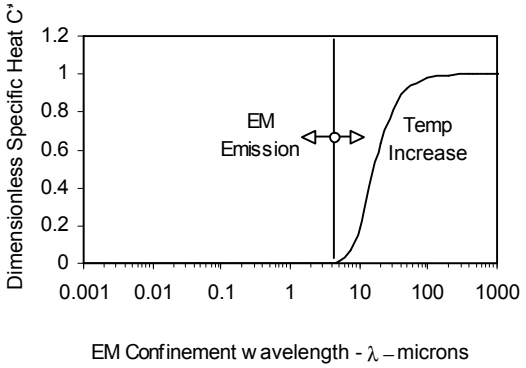

Figure 4 Dimensionless Specific Heat C* at T ~ 300 K

D. Collisonal Power and QED Induced Photons and Rate

The power Q_C transferred [18] in collisions of intracellular water molecules to the NPs,

$$Q_C = \frac{\pi}{2\sqrt{3}} pPD^2 \sqrt{\frac{kT}{m}} \quad (3)$$

where, p is the probability of full kT energy transfer, and P is the ambient pressure. For inelastic collisions, p is unity. The mass m of the water molecules is, MW/N_{avag} where MW = 18 is molecular weight and N_{avag} is Avagadro's number.

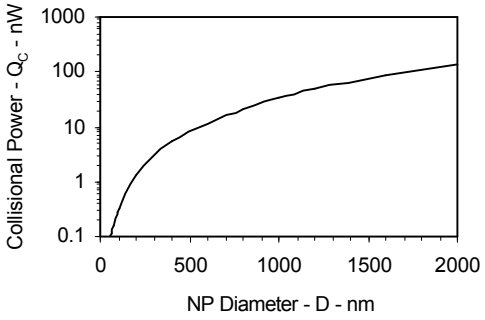

Figure 5 Collisional Power Q_C v. NP Diameter D

Absent an increase in NP temperature, the power Q_C is conserved by the emission of EM radiation,

$$E_P \frac{dN_P}{dt} = Q_C \quad (4)$$

where, dN_P/dt is the rate of QED induced photons produced in the NP having Planck energy E_P. For silver NPs having n = 1.35, the QED induced photon energy and rate is shown in Fig. 6.

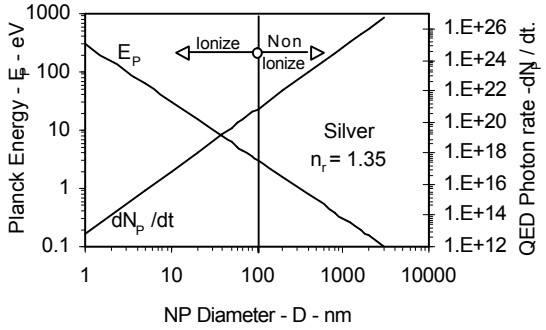

Figure 6 Planck Energy E_P and Photon Rate for Silver NPs

Silver NPs <100 nm emit ionizing radiation beyond the UV. DNA SS and DS breaks at 5.2 and 7 eV (238 and 123 nm) occur for d = 88 and 65 nm NPs, respectively. But NPs > 100 nm, emit non-ionizing radiation in the VIS and NIR.

V. ANALYSIS

In QED Induced EM radiation, the NP oxidative stress paradigm for fine <100 nm NPs need not be invalidated by the greater DNA damage found in coarse 300-1500 nm NPs. To show this, consider an arrangement of fine NPs of diameter d in relation to coarse NPs of diameter D shown in Fig. 7.

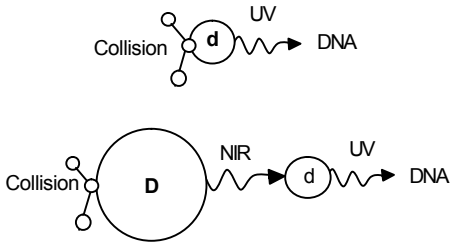

Figure 7 NIR enhanced UV Radiations

Collisions induce the fine NPs to emit UV and the coarse NPs to emit NIR power,

$$Q_{UV} = \frac{\pi}{2\sqrt{3}} pPd^2 \sqrt{\frac{kT}{m}} \text{ and } Q_{NIR} = \frac{\pi}{2\sqrt{3}} pPD^2 \sqrt{\frac{kT}{m}} \quad (5)$$

Mie theory [19] gives the efficiency Q_{abs} of the fine NPs absorbing the NIR radiation from the coarse NPs,

$$Q_{abs} = F\left(\frac{d}{\lambda_{NIR}}\right) \quad (6)$$

where, $$F = \frac{24\pi ab}{\left(a^2 + b^2 + 2\right)^2 + 4a^2b^2}$$

The NIR wavelength $\lambda_{NIR} = 2n_rD$, where n_r is the refractive index of the coarse NPs. The parameters a and b are the real and complex refractive index of the fine NPs.

The collisional power absorbed by the DNA is,

$$Q_{UV-NIR} = Q_{abs} Q_{NIR} + Q_{UV} \quad (7)$$

The ratio R of UV enhancement,

$$R = \frac{Q_{UV-NIR}}{Q_{UV}} = \left(\frac{D}{d}\right)\frac{F}{2n_r} + 1 \quad (8)$$

For fine silver NPs, the parameters a = 1.35 and b = 4 and silica coarse NPs having n_r = 1.45, the enhancement ratio R is shown in Fig. 8.

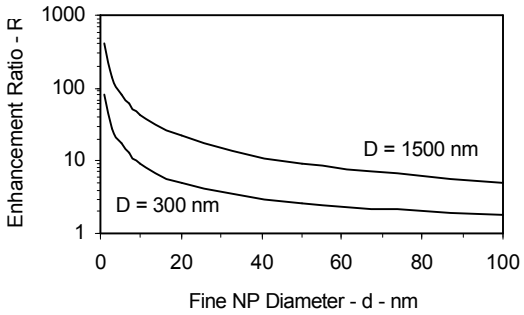

Figure 8 Enhancement Ratio R of Fine NPs by Coarse NPs

DNA damage by silver NP induced hydroxyl radical SS breaks at 5.2 eV and DS breaks at 7 eV occurs at d = 88 and 65 nm is shown enhanced for coarse 300 and 1500 nm NPs by ratios R of about 2 and more than 5, respectively.

VI. DISCUSSION

A. Modified NPInduced Oxidative Stress Mechansims

The NP oxidative stress paradigm that claims the ROS correlate with the area of fine <100 nm NPs is not modified because of the greater DNA damage found with the coarse 300-1500 nm NPs. The only modification necessary is that the coarse NPs should not be considered as damaging, but rather acting to enhance the DNA damage caused by the fine <100 nm NPs.

B. Consequences of QED Induced EM Radiations

1) Similarity of NP induced UV to Ionizing Radiations. Air pollution [8,9] studies give direct evidence of DNA damage by PM_{10} having < 50% by mass of combustion derived nanoparticles (CDNPs) in < 100 nm NPs. The CDNPs are carbon centered NPs from automobile exhausts, but NP induced DNA damage mechanisms are currently not known.

QED induced EM radiation claims <100 nm NPs produce at least UV radiation from which molecular mechanisms for DNA damage may be formulated. Indeed, NP induced respiratory DNA damage mimics [1] that by ionizing radiation, albeit at lower UV levels. This means DNA damage mechanisms under ionizing radiation are applicable to NP induced DNA damage.

2) NP Induced Oxidative Stress Paradigm. Recent pulmonary studies on rats [11] present evidence to contradict the NP induced oxidative stress paradigm that states DNA damage is caused by toxicity that correlates with the surface area of <100 nm NPs. Indeed, the toxicity of 500 nm mined α-quartz (Min-U-Sil) particles was found equivalent to that from synthetic 12 nm quartz NPs. The hemolytic potential of α-quartz in red blood cells was attributed to the surface activity [6] caused by defects, jagged edges, or the vague ease in producing ROS. Silica is known to generate hydroxyl ions from hydrogen peroxide, and indeed both have been detected aqueous suspensions α-quartz. However, the specific reactions leading to the formations of hydrogen peroxide from silica have never been identified.

In contrast, QED induced EM radiation from <100 nm NPs unequivocally provides the UV to directly form the hydroxyl radicals and hydrogen peroxide. In fact, the surface activity of α-quartz thought to produce the hemolytic potential in RBCs is most likely caused by the absorptions of QED induced UV radiations in the solution adjacent the NP surfaces. There is no need to invoke the unquantifiable notion of surface activity to explain ROS for α-quartz.

3) Anti-Microbial Silver Nanoparticles. Silver NPs having the greatest degree of commercialization are of interest in DNA damage because of the potential treatment of inflammations in the blood. Indeed, the antimicrobial activity in controlling infections [20] and limiting bacterial growth [21] in the food industry are only a few of the many applications of silver NPs.

However, silver NPs also damage [12] the DNA. The ROS including hydroxyl radicals and hydrogen peroxide are thought produced by surface chemistry. But surface chemistry cannot be the mechanism for bactericidal action of silver NPs because polysaccharide coated silver NPs produced greater DNA damage than for bare silver NPs. EM energy is required to produce ROS that cannot be produced by surface chemistry. But QED induced EM radiation is produced in NPs beyond the UV. Indeed, the 3-fold increase in the diameter of the coated to bare NPs corresponding to EM confinement wavelengths from 68 to 200 nm suggest the NPs exposed the DNA to UV beyond 6.2 eV. To avoid DNA damage, NPs larger than 100 nm are required, but this would negate the bactericidal action of the silver NPs.

4) Sun Screen. The interaction of sunlight with the human skin has led to a fragile equilibrium between the EM radiation necessary for life and UV levels that damage the DNA. Prompted by the nearly epidemic increase in skin cancers over the past few decades, the European Commission has lowered the acceptable ratio UVA/UVB. Here UVA (320-400 nm) and UVB (280-320 nm). However, only about 6 % of sunlight is in the UV with 52% in the VIS and 42% in the NIR suggesting the VIS and NIR may also be producing DNA damage.

Indeed, the ROS in the form of free radicals were found [14] in human skin under both UV and VIS/NIR radiation. UVB is ionizing radiation that is expected to produce free radicals, but the VIS/NIR is not. The free radicals measured were thought caused by heat from the VIS/NIR increasing the skin temperature. But there is no known mechanism by which simply raising the temperature produces free radicals.

QED induced EM radiation at UV levels produces ionizing radiation provided <100 nm NPs are present on the skin surface. Adherent subcutis and fascia [14] were removed, but the concentration of the remaining natural NPs was not given to assess the importance of QED induced EM radiation in producing free radicals directly from VIS/NIR radiation. Nevertheless, it is highly likely NPs were in fact present to explain the free radicals observed.

Sunscreens use white colored zinc oxide particles to deflect [22] damaging UV radiation, but the zinc oxide may be made transparent and more absorbent by shrinking the particles down to <100 nm NPs. By QED induced EM radiation, the zinc oxide NPs absorb fractions of the UV/VIS/NIR radiation only to produce higher energy UV radiation that damages the DNA. To avoid DNA damage, the <100 nm NPs should be replaced by NPs > 100 nm that would convert the UV content in sunlight to VIS/NIR levels. The claim [22] that NPs are absorbed in the skin and therefore cannot cause DNA damage to the brain or liver does not consider the capability of UV radiation to penetrate the skin an induce DNA damage in the RBC. With the wide use of NPs in sunscreens, it is no wonder that the increase in skin cancer has reached epidemic levels over the past 20 years.

5) Gold Nanoparticles The interaction between gold NPs and aniline give [13] the formation of free radicals in contrast to the hydroxyl radicals [12] formed between silver NPs in intracellular water solutions. Both gold and silver NPs are induced by QED to produce UV radiation and free radicals that depend on the solution. Surface activity is almost inconsequential to the ionizing UV radiation.

6) Cancer Therapy. In PDT, photosensitizers in the form of NPs that preferentially attach to cancer cells and activated by NIR radiation are claimed [23] to produce singlet oxygen, thereby destroying the cells by chemical reaction. But cancer cells are destroyed without photosensitizers, thereby begging the question of what actually induced cancer necrosis in PDT.

Prior to photosensitizers, high temperature was thought to induce cancer necrosis in PDT. But NPs lack the specific heat [15, 16] to allow a temperature increase to conserve the absorbed NIR radiation, and therefore QED induces the NP to emit EM radiation beyond the UV that causes cell necrosis, thereby obviating the need for photosensitizers in PDT to activate the oxygen singlet state.

Whether DNA is not damaged by a certain frequency range of ionizing radiation that is damaging to a specific cancer is an unlikely conjecture. But if research shows otherwise, the selection of a NP size tuned solely to the frequency causing necrosis of the cancer may be possible. Only then may NPs be justified in cancer therapy.

VII. Conclusions

Conclusions based on NP induced DNA damage and cancer risks rely on the theory of QED induced EM radiation to allow NPs to produce UV radiation that mimics that caused by conventional sources of ionizing radiation.

- NPs < 100 nm produce EM radiations beyond the UV that damages DNA and increases the risk of cancer.
- NIR lasers used to activate NPs of photosensitizers are not necessary to produce UV radiation. The EM energy required to produce the UV radiation is the thermal kT energy of surrounding extra and intracellular water molecules that collide with the NPs and upon absorption is induced by QED to be frequency up-converted to UV levels.
- QED only induces EM radiation beyond the UV at NP diameters <100 nm NPs.
- QED induces the large 300-1500 NPs to produce VIS/NIR radiation that enhance the UV emission from adjacent <100 nm NPs.
- The NP induced oxidative stress paradigm that DNA damage is caused by ROS produced proportional to the area of <100 nm NPs needs to be modified to exclude the larger 300-1500 nm NPs.
- Surface activity based on the area of <100 nm NPs. Is non-quantifiable.
- Sunscreens having NP < 100 nm NPs should be banned in favor of NPs > 100 nm that would absorb UV radiation that then is frequency down-converted to DNA non-damaging VIS and NIR radiation.
- The widespread use of silver NPs in limiting bacteria in food processing and anti-microbial action should cease immediately for risk of developing cancers.
- The DNA damage induced by NPs is a cancer risk if not properly repaired. Given that NPs produce ionizing radiation beyond UV levels from the QED induced kT energy of surrounding solution molecules, and that natural and man-made NPs are ubiquitous, the conjecture is made that NPs are the most likely cause of cancers in ALL mammals. Where possible, the US and European Union therefore should ban the use of <100 nm NPs in all man-made products.
- The sensitivity of DNA to ionizing radiation should be determined to see if cancers can be selectively targeted by NPs without causing DNA damage.

References

[1] R.M. Mroz, et al., "Nanoparticle-driven DNA damage mimics irradiation-related carcinogenesis pathways," Euro. Respir. J, vol. 31, pp. 241–251, 2008.

[2] T. Prevenslik, "QED induced EM radiation," See www.nanoqed.org, 2004-2009

[3] B. Boudaiffa, P. Clotier, D. Hunting, M.A. Huels, and L. Sanche, "Resonant Formation of DNA Strand Breaks by Low-Energy (3-20 eV) Electrons," Science, vol. 287, pp. 1658-1660, 2000.

[4] K. Hieda, "DNA damage induced by vacuum and soft X-ray photons from Synchrotron radiation," J. Radiat. Biol., vol. 66, pp. 561, 1994.

[5] M. Kuwarara, A. Minegishi, K. Takakura, K. Hieda, and T. Ito, "Photolysis of water VUV radiation and reactions with DNA and related compounds in aqueous systems," In Photobiology, Edited by E. Riglis, New York, Plenum, pp. 355-363., 1991.

[6] B.L. Razzaboni and P. Bolsaitis, "Evidence of an Oxidative Mechanism for the Hemolytic Activity of Silica Particles," Environ. Health Persp, vol. 87, pp.337-341, 1990.

[7] X. Shi, N.S. Dalal, and V. Vallyathan, "ESR evidence for the hydroxyl radical formation in aqueous suspension for quartz particle and its possible significance to lipid peroxidation in silicosis." J. Toxicol Environ Health, vol. 25, pp. 237-245, 1988.

[8] K. Donaldson, "The biological effect of coarse and fine particulate matter," Occup Environ Med, vol. 60, pp. 313-314, 2003.

[9] J.T. Shi, et al., "Temporal variation of hydroxyl radical generation and 8-hydroxyl radical generation and 8-hydroxy-2'-deoxyguanosine formation by coarse and fine particulate matter," Occup. Environ Med, vol. 60, pp. 315-321, 2003.

[10] T. Xia, et al., "Comparison of the Abilities of Ambient and Manufactured Nanoparticles to Induce Cellular Toxicity According to the Oxidative Stress Paradigm," Nano Lett., vol. 6, pp. 1794-1807, 2008.

[11] D.B. Warheit, T.R. Webb, V.L. Colvin, R.L. Reed, and C.M. Sayes, "Pulmonary Bioassay Studies with Nanoscale and Fine-Quartz Particle in Rats: Toxicitry is not Dependent upon Particle Size but on Surface Characteristics," Toxicological Sciences, vol. 95, pp. 270-280, 2007.

[12] M. Ahmed, et al., "DNA damage .response to different surface chemistry of silver nanoparticles in mammalian cells," Toxico Appl. Pharmacol, vol. 233, pp. 404-410, 2008.

[13] P. Ionita, F. Spafiu, and C. Ghica, "Dual behavior of gold nanoparticles, as generators and scavengers for free radicals," J. Mater. Sci, vol. 43, pp. 6571-6574, 2008.

[14] L. Zastrow, et al., "The Missing Link – Light-induced (280-1600nm) Free Radical Formation in Human Skin," Skin Pharmacology and Physiology," vol. 22, pp. 31-44, 2008.

[15] T. V. Prevenslik, "Nanoparticles in Cancer Therapy," presented at GEM Conference on Cancer in conjunction with Inter. Conf. Mat. App. Tech. 2007, Singapore, July 1-4, 2007.

[16] T. Prevenslik, "Cancer Therapy by QED induced EM radiation," See www.nanoqed.org , 2007.

[17] R.W. Christy and A. Pytte. The Structure of Matter: Introduction to Modern Physics. New York: Benjamin, 1965.

[18] K.K. Das, Y.V. Rostovtsev, K. Lehmann, M.O. Scully, Optics Communications, vol. 246, pp. 551, 2005.

[19] C. F. Bohren and D. R. Huffman, Absorption and Scattering of Light by Small Particles, J. Wiley & Sons, 1983.

[20] J.S. Kim, E. Kuk, J.H. Kim, S.J. Park, and H.J. Lee, "Antimicrobial effect of silver nanoparticles," Nanomedicine, vol. 3, pp. 95-101, 2007.

[21] C.F. Chau, S.H. Wu, and G.C. Yen, "The development of regulations for food nanotechnology," Trends Food Sci. Technol., vol. 16, pp.269-280, 2007.

[22] D. Biello, "Do Nanoparticles and Sunscreen Mix?," 2007 www.sciam.com/article.cfm?id=do-nanoparticles-and-sunscreen-mix

[23] X. Huang, I.H. El-Sayed, W. Qian,, and M.A. El-Sayed, "Cancer Cell Imaging and Photothermal Therapy in the Near-Infrared Region by Using Gold Nanorods," J. Am. Chem. Soc., pp. 2115-20., 2006.