Correlating ^{13}C Isotope in Oligomeric Proanthocyanidins with Their Anticancer Properties

Reginald B. Little

Stillman College

Tuscaloosa, Alabama 35401-2601

Abstract

Upon considering the anticancer effects of larger oligomeric proanthocyanidins and observing various papers reporting the high resolution mass spectroscopy of the oligomeric proanthocyanidins, it is determined that the unusual ^{13}C enrichment in some plant oligomeric proanthocyanidins may be responsible for the anticancer activities of these food products. Such correlation of the ^{13}C in the oligomeric proanthocyanidins also correlate with their scavenging of free-radicals, anti-virial and anti-bacterial properties. Such ^{13}C interactions from oligomeric proanthocyanidins with nucleic acids and proteins involved in replications, transcriptions and translations in cancer cells for interacting and chemically altering with anabolism and cell division of the cancer cells are consistent with the author's mechanism for normal cell to cancer cell transformations via possible replacements of primordial ^{1}H, ^{12}C, ^{14}N, ^{16}O, and ^{24}Mg isotopes by nonprimordial ^{2}D, ^{13}C, ^{15}N, and ^{17}O and ^{25}Mg isotopes in the proteins and nucleic acids. Such is also consistent with the proposed treatment for cancer by the author by use of foods containing proteins, nucleic acids, carbohydrates and/or drug molecules enriched with the nonprimordial isotopes of ^{2}D, ^{13}C, ^{15}N, and ^{17}O and ^{25}Mg.

Introduction

Cancer and Metabolism

Cancer is abnormal cell reproduction exhibiting unusual metabolic processes. Cancer occurs as cells alter various normal catabolic and anabolic metabolisms. Warburg Effect involves accelerated glycolysis and suppressed Kreb cycle (catabolism). Glycolysis is catabolic process of enzymatic conversion of glucose to pyruvate. The cellular transformations to cancer lead to accelerated glycolysis. Kreb cycle is catabolic enzymatic conversion of pyruvate to carbon dioxide. The cellular transformation to cancer lead to suppression of Kreb cycle. The anabolism of genetic code is also altered during cancer formation as DNA replications and RNA transcriptions are altered (chaotically and anabolically). Such anabolic chaos with altered consequent protein translations leads to cancer cell genesis and multiplying genetically altered cells rapidly. In this theory, the anabolic alterations of genes cause altered protein translations for producing proteins of glycolysis that accelerate glycolysis while producing proteins, associated with Kreb that suppress the Kreb cycle. A big mystery of cancer is the nature and mechanism of the DNA mutation, RNA mutation and altered protein translation.

Isotopic DNA, RNA and Protein Alterations for Mechanism

In this work, the theory [1-3] of stable isotopic replacements and substitutions of primordial, stable ^{1}H, ^{12}C, ^{14}N, ^{16}O, ^{24}Mg and ^{32}S by nonprimordial, stable ^{2}D, ^{13}C, ^{15}N, ^{17}O, ^{25}Mg, and ^{33}S is further developed. This work focuses more on DNA, nucleotides and telomeres. In normal cells, the ends of DNA have unbounded, telomeric regions, which are shortened to terminate replication of genes but in cancer the telomeres do not shorten and induce apoptosis, but the telomeres mutate and involve telomerase with acceleration of replications [4]. Telomerase is a protein that is associated with elongation of telomeres. It is unknown why shorten telomeres in cancer cells continue to replicate by telomerases and accelerate replications and transcriptions of DNA and RNA. In this work, the epigenetic stable-isotopic alterations by nonprimordial isotopes (^{2}D, ^{13}C, ^{15}N, ^{17}O, ^{25}Mg and ^{33}S) of DNA, RNA and consequent proteins during normal cells to cancer cells transformations are proposed for fundamental chemistry of cancer origins and habitats [1-3] and possibly explain why the shorten telomere in cancer continue to replicate rather than terminate cell life as the shorten telomeres in normal cells.

This theory [1-3] determines that isotopic replacements in normal cells with epigenetic modifications are not prevented from the shortening of the telomeres for causing apoptosis as the nonprimordial isotopes interferes with signaling to apoptosis by the nonprimordials binding of the telomere and consequent continued replication of the DNA with more and more replications such that the DNA becomes too enriched in nonprimordial isotopes (^{2}D, ^{13}C, ^{15}N, ^{17}O, ^{25}Mg and ^{33}S) of different nuclear magnetic moments (NMM) [1-3] for normal cellular functioning. But with aging of the host (unusual diet and/or external magnetism), this theory [1-3] proposes more and more biomolecules enrich in the nonprimordial isotopes (^{2}D, ^{13}C, ^{15}N, ^{17}O, ^{25}Mg and ^{33}S) relative to the primordial isotopes (^{1}H, ^{12}C, ^{14}N, ^{16}O, ^{24}Mg and ^{32}S) for greater probability of simultaneous, multiple nonprimordial clumpings in both proteins and nucleic acids such that the simultaneous nonprimordials in the proteins and the DNA and RNA prevent the normal telomeric (and other gene expressions) induced cell apoptosis by primordial isotopic interactions with the proteins; the nonprimordial isotopes interact between the telomere and telomerase to prevent apoptosis for causing continued cancerous DNA, RNA, and protein reproductions and malfunctions of the normal cells to transform them to carcinomic cells by the prior theory [1-3]. The prior theory [1-3] proposes that the clumpings of nonprimordial isotopes in the telomere

change the binding of the base pairs in the genes, so that the shorter telomere (and indeed for other genes and their expressions) does not express apoptosis as the telomere is bound more tightly by the nonprimordial isotopes and the telomeric gene is bound more strongly to binding proteins for telomerase expression so that the stronger bound nonprimordial, isotopic, shorter telomere continues to allow the DNA to replicate and the resulting nonprimordial DNA to replicate further transcribe nonprimordial RNA and the resulting nonprimordial RNA continues to produce nonprimordial proteins. In the DNA and RNA the accumulation of nonprimordials by ^{2}D, $^{13}C^{1}H_3$, $^{15}N^{1}H_2$ and $^{17}O^{1}H$ (and $^{13}C^{2}D^{1}H_2$, $^{15}N^{2}D_2{}^{1}H$, $^{17}O^{2}D$) functional replacements on nucleotides of guanosine (G), adenosine (A), cytidine (C), uridine (U) and thymidine (T) rather than primordial ^{1}H, $^{12}C^{1}H_3$, $^{14}N^{1}H_2$, $^{16}O^{1}H$ replacements cause altered stronger bonding of the AT and GC in nonprimordial DNA and stronger altered bonding of AU and GC in nonprimordial RNA. By the author's model [1-3], the nonprimordial isotopes in the ^{2}D, $^{13}C^{1}H_3$, $^{15}N^{1}H_2$ and $^{17}O^{1}H$ (and $^{13}C^{2}D^{1}H_2$, $^{15}N^{2}D_2{}^{1}H$, $^{17}O^{2}D$) on guanosine, adenosine, cytidine, uridine and thymidine cause a magnetic bonding in addition to the hydrogen bonding to reduce and hinder the separation of the DNA base pairs for causing normal cells to transform to cancer cells. But by the prior theory [1-3], such can cause with greater nonprimordial uptake by the cancer DNA so the over nonprimordial enriched cancer DNA becomes inseparable with killing of the cancer cells by over enriching the nucleic acids and proteins in the cancer.

Theory for Mechanism of Cancer and Cure

Atomic and Molecular Dynamics for Altered Bochemistry

The altered enzymatics of proteins and nucleic acids as by this prior theory [1-3] of cancer are based upon the different nuclear magnetic moments (NMMs) and masses of nonprimordial isotopes (^{2}D, ^{13}C, ^{15}N, ^{17}O, ^{25}Mg and ^{33}S) relative to primordial isotopes (^{1}H, ^{12}C, ^{14}N, ^{16}O, ^{24}Mg and ^{32}S) as well as their tiny relative mass differences. Hydrogen has 2 important stable isotopes with different NMMs, spins, masses and relative abundances: ^{1}H {99.988%, 1 ½ (I) spin, 2.79(μ/μ_N) NMM} and ^{2}D {0.0115%, 0 (I) spin, 0.86 (μ/μ_N) NMM}. Carbon has 2 important stable isotopes with different NMMs, relative abundances, masses and spins: ^{12}C {98.9%, O (I) spin, 0 (μ/μ_N) NMM} and ^{13}C {1.1%, ½ (I) spin, 0.70 (μ/μ_N) NMM}. Nitrogen has 2 important stable isotopes with different NMMs, relative abundances, masses and spins: ^{14}N {99.6%, 1 (I) spin, 0.40 (μ/μ_N) NMM} and ^{15}N {0.4%, ½ (I) spin, -0.28 (μ/μ_N) NMM}. Oxygen has 3 important isotopes with different NMMs, spins, masses and relative abundances: ^{16}O {99.8%, 0 (I) spin, 0 (μ/μ_N) NMM}, ^{17}O {0.03%, 5/2 (I) spin, -1.89 (μ/μ_N) NMM, ^{18}O {0.205%, 0 (I) spin, 0 (μ/μ_N) NMM}. Magnesium has 3 important isotopes with different NMMs, spins, masses and relative abundances: ^{24}Mg {79.0%, 0 (I) spin, 0 (μ/μ_N) NMM} ,^{25}Mg {10.0%, 3/2 (I) spin, -0.86 (μ/μ_N) NMM}, ^{26}Mg {11.0%, 0 (I) spin, 0 (μ/μ_N) NMM}. Phosphorus has 1 important isotope: ^{31}P {100%, ½ (I) spin, 1.13 (μ/μ_N) NMM}. Sulfur has 3 important isotopes with different NMMs, spins, masses and relative abundances: ^{32}S {94.9%, 0(I) spin, 0 (μ/μ_N) NMM}, ^{33}S {0.8%, 3/2 (I) spin, 0.64 (μ/μ_N) NMM}, ^{34}S {4.3%, 0 (I) spin, 0 (μ/μ_N) NMM}.

Changes in Isotopic Abundances

This theory [1-3] proposes that the relative abundances of the unusual, uncommon nonprimordial isotopes have changed in the environment and food supplies of plants, animals and humans such that humans have increased levels of the nonprimordial stable isotopes (^{2}D, ^{13}C, ^{15}N, ^{17}O, ^{25}Mg and ^{33}S) in their cells during the last 150 years for increased prevalence of

cancer. The technologies of the industrial revolution, nuclear reaction uses and industry, agricultural changes, automobile technology and radio-technology are proposed by this theory [1-3] to increase nonprimordial isotopes and even redistribute isotopes into key chemical bonds in biomolecules. By the author's theory [1-3] for instance, radiowaves are able by broad band excitations to stimulate the continua states by the author's theory [1-3] to redistribute nonprimordial isotopes into specific chemical bonds even in normal relative abundances relative to distributions in the absence of radiowaves. Thereby with increase enrichment, the radiowaves compound the clumping of non-primordial isotopes into specific chemical bonds in proteins, RNA and DNA.

Changes in Biomolecular Chemical Dynamics

These non-primordial isotopes reversibly, fractionally fiss and fuse to momentarily transmute to different quantum fields about the nuclei in atoms and molecules relative to the reversible, fractional fissing and fusing of primordial isotopes. Moreover, on the basis of this theory [1-3], the author has determined that the fractional, reversible fissing and fusing of the nonprimordial isotopes are more sensitive than nuclei of zero NMMs to tiny intensity surrounding fields of thermal space as by Little's Rules 1, 2 and 3. Such reversible, fractional fissing and fusing of the stable isotopes by the author's theory [1-3] alters the enzymatic dynamics along the reaction coordinates of proteins, nucleic acids, lipids, and carbohydrates biochemical dynamics. The fractional, reversible fissing and fusing of nuclear (NMM) alter surrounding atomic orbitals and such altered atomic orbital alter molecular orbitals and alter chemical dynamics, catalysis and enzymatics by the Little Effect: "spins alter orbitals during chemical reactions and orbitals altering spins". The Little Effect not only involves e^- spins altering orbitals but nuclear spins also alter orbitals for Little Effect as manifested by these nonzero NMMs of nonprimordials relative to more null NMMs of primordials. For instance, the fractional, reversible fissing and fusing of the nonprimordial isotopes in enzymes can alter the stereochemistry of the substrate as the enzyme catalyzes the chemical transformation of the substrate. For instance, ^{14}N and ^{15}N nuclei motions have different chiralities as ^{14}N has positive NMM and ^{15}N has negative NMM; so changing ^{14}N to ^{15}N by this prior theory (1-3) would cause the fractional fissed field of ^{15}N (relative to native ^{14}N in the enzyme) to alter the chirality of wavefunctions from the enzymatic catalyzing transition state of the substrate relative to such fissed fields from primordial ^{14}N. As the biomolecules have specific stereochemistry and manifest chiral environment in healthy organisms, the altered chirality can be a basis of disease as caused by ^{2}D, ^{13}C, ^{15}N, ^{17}O and ^{25}Mg. These alterations by the author's theory [1-3] transform normal cells to cancer cells. Such altered chemical dynamics by isotopic replacements in DNA, RNA and proteins are manifested by the accelerations of cellular reproduction, replication, transcription and protein translation with consequent acceleration of the glycolysis process and the suppression of the Kreb cycle. On the basis of the author's theory [1-3] the surrounding radiowaves accelerate such faster glycolysis and slower Kreb cycle.

Hypothesis

In this paper, it is hypothesized that during replications and transcriptions, the primordial isotopes code active genes, but nonprimordial isotopes accumulate in inactive regions of genes. It is further hypothesized that the shorten telomeres occur in normal cells due to the accumulations of primordial isotopes in the growing telomeres and telomerases; so the primordial telomerases cannot bind as well with the shorter primordial telomeres to prevent their

opening and unraveling of the telomere at ends having primordial isotopes; so in normal cells the shortened telomeres unravel at the end by the primordial isotopes to induce apoptosis. It is also hypothesized that as nonprimordial isotopes accumulate in normal cells, and DNA, RNA and proteins (like telomerase) through processes of deuterations, methylations, aminations, hydroxylations and carboxylations (involving ^{2}D, ^{13}C, ^{15}N, ^{17}O, ^{25}Mg and ^{33}S) then the interactions between the telomerases and the DNA change (becomes stronger due to magnetics of fractional, reversible fissing and fusing of the nonprimordial isotopes) so that the telomeres of the DNA open and close like regions in normal replicating and transcribing nucleic acids; so the nonprimordial telomeres themselves continue to replicate to elongate due to the stronger binding of the nonprimordial telomerases to the nonprimordial telomeres and this causes cancer. Thereby cancer develops by random isotopic editing of DNA such that the nonprimordial telomerases bind the nonprimordial telomeres via nonprimordial ··· nonprimordial interactions; so the telomeres continue to elongate and the DNA continues to replicate. The nonprimordial telomeres thereby fail to shorten and induce apoptosis before the cell reproduces multiple times to transfer epigenetic mutations in DNA, RNA and proteins for cancer habitat. Thereby the theory [1-3], it is hypothesized that the normal cells have shortened telomeres that stop replicating as they enrich with primordial isotopes: ^{1}H, ^{12}C, ^{14}N, ^{16}O, and ^{24}Mg. But it is further hypothesized that the cancer cells have elongated telomeres with nonprimordial isotopes of ^{2}D, ^{13}C, ^{15}N, and ^{17}O that accelerate rather than stop replication due to stronger binding of the telomere to proteins with nonprimordial isotopes: ^{2}D, ^{13}C, ^{15}N, and ^{17}O. In prior paper [5], the mass spectra of larger pieces of DNA comparable to telomere codon of cancer, white blood cells and red blood cells were compared. In this work, this hypothesis is tested by mass analysis of smaller pieces corresponding to individual nucleotides.

Method

In order to test some aspects of this hypothesis normal red and white cells and Leukemia cancer cells were obtained studied in vitro. The DNA from the normal and cancer blood cells were harvested after growth of cancer cells and normal cells in vitro. The DNA was mass analyzed by MALDI mass spectrometry. The mass spectra of the normal and cancer cells were analyzed and compared for isotopic differences. A comparisons of the oligonucleotides of DNA and the oligomeric proanthocyanidins from various fruit and vegetable food sources were done along with the corresponding mass spectra. Similar chemical structures of the proanthocyanidins and DNA oligonucleotides [7] were observed and the anti-cancer effects [8] of proanthocyanidins were determined based on exchange of nonprimordial isotopes between the proanthocyanidins and the oligonucleotides.

Results

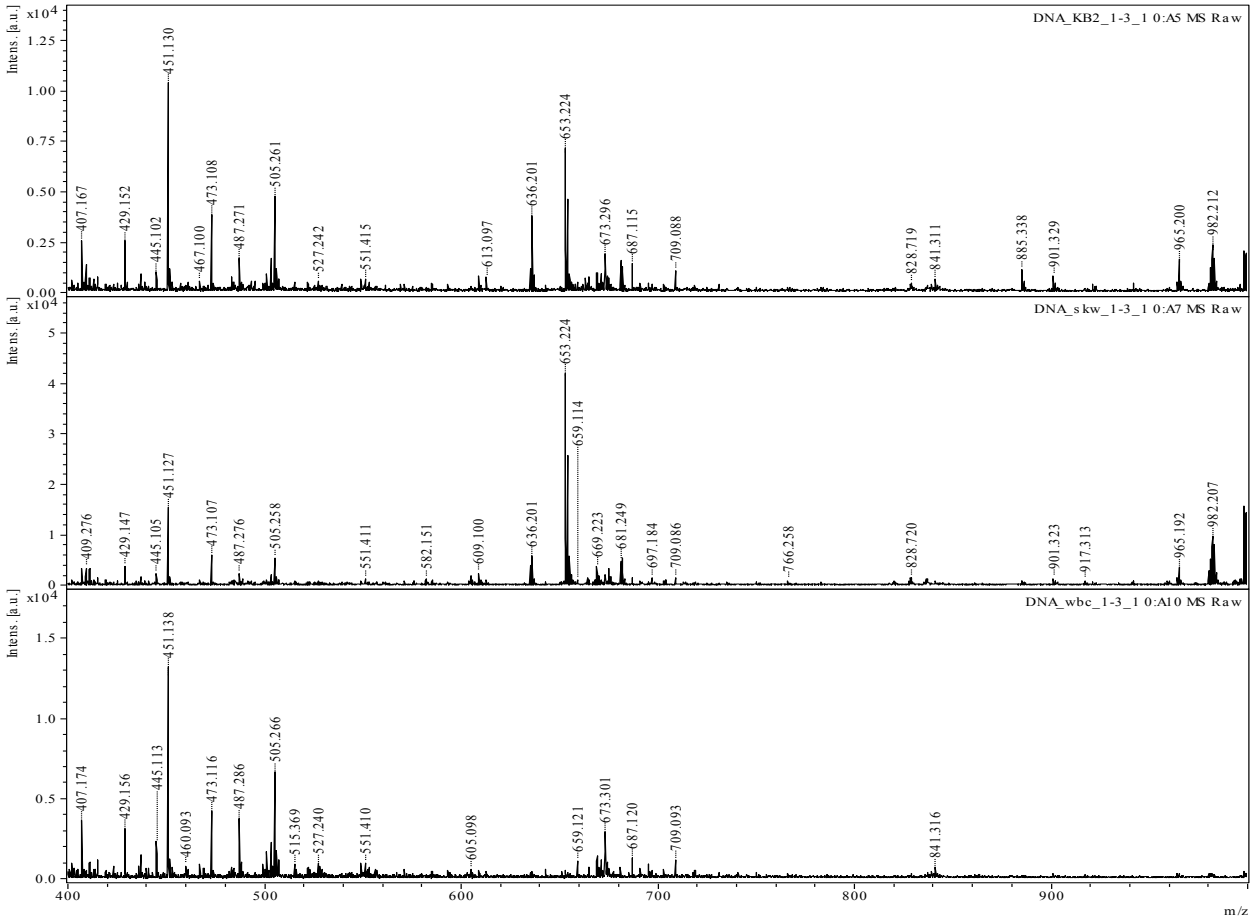


Figure 1 – Mass Spectra (400 Da to 1000 Da) of DNA from Normal Cells and Cancer Cells

In Figure 1, the top mass spectrum is for K562 Leukemia Cancer Cells. The middle mass spectrum is for SKW6 Normal Red Blood Cells. The bottom mass spectrum is for tWBC Normal White Blood Cells. Next, the different peak positions for red, white and cancer cells are noted. Tables 1-10 provide details of specific relative intensities at noted peaks. Comparing the spectra, it seems that cancer cells are less abundant at 407.17 Da peak verses more abundant 409.28 Da peak. See Table 1. The peaks at 407 Da - 408 Da correspond to uridine diphosphate or thymidine diphosphate of RNA. The 402 and 403 peaks may be from cytidine diphosphate. The normal cells are more abundant at 407 Da verses 409 Da peaks for more primordial isotopes. Beyond 409 Da to 415 Da peaks (cytosine diphosphate), the cancer cells are less abundant in nonprimordial isotopes, but normal cells are more abundant from 409 Da to 415 Da peaks in nonprimordial isotopes. The peak at 429 Da is from the adenosine diphosphate nucleotide and this nucleotide fragment is found enriched nonprimordials in cancer cells of heavier isotopes in DNA of the cancer cells. See Table 2. The 445 Da peak is from guanosine diphosphate (GTP - PO_3^{3-}) from RNA seems more abundant in nonprimordial isotopes for white normal cells and red blood cells relative to that in cancer cells. The 444-446 Da peaks distribution in red cells seems heavier than 444-446 Da peaks distribution for white normal cells. See Table 3. The role of ^{17}O may also explain the unusual isotopic content about the 445 Da peak. The 483 and 484 Da peaks

correspond to defunctionalized adenosine triphosphate. See Table 4. The clumping as by fewer peaks in cancer DNAs is observed and more peaks and finer structure of peaks are observed in normal DNA. The cancer DNA at 483 Da appears to have loss a nonprimordial, more massive functional group from a more massive peak and the normal cells at 484 Da appear appear to have loss a primordial, less massive functional group from a more massive peak. Adenosine triphosphate is observed at peaks of 506 Da and 507 Da; cancer DNA is enriched with nonprimordials at heavier 506 Da peak relative to 503 Da peak for more clumped nonprimordials. See Table 5. Normal DNAs have more enrichment at 503 Da peak with primordials. The peaks at 523-525 Da correspond to guanosine triphosphate and appear enriched with nonprimordials in the cancer DNA. See Table 6. 669 Da and 671 Da peaks are enriched in cancer DNA due to AT monophosphates. See Table 7. The 671 Da peak is enriched in nonprimordial isotopes in cancer relative to 669 Da peak; the white blood cells are enriched in primordial isotopes at 669 Da peak. 675 Da - 676 Da peaks may be AC monophosphates and these peaks reveal cancer is enriched in nonprimordial isotopes relative to white blood cells but these peaks reveal red blood cells are enriched in nonprimordial isotopes at 675 Da peak relative to cancer cells at 673 Da peak. The heavier 675 Da peak in cancer is due to ^{13}C and its ^{17}O. 681 Da - 683 Da peaks may be GC or GT monophosphates. See Table 8. The 680 Da and 681 Da peaks are enriched in primordial isotopes as by the T and C and the cancer is enriched in nonprimordials at 681 Da and 682 Da peaks. See Table 8. 697 Da - 698 Da peaks may be AG monophosphates; the 695 Da peak is enriched in primordial isotopes in white blood cells. See Table 9. The 697 Da peak is enriched in nonprimordial isotopes in the cancer cells. The cancer DNA may have ^{17}O on guanosine and the normal cells may have less guanosine. The peaks at 703 and 709 Da correspond to functionalized AG by OH or NH and the cancer DNA manifest clumped nonprimordials as observed by fewer peaks compared with the finer structure and many peaks of the normal DNA.

Discussion

General

The observed mass spectra of the DNA of normal and cancer cells and the displacements of the peaks in the range 400 Da to 1000 Da may be interpreted on the basis of the fragmentation of the DNA into nucleosides, nucleotides and oligonucleotides during MALDI mass analysis process with varying characteristic isotopic compositions of $^{2}D/^{1}H$, $^{13}C/^{12}C$, $^{15}N/^{14}N$, $^{17}O/^{16}O$ and/or $^{25}Mg/^{24}Mg$ within the fragments. The C to T → U and A → G has methylation, dehydration, deamination and hydration rings of aromatic, purine, pyrimidine and ribose rings and phosphate group into isotopically exchanged functional groups. On the basis of these varying isotopic compositions of the DNA in cancer and normal cells, the differing fragmentation patterns of the DNA can be reasoned. The varying isotopic contents can also be reasoned by different interactions, formations, replications, transcriptions, and translations of these nucleic acids in normal cells verses cancer cells.

Discussion of $^{13}CH_3$ for Enriched Thymidine

The 400-409 Da peaks may be U, T or C. The interconversions may be due to the nonprimordials so as to cause enrichments and depletions. See Figure 2. The T is at 402 Da, the C is at 403 Da and the U is at 404 Da. The U can ↔ T dehydroxylation and methylation and vice versa. And the T can ↔ C by dehydroxylation and amination and vice versa. By the prior theory of Little [1-3] Hydroxylation is critical for both these interconversions as the OH is strong

nucleophile and less subject to rehybridizations due to more electron electron interactions about O nuclei. The ^{17}OH accelerates both the bond breakage for methylation and ammination. This theory determines important $^{17}OH_2$ and $^{15}NH_3$ nanosolutions inside cancer cells such that the solvent $^{17}OH_2$ and $^{15}NH_3$ weaken bonds in the nucleotides to accelerate $^{13}CH_3$ nucleophillic replacement in the aromatic. Typically, aromatics are so stable that they are more difficult for nucleophilic aromatic substitutions but the negative NMMs of ^{17}O and ^{15}N lower the activation energies for accelerated substitutions on the nucleophiles. The ^{1}H on ^{17}O and ^{15}N modulates the negative NMMs of ^{17}O and ^{15}N for perturbing the covalent bonds in the nucleotides via oscillating e e pairs of the bases. The $^{15}NH_3$ and ^{17}OH are by their negative NMMs softer bases and better leaving on the basis of this theory so they explain the large massive loss from mass spectra of cancer DNA. These results and predictions of this theory are consistent with prior experimental observations of NH_3 recycling by breast cancer cells. But researchers have not experimentally explained $^{17}OH_2$ in cancer cells. But this theory predicts large anomalous $^{17}OH_2$ in cancer cells. The methylation is faster as ^{13}C has positive NMM. The negative NMM of ^{15}N may slow its amination. Dense nonzero NMM may accelerate the $^{15}NH_2$ deamination. The nonprimordials in U may cause it to manifest 409 Da peak in the cancer and the enrichment of nonprimordials and clumping in the cancer DNA. Such nonprimordials in cancer U may accelerate its conversion to T by accelerating dehydroxylation of ribose and accelerating methylation of pyrimidineby $:^{17}OH_2$ complexation and $:^{15}NH_3$ complexation of U surrounding nano NH_3 and H_2O nanosolution. The ^{16}OH in the ribose of cancer U more rapidly fragments its bond to the ribose due to the null NMM of the ^{16}OH and the ^{17}OH rapidly replaces ^{16}OH but during mass analysis may be fragmentation from T nucleotides of cancer DNA to explain the spectra. The $^{13}CH_3$ better stronger nucleophile more rapidly attacks the pyrimidine due to the positive NMM of ^{13}C of $^{13}CH_3$. The nonprimordials at 409 Da peak may thereby more rapidly form the T at 402 Da peak with enriched nonprimordials at 402 Da in cancer. It is important to note further that this theory determines greater solubility and greater complexation of $^{13}CH_3$ by nanowater of $:^{17}OH_2$ and $:^{15}NH^3$ relative to primordial $^{12}CH_3$. The cancer DNA is heavier at 403 Da and indeed the clumping of nonprimordials in the formed T has even greater intensity in cancer DNA at 405 Da (vs 403 Da) relative to normal cell at 403 Da vs 405 Da. The methylation of the U and dehydroxylation are expected by prior theory [1-3] to have enriched nonprimordials in the T in cancer DNA. (So in general the $^{13}CH_3$ is a stronger better nucleophile and stronger base than $^{12}CH_3$ due the harder basicity $^{13}CH_3$ relative to $^{12}CH_3$. So now in general ^{17}OH ($^{15}NH_2$) is a better leaving group than ^{16}OH ($^{14}NH_2$) and a weaker base than ^{16}OH ($^{14}NH_2$) due to the greater polarizability of ^{17}OH ($^{15}NH_2$) to ^{16}OH ($^{14}NH_2$)).

Discussion of Interconversion of Pyrimidines (C, T and U)

The T may convert to C by deamination and hydroxylation and demethylation. So in cancer the nonprimordials may accelerate the deamination of T as $^{15}NH_2$ is a good leaving group relative to $^{14}NH_2$. See Figure 2. But the hydroxylation may be less in cancer (as ^{17}OH is a weaker nucleophile than the ^{16}OH) relative to deamination in cancer DNA. But the $:OH_2$ is stronger base than $:NH_2$ and electronic rehybridization is more labile in OH due to greater electron density about the O nucleus relative to N nucleus. In cancer, the demethylation may be slower than in normal cells as $^{13}CH_3$ of the cancer may be a poor leaving group. So the cancer may less transform its T* to C* relative to the normal cells so the peaks for C* (403 Da) in cancer should be depleted in nonprimordials isotopes. The 402 Da peak for C is observed depleted in primordials in the cancer relative to the observed 402 Da in the normal DNA,

revealing more primordial in normal DNA. In fact, the normal cells show peaks at both odd and even masses 402, 403, 404, 405, 406, 407, 408, 409 and 410 Da as the T* at 403 Da shows nonprimordials for odd masses and the T at 402 Da for less clumping of even masses of nonprimordial isotopes in the normal cells. But the cancer DNA shows only odd peaks 403 Da, 405 Da, 407 Da and 409 Da for the formation of T* (403 Da) but no formation of C* (404 Da) in the cancer as the U* → T* but T* does not → C* in cancer. The data thereby reveal depleted C* in the cancer enriched **T***. But all these transformations occur in normal cells. It may be that during cell division cancer nuclei have too much thymidine (T) and too little cytidine (C) as thymidine accumulates in normal cells to cause the normal cells to transform to cancer cells.

The depletion of the primordials in cancer DNA at 409 Da peak for UTP and enrichment of primordials in cancer DNA at 407 Da peak may be reasoned by cytidine becoming more rapidly methylated with $^{13}CH_3$ relative to $^{12}CH_3$ during cancer genesis so that the cytidine may transform to uridine and thymidine for altering the ($^{13}CH_3$) isotopic compositions of C, T and U in the cancer cells as the nonprimordial C in cancer more rapidly functionalizes and defunctionalizes to enrich T and U with nonprimordials (^{13}C) and during the mass spectra under the electromagnetism the cancerous DNA less readily fragments (under the strong electromagnetism of the mass spectrometer) near dense regions of nonprimordial kernels so the cancer DNA has fragments nonprimordial enriched at 409 Da peak and enriched primordials at 407 Da peak. The $^{13}CH_3$ is a stronger nucleophile by this theory so it more rapidly attacks C in the $^{15}NH_3$, $^{17}OH_2$ DNA in nanosolutions in cancer cells. This theory thereby predicts explain more rapid methylation of C in DNA of cancer. Moreover this theory further discloses the more rapid conversion of C* to U as the surrounding nanowater in cancer cells complexes the ^{16}O and ^{14}N in the C to accelerate conversion in C* (methylated) C and to then accelerate the deamination of pyrimidine of C* for loss of ^{14}N and replacement by OH to form U*. Thereby C is not only by this theory prevented from forming from T*. The C in cancer is accelerated to form U*. It may also be possible that ^{17}O is enriching in the phosphorate group of the nucleotide. So the cancer is heavier beyond 409 Da to 415 Da peaks due to ^{17}O on phosphates and varying protonation ^{17}O on $^{31}PO_3^-$ and ^{17}O and ^{15}N on the purines of guanosine diphosphate. Normal cells have ^{16}O on $^{31}PO_3$ with stronger protonation and ^{16}O and ^{14}N on purines of guanosine, but cancer DNA may accumulate ^{17}O in $^{31}PO_3$.

Clumping of Nonprimordials in Cancer DNA and Enriched Adenosine from Guanosine

The 429 Da peak may be enriched in nonprimordials in cancer due to adenosine diphosphate and its formation from guanosine diphosphate by dehydration and deamination G and aminating the intermediate may thereby result from faster functionalizations and defunctionalizations of ribose and pyrimidine rings by methylations and deaminations for thymidines having $^{13}CH_3$. See Figure 3. The normal cells have more local peaks about 429 Da relative to cancer 429 Da peak as cancer has more clumped nonprimordials. So the 429 Da nucleotide with nonprimoridial ^{17}O in cancer DNA rapidly loses functional group; so the 429 Da peak is less present, but the fragments in the cancer having primordial ^{16}O show larger peaks as they fragment less by loss of their ^{16}O. The formation of the 429 Da from the 445 Da for G ↔ A involves deamination and dehydration of G then amination to A. The cancer cells have nanowater with $^{17}OH_2$ and $^{13}NH_3$ to complex the OH and NH_2 of G in cancer to convert to G* with ^{15}N and ^{17}O replacement in cancer G*. The ^{15}N and ^{17}O more readily undergo nucleophilic aromatic substitution by ^{14}N to form the A from the G by this theory to explain the data. The

clumping of nonprimordials in cancer DNA and G nucleotide may accelerate the deamination and dehydroxylation of G as cancer has $^{15}NH_2$ and ^{17}OH which are poor nucleophiles and good leaving groups due to their negative NMMs. The $^{15}NH_2$ is a stronger nucleophile than ^{17}OH as ^{15}N has a less negative NMM than ^{17}O; so $^{15}NH_2$ is expected to be harder than ^{17}OH and $^{15}NH_2$ a better nucleophile than ^{17}OH for replacing ^{17}OH of G* to form $^{15}NH_2$ of A*so the $^{15}NH_2$ harder base should attack the purine rings faster than ^{17}OH weaker nucleophile. Thereby cancer DNA should readily transform G* ↔ A* for unusual mutations. It may be that cancer cells show excess of adenosine and deficiencies of guanosine so this may reflect in anomalous RNA transcription and protein translation in cancer anabolism. Red blood cells show similar isotopic distributions relative to normal cells as the thymidine and cytidine may not functionalize their ring with ^{17}O as ^{17}O defunctionalizes from guanosine.

Clumping of Nonprimordials in Cancer DNA and Enriched Guanosine → Adenosine Diphosphate

The unusual enrichment in the 445 Da peak of cancer with nonprimordials (relative to 443 Da for primordial G) may be explained by the A* in the 429 Da peak of A* as the A may reversibly undergo uphill slower process of deamination, hydroxylation and amination to form the G at 445 Da peaks so that the clumped nonprimordials in G more accelerate the loss of ^{15}NH and the gain of ^{15}N and ^{17}O to form A, relative to primordials in A of normal cells to form the nonprimordial G in the cancer; so the peak at 445 Da peak is nonprimordial enriched in cancer DNA as the nonprimodials compose A leaving the nonprimordial enriched G at 445 Da peak rather than 443 Da. See Figure 3. Cancer DNA at 445 Da is heavier with nonprimordials relative to normal white cells DNA. Vice versa in the cancer cell the G* may readily due to its clumped nonprimordial isotopes transform to A*. The dehydroxylation and deamination and amination of the G* in cancer DNA is accelerated due to the negative NMMs of the ^{15}N and ^{17}O for ease of leaving ability in the dehydroxylation and deamination. But the amination to form the final A* is expected in cancer DNA as the $^{15}NH_2$ is a stronger base and better nucleophile than ^{17}OH due to the harder basicity of $^{15}NH_2$ relative to ^{17}OH and due to the less negative NMM of $^{15}NH_2$ relative to more negative NMM of $^{17}OH^-$. The hydrogens also help harden the $^{15}NH_2^-$ as there are more H in $^{15}NH_2$ relative to ^{17}OH.

Uridine, Cytidine and Thymidine Triphosphides and Nonprimordial Clumps Block T → C in Cancer DNA

So the heavier U (484 Da), T (482 Da) and/or C (483 Da) in cancer couples by chemical transformation to 502 Da of U* (methylated U) in cancer by methylation ($^{13}CH_3$, 16Da); by U* dehydroxylates to form T* (with $^{13}CH_3$, 16Da); by U* dehydroxylate (^{17}OH, 18Da) and aminates ($^{15}NH_2$, 17 Da) to form C*. See Figure 2. Such many possible dynamics manifest nonprimordial accelerated functionalization and defunctionalizations in cancer cells relative to normal cells and the resulting nonprimordial induced chemical transformations of U → U*, U* → T* and/or C* → U* for a new mutation of DNA and RNA as here disclosed not by changing isomeric connectivity along chains but by interchemically converting nucleotides of U, T and C! Such complex inter-chemical conversions are observed in the mass spectra of the cancer relative to the normal cells. So the normal cells have finer peaks in this range 400 - 409 Da. The cancer DNA less fragments to form U, T, and C and have less fine spectra due to nonprimoridial clumping from 400 to 409 Da. The nonprimordials ($^{13}CH_3$) in the cancer nucleotides may cause less fragmenting of cancer DNA for fewer of these peaks from 400 - 409 Da. The normal cells have

random methylations and the random methylations of C and random $^{13}CH_3$ in C, T, and U can lead to such fine peaks in the normal cells. But the cancer cells have nonrandom, clumping of $^{13}CH_3$ and $^{15}NH_2$ and ^{17}OH and the $^{13}CH_3$ causes stronger binding of the cancer DNA for less fragmentation under electromagnetic fields in mass spectrometer. There is more 483 Da in cancer DNA and there is more 484 Da in normal DNA so more T* in cancer DNA and more C* in normal DNA. These trends for cytidine, uridine and thymidine triphosphates are consistent with the peaks at 400-409 Da for the diphosphates as the diphosphates also revealed less T to C for cancer DNA. The T* → C* conversion in cancer would involve the dehydroxylation of pyrimidine and hydroxylation of ribose and the amination of the pyrimidine. The negative NMMs of the ^{17}OH and $^{15}NH_2$ in cancer makes this less likely. As the negative NMM of ^{15}N and ^{17}O make $^{15}NH_2$ weaker nucleophiles for the conversion of the pyrimidine to C* in cancer DNA. The cancer DNA thereby less expresses C* at 483 Da.

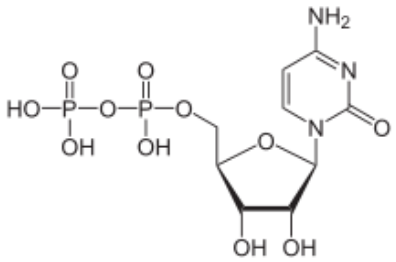
Dehydration of Adenosine Triphosphate and Suppresion by Clumping $^{13}CH_3$

The 487 Da is from the dehydroxylation (^{17}OH or ^{18}OH $^{17}O^2D$ or $^{18}O^2D$ of 18 Da to 20 Da) of ribose in adenosine triphosphate at 507 Da. See Figure 3. In normal cells the dehydroxylation is more than in cancer cells as the cancer cells have more ^{13}C in the ribose, which bind the ^{17}OH more strongly. So cancer at 507 Da should be heavier. But cancer is observed not to be heavier at 507 Da as the 507 Da is coupled to 523 – 525 Da by dehydroxylation. So 523 to 525 Da of G* in cancer losses ^{17}O (rather than ^{16}O) to cause less massive peaks at 482 Da cancer hydroxylates better if it is heavier (clumped with ^{13}C or $^{15}NH_3$) so the 507 Da peak in cancer lacks heavier nonprimordials as they are in loss ^{17}OH from 523 – 525 Da to G* so they are missing 507 Da peak A* at 525 Da peak in G*. This conversion of nonprimordial G* to nonprimordial A* in the cancer DNA is expected as the G* to A* involves the deamination and hydroxylation and amination of the purine. The cancer having clumped nonprimordials may accelerate this as the $^{15}NH_2$ and ^{17}OH in the cancer DNA are weaker nucleophiles (due to their negative NMMs) relative to $^{14}NH_2$ and ^{16}OH. But in principle $^{15}NH_2$ and ^{17}OH should be poorer entering groups due to their negative NMMs but ring ^{15}N can pull in the $^{15}NH_2$ and ^{17}OH nucleophiles. It is observed that cancer is heavier at 525 Da G* relative to normal cells being lighter at 523 Da. As the 523-525 Da is guanosine triphosphate and the ^{17}O on the guanosine triphosphate stabilize the ^{17}O and the $^{13}CH_3$ for less massive peaks at 505 Da and less massive peaks 487 Da in the cancer DNA samples due to losses of heavier ^{17}O and ^{13}C respectively. So this is general principle when ^{17}O is active in fragmenting the daughter peaks are enriched in less massive than peaks in primordial normal DNA. When $^{13}CH_3$ is active in the fragmenting the daughter peaks are enriched in nonprimordials as the $^{13}CH_3$ fragments stabilize by $^{13}CH_3$ with consequent heavier daughter peaks. The nonprimordials and the ^{17}OH destabilize stabilized by $^{13}CH_3$ the primordials. So the enrichment in the daughter by primordials is actually due to lack or less fragmenting of nonprimordial than primordials.

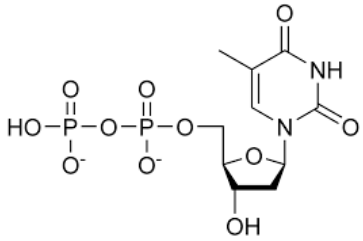
Adenosine Triphosphate Form From Guanosine Triphosphate

The 506 Da and 507 Da peaks can also be explained on the basis of their A contents. It can be that guanosine triphosphate at 523 Da peak loses ^{17}O to form 507 Da peak {which corresponds to adenosine triphosphate} and the clumped nonprimordials help loss of ^{17}O to explain the patterns. G → A. See Figure 3. The nonprimordial G at 525 Da peak more rapidly loses ^{17}O to produce more than 50% than ^{16}O is lost to produce 507 Da in the cancer. Thereby here it is proposed that nonprimordial isotopes epigenetically alter nucleic acids in cancer by

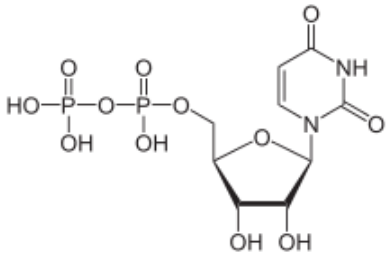
cause G → A. The 523 Da peak may involve transformations between A and G with a surrounding peak; so that in cancer the peak enriches in primordial isotopes. A → G by hydroxylation, deamination, and amination. G → A by dehydroxyation, deamination and amination. Ammonia in tumor can encourage aminating and deaminating G and A, also in C → T.



Cytidine diphosphate



Thymidine diphosphate



Uridine diphosphate

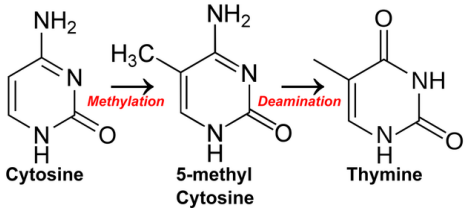
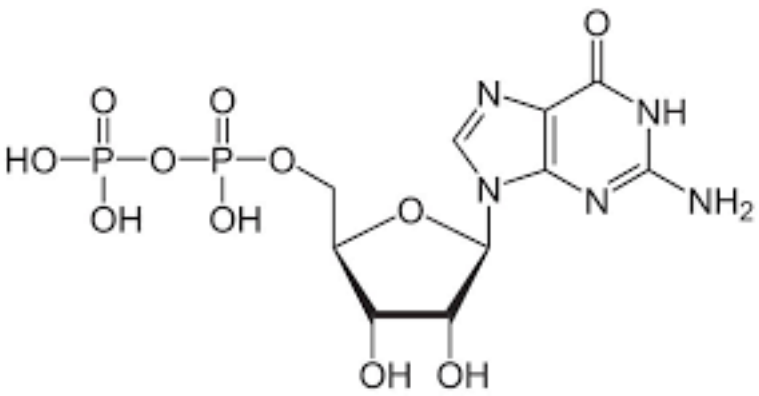
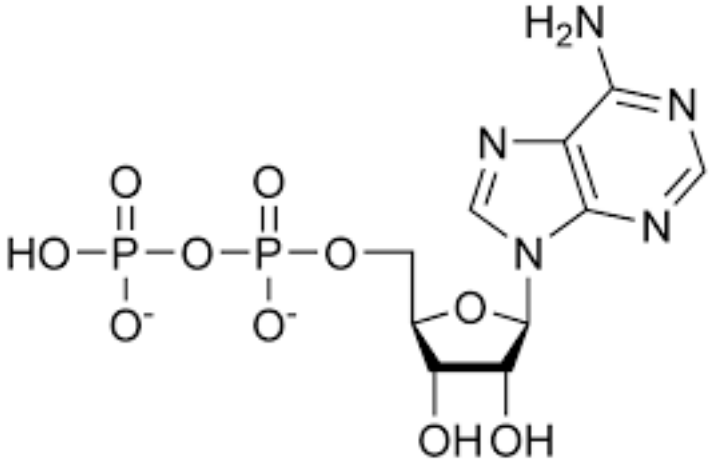


Figure 2 – Pyrimidine Nucleotides



Guanosine Diphosphate



Adenosine Diphosphate

Figure 3 - Purines

Heaviness of AT in Cancer DNA

The AT fragment associated with the 669 Da to 671 Da peaks and T in AT may be the reason the cancer DNA is enriched in nonprimordial isotopes as the T may form from $^{13}CH_3$ methylation of cytosine and the cytosine may undergo deamination and dehydroxylation or the C may → U by deamination and hydroxylation under acidic conditions as in altered nuclei (isotopic replacements) as nuclei are more basic than cytoplasma. The more basic nucleus in cells stabilize T and U as T is more basic and nonpolar relative to U. So in cytoplasma the T → U as the more acidic cytoplasma can push out $^{13}CH_3$. Quite interesting that AT are detected as in cancer AT are thought enriched and GC are thought deficient in cancer. Again in the cancer the accumulation of nonprimordial T* is observed as the T* cannot (due to clumped nonprimordials) convert to C* as the conversion of T* would require demethylation (loss of $^{13}CH_3$). The $^{13}CH_3$ is a strong base and good nucleophile and the cancer cells cannot as well lose $^{13}CH_3$. The heavier 675 Da peak in cancer is due to ^{13}C and its ^{17}O.

The 680 Da and 681 Da peaks may be explained by isotopic distributions in GC or GT. The 680 Da and 681 Da peaks of normal cells are enriched in primordial isotopes as by the T and C have more $^{12}CH_3$ and $^{14}N_2$ but the cancer DNA is enriched in nonprimordials at 681 and 682 Da peaks due to the isotopic clumping of nonprimordials to enrich the ^{13}C methylation of C to form $^{13}CH_3$ in T* also having ^{15}N. There is more GT in cancer than normal cells. There is more GT in cancer than AT. GT has stronger binding due to the 3 hydrogen bonds relative to only 2 hydrogen bonds in AC. G is deficient so why so much TG? Although deficient G binds strongly to T. Again the enrichment of $^{13}CH_3$ in T* in nonprimordial cancer is detected and the inability to convert T* to C* in the cancer Red Blood Cells are enriched at 682 Da peak relative to cancer at 681 Da peak and this could be due to ^{17}O in G, C and T in the red blood cells as the red blood cells couple to air for ready oxygenation. It may be possible to relate cancer to ^{17}O from the air as well as ^{17}O in the water. So the blood can accumulate ^{17}O from $^{17}O_2$ and $H_2{}^{17}O$ and $^{13}C^{17}O$. The red blood cells are different from white blood cells. The red blood cells may be a basis for the cancer spreading the ^{17}O to normal cells.

GA and Loss of G in Cancer DNA

The unusual enrichment of primordial isotopes in cancer AG at 695 Da and 697 Da peaks may be reasoned on basis of G content in GA and the cancer may have ^{17}O and ^{15}N on guanosine and many normal cells have less ^{17}O and ^{15}N on guanosine. There is observed that there is less GA in cancer DNA than GT or GA fragments less than GT. Less observed GT is consistent with the discovery of transforming G to A in cancer genesis of this work. But the observed greater 695 Da relative to 697 Da in cancer may be explained by this theory. So the ^{17}O is more rapidly lost from guanosine of cancer DNA relative to less lost of ^{16}OH from guanosine for the greater 695 Da peak relative to 697 Da peak for cancer. The 695 Da may be coupled thereby to 695 Da+ 14 Da = 709 Da peak or the 695 Da + 9 Da = 703 Da. This 703 Da peak should be enriched in clumped nonprimordials in the cancer as by loss of O^{2-} from G or A. The 14 Da may be loss of 14 Da or NH_2 – from G or A. The cancer DNA shows both 703 and 709 Da peaks and manifest this clumping. But the normal cells do not show such peaks at 703 Da and show a small peak at 709 Da in support of this reasoning. The guanosine may be more reactive due to ^{17}O relative to ^{15}N as the ^{17}OH is stronger nucleophile than the $^{15}NH_3$ and NH_3 is less abundant in normal cells!

It seems in general ^{17}O helps decomposition and fragmentation. The $^{17}OH_2$ and $^{15}NH_3$ in surrounding nano-water in cancer cells may accelerate exchange of $^{12}NH_2$ and ^{16}OH by $^{13}NH_2$ and ^{17}OH. Scientists have not measured ^{17}O in mass spectra and NMR enough to see this effect of ^{17}O as determined in this work. Most prior work on O has focused on ^{16}O and ^{18}O. The complexation of this biomolecules by ^{17}OH and $^{15}NH_2$ causes a softening of the bonds for faster substitution and replacement reactions due to the negative NMMs of ^{17}O and ^{15}N.

So in general where $^{13}CH_3$ reactions are accelerated in cancer the methylation consistently shows heavier peaks in cancer DNA and its pieces. But where ^{17}OH and $^{15}NH_2$ are involved the amination and hydroxylation consistently show smaller masses in the mass spectra of cancer DNA and its pieces. The larger massive pieces during methylation result and are explained by the addition of more massive $^{13}CH_3$ in the functional of DNA nucleotides. The less massive pieces during amination and hydroxylation are explained as resulting from loss of more massive ^{17}O and ^{15}N from the functionals of cancer DNA and its nucleotides. In general the $^{13}CH_3$ and its postitive NMM strengthen the covalent bonds in cancer DNA for binding $^{13}CH_3$ is a stronger nucleophile for more rapid replacement in DNA and its nucleotides. But the $^{15}NH_2$ and ^{17}OH and their negative NMMs weaken the covalent bonds in cancer DNA for bond breakages and ^{17}OH and $^{15}NH_2$ one better leaving groups for more frequency of ^{15}N and ^{17}O of nucleotides under electromagnetic fields during NMR analysis to explain these observed mass spectra.

It may not be that ^{17}O and ^{13}C attract or repel by internal C frame magnetism. It may be that they self conform to form quanta. So all + NMM → classical or all – NMM → classical, but balanced + NMM and – NMM → quantum and the monopoles separate locally but bind globally. So on one scale they may bind and on larger scale repel or vice versa. So ^{14}N drives biomolecules by imbalance perturb e^- e^- quanta ^{15}N may disrupt such natural imbalance of ^{14}N; ^{17}O also disrupts the ^{14}N imbalance ^{13}C disrupts e^- e^- quantum mechanics and ^{14}N cannot help ^{13}C. But ^{17}O can help ^{13}C. But ^{15}N can help ^{13}C at higher temperature. ^{17}O disrupts ^{15}N quantum mechanics but together they help pull in ^{13}C and less ^{14}N causes loss of protein nuclear perturbation. ^{13}C may overdrive classical of protein with ^{1}H and ^{14}N. ^{13}C causes accelerated glycolysis as driven fragmentation of glucose. But the combining of C to O is opposed by ^{13}C and ^{14}N in the Kreb cycle or they oppose $sp^3 \rightarrow sp$, sp^2. + NMM favor sp^3, - NMM favor sp and sp^2 for ^{13}C but not for ^{17}O. So ^{13}C favor sp^3 and ^{17}O favor sp^3 (for different reasons) as higher e^- e^- density for + NMM of ^{13}C increases electron density on C and less e^- e^- repulsion for negative NMM of ^{17}O reduces electron repulsion about O. So ^{13}C and ^{17}O accelerate glycolysis by one environment. But ^{13}C and ^{17}O suppress the Kreb cycle as in the Kreb cycle the sp^2 and sp hybridizations are catalyzed about C and O and the ^{13}C and ^{17}O oppose such sp and sp^2 hybridization but favor sp^3 hybridization. But ^{17}O and ^{13}C decelerate Kreb cycle by different environment.

In this work, the author proposes a new way to alter functional groups of uridine, thimine, cytosine, adenine and guanine (by isotopic substitution/replacements of ^{1}H, ^{16}OH, $^{14}NH_3$, $^{12}CH_3$, and ^{24}Mg by nonprimordials of ^{17}OH, $^{15}NH_3$, $^{13}CH_3$, ^{2}D and ^{25}Mg) as nonprimordial functional groups enter and to replace primordial functional groups of nucleotides by this new theory as by the many aromatics of the purines and pyrimidines oscillating their electrons to couple the many nonzero NMM of these nonprimordial functional groups for activating their nucleophilic substitutions of primordial functional groups. The theory [1-3] introduces novel chemical

dynamics of multiple electrons and multiple functional groups in nano-domains behaving nonclassically to couple their spins and electronic motions to violate the 2nd Law of Thermodynamics momentarily as energy is focused into specific fewer atoms of the group to catalyze transport, transformations and momentary transmutations for novel chemical dynamics of many bodies as the nanodomains by this theory gets quantum mechanically into a single atom or small molecule by Little Effect the fermionic atoms by their nuclei (NMM) are in analog to fermionic electrons in atoms so that the atoms in the domain have a wave nature and the exchange and correlate to move and alter a wave nature and the exchange and correlate to move and alter motion and position in the nanosolution so as to lower energy. Such motions and altered positions manifest new chemical changes of the atoms, small functional groups in the nano-domains of proteins, nuclei acids and nanowater and ammonia. So that the biochemical transformations have been previously described by the author as a nanoscale quantum wave mechanics that manifest at lower temperatures for fermionic nuclei having nonzero NMM, but higher temperatures and pressures and E, B can induce the quantum wave mechanics of nanosolutions composed of null NMMs.

So inside the nucleus GATC are the nucleotides but outside nucleus GAUC are the nucleotides. Methylation $^{13}CH_3$ of U causes T*. So isotopic effects in cytoplasma get into nuclei by U + $^{13}CH_3 \rightarrow$ T* in cytoplasma and transfer of T* into nucleus. So $^{13}CH_3$ on T* in nucleus causes altered genetics as reasoned by this theory. In prior work, it was previously published U expresses as T* due to $^{13}CH_3$. So $^{13}CH_3$ seems like H (by their positive NMMs); so T* becomes as U; and U in nucleus alters genes. Normally U is in cytoplasma and T is in nucleus. So by U $\rightarrow$ U* $\rightarrow$ T*, U* is transport into the nucleus via T*, the replication of DNA is altered by such U* and T* in the nucleus of cells as $^{13}CH_3$ methyl on the thymine alters biochemical dynamics. Also $^{13}CH_3$ in T* may accelerate T* $\rightarrow$ C* by dehydroxylation, deamination, and amination. So this causes mixing of nucleotides and mutations by chemically interconverting of nucleotides. T $\rightarrow$ U. U $\rightarrow$ C. Such chemical transformations of nucleotides alter the genetic code to cause cancer and other diseases. This theory [1-3] further proposes that the external static magnetic fields and radiofrequency fields can excite these nanosolutions to accelerate these nonprimordial substitutions. It may be that such chemical transformations of nucleotides in normal cells to mutate normal cells to cancerous cells are kinetically and thermodynamically possible by a few nonprimordial substitutions, but with more and more nonprimordial substitutions the replacements are slower or not allowed. Such chemical transformations may occur as normal cells transmute to cancer cells with higher amounts of NH_3 in the cancer environment. But this theory proposes that the use of external magnetic fields for stimulating cancer cells so their DNA pull in more nonprimordials so the excess nonprimordials kill the cancer. With such rapid replication of cancer DNA it should be easy to disrupt the genes in cancer so the cancer cannot produce its proteins for glycolysis to kill the cancer.

Adenine is unique as it is the only nucleoside lacking O group and has only N functionals and the N is weaker base and weaker nucleophile than O as in guanine, uridine, thymidine and cytidine. It is on this basis of RBL that the ^{17}O in water is the basis for the enrichment of ^{17}O in DNA and RNA and the ^{17}O in the many rings help the ring pull in ^{13}C as by ^{17}O activating bond cleavage of ^{17}OH and + NMM but may ^{14}N, ^{1}H and ^{33}S and other ^{13}C can induce, new bond formations, but careful as excess + cleaves + ⋯ + bonds and excess – cleaves - ⋯ - bonds in quantum fields. So quantum fields + ⋯ - globally bond and + ⋯ + locally bond and - ⋯ -

locally bond and as the nonprimordial isotopes clump they manifest new enzymatics of the DNA and RNA. So this theory of RBL introduces totally new chemical dynamics as here it is determined novel nonlocal chemical bonding but local chemical decomposition and/or nonlocal chemical decomposition but local chemical bonding. But when do these manifest chemical bonding? But when do these manifest nuclear bonding? The patterns of null, + and – NMM (needles in haystack) can cause local bonding while globally the fermions are unbound. So RBL determines that systems of + and – Nuclear Frames bind the atoms whole on large scales they repel and are chemically broken. This is why ^{13}C and ^{17}O and ^{15}N activate transition states and lower the barrier to chemical substittions of isotopes. But RBL, the + and- NMMs as is more common in our sector of the Universe (or in other sectors – and -) locally on nuclear scales repel but on global scales they bind/attract. So this also in other sectors of Universe with – NMMs have – NMM interacting with – NMM repel locally in nuclei but bind attract globally as in Ag nanoparticles and other rare elements having all – NMMs. But such considerations, RBL gives a totally new model for transport (superconductivity) and transformations {chemical and biological dynamics}. So prior chemistry and transport have focused primarily upon + ⋯ + NMMs and the globally binding by $e^- e^-$ and the locally repelling /unbinding by NS Frame with less chemistry and transport possibilities. Such manifest in primordial nanosolutions in cells having + NMMs of ^{14}N and ^{1}H and ^{31}P and null moments of ^{12}C, ^{14}N, ^{16}O and normal primordial biology manifest on such basis of repulsions on NS Frames and motions and biochemistry of binding on L frame of wavefunctions. But RBL introduces totally new effects of – NMMs + ⋯ + NMMs binding locally in NS Frames and repelling globally in L frame. So bonds are broken globally to isolate the $e^- e^-$ but locally the $e^- e^-$ bind by the + NMM and – NMMs to manifest a Reggie Pair bond by NMMs of + and – NMMs as this occurs in nanosolutions in cells as $^{17}OH_2$ and $^{15}NH_3$ enrich with $^{13}CH_3$ in the nanosolution and proteins and nucleic acids so the nanosolutions bindon NS Frame but globally the $e^- e^-$ are more broken chemically so the proteins and nucleic acids have different motions, binding enzymatics and biochemical reactivity. Such theory explains the cancer cell as the protein nucleic acids interactions are altered by the + and – NMMs cause wavefunction repulsions but the nuclei still pin the atoms together for cancer habitat.

It is important to consider that by such model of RBL, in normal cells the ^{14}N and ^{1}H can modulate the bond cleavage and bond formations of PO_3^- and the ribose as the compression may induce bond cleavage of $^{31}PO_3^-$ to release energy and the chemical compostion of ribose) of null NMMs). As compression break + NMMs of PO_3^- but bind C-C-O-H of ribose of O (null) NMMs. But then the rarefaction binds PO_3^- and fractional decomposes ribose and these can couple to pull apart base pairs or also such dynamics couple to surrounding proteins to bind or decompose the protein to pull in or push out proteins. And such can explain DNA replication quantum mechanically as bases recognize quantum mechanically by patterns of NMMs and compress/rarefy pulling in and pushing out. And likewise for transcription. And in ribosome such acts vice versa as pulling in amino acids under condiyions whereby the oligonucleotide , RNA is stable.

The clumping may help ^{15}N incorporation into the oligonucleotides. The functional groups can dynamically shift the functionals to find equilibrium with the kernelling of nonprimordials lowering the energy relative to random distributions of the nonprimordials in normal cells. Such clumping of dense regions of nonprimordials isotopes alters nuclei acid binding, bond strengths and chemical stability as by enzymatic actions on the kernel regions. But the clumps in normal cells may be linked to noncoding regions of DNA. So later the

oligomers of food tannins can modify the functionals in cancer cells more than in normal cells to kill the cancer cells!

The guanosine may be more reactive due to ^{17}O relative to ^{15}N as the ^{17}OH is stronger nucleophile than the $^{15}NH_3$ and $^{15}NH_3$ or $^{14}NH_3$ is less abundant in normal cells! It could be that the presence of $^{14}NH_3$ causes the genetic alterations of normal cells to cancer cells and the $^{15}NH_3$ helps as by mutating genes. Comparing the various signals, the FWHM of signals from fragmented DNA in normal cells appear broader relative to the signals of fragmented DNA from cancer cells (note that this points to clustering of nonprimordials in cancer DNA and this narrow FWHM of cancer DNA is consistent with clustering of nonprimordials to dense kernels in the cancer DNA). The smaller FWHM in cancer DNA fragments may be near and from the clumping of nonprimordial functional groups of deuteron, hydroxyl, amine, deuterated, and methyl groups. Such clumping of nonprimordials leads to sharper distinct fragmentations during the mass analysis of DNAs for sharper peaks relative to broader peaks in fragmenting of the primordial regions of DNA. By the theory, the incorporation of nonprimordials of ^{2}D, ^{13}C, ^{15}N, ^{17}O and ^{25}Mg into cancer DNA by functionalizations and defunctionalizations of the nucleotides appear to explain these observations of DNA isotopic differences between cancer and normal cells.

So after considering these different causes of the functional groups in cancer and in normal cells on the basis of based on the spectra a discussion of the proclivity of nucleotides and oligonucleotides to the new chemistry is next given. The aromatic and the ring structures as RBL previously modelled such biomolecules on the basis of Na^+ and K^+ interaction with graphene oxide. It was determined that Na^+ and K^+ NMM, interact favorably with graphene oxide with its sp^2 and sp^3 mixed hybridization and magnets via the nonzero NMM of K^+ and Na^+. Thereby likewise RBL reasoned similar NMMs interact with sp^2 and sp^3 now networks in biomolecules like DNA. So that RBL introduced changes in interactions in the DNA as primordials of ^{1}H, ^{12}C, ^{14}N, ^{16}O, ^{24}Mg, and ^{32}S are replaced by nonprimordials of ^{2}D, ^{13}C, ^{15}N, ^{17}O, ^{25}Mg and/or ^{33}S of different NMMs. AS the purines and pyrimidines in nucleic acids regions of sp^2 aromatic and region of sp^3 nonaromatic as the different regions in graphene oxide.

Why Do Nucleotides Transform on Atomic Scale

Thereby RBL realized nuclear spins could couple to carbon covalent dynamics from his prior graphene work. So by considering graphene an analog for proteins and other biomolecules, RBL proved his prior theory that nuclear spins in general can couple to biomolecules to alter catalysis and enzymatics of biochemical reactions. Next in this work, the mechanism by Little's Effect are given for driving the replacements and substitutions of null NMMs by nonzero NMMs. The more extended aromatic rings may couple spins of the nuclei for faster clumped accelerated isotopic enrichments of the ring systems via the aromatic π electrons as the aromatic electrons couple the separated nuclear magnetic moments (NMMs) and induced transport exchange and replacement of the different NMMs. This extended π electrons and orbital exchange and bonding about many atoms may be a mechanism for coupling the spins {Reggie Acids and Bases of electron radicals (fermions) and nuclear spins (fermions) and nuclear radicals and orbitals} to orbitals (of Lewis Acids and Bases, both electronic and nucleonic) via the π electrons the nuclear spins and the nuclear orbital angular momenta are thereby exchanged and coupled via delocalized π e^- e^-. Also by this model of RBL, such spins are not limited to e^- spins;

nuclear spins are also coupled, transformed, transported and transmuted by π e^- e^- and d orbitals of transition metals. The localized bosons, the localized fermions, the delocalized bosons and delocalized fermions may be driven by surrounding thermal perturbations, gravity, electric, magnetic and QF driving forces. The relative stabilities and interactions for stable ferromagnetism, paramagnetism and diamagnetism are by Little's Rules as diamagnetism in such systems may obey Little's Rules 1 and 3 but ferromagnetism in such systems may obey Little's Rules 1 and 2. The diamagnetism may be by the bosons localized as in diamond, but in graphene the bosons are delocalized bosons. Such happens in graphene to cause electronic spin paired fermions in the delocalized electrons. These unpaired delocalized fermions cause the delocalize to rehybridize to localize as sp^2 to sp^3. The theory of RBL determines some transient spin induced finer azimuthal fractional continua quanta numbers for transition stages during transport and transmutations. And likewise with the nuclei, as the nuclei interact with the electrons and bosons in graphene the nuclear spins and orbitals angular momenta in nuclei alter the electronic delocalization for singlet to triplet on other spins. The fractional fissing and fusion of nuclei seep QF into electronic shells as by RBL so as to transiently create ultrafine continua of azimuthals for mixing, coupling, transporting, transforming and transmuting electrons for novel superconduction, chemistry and catalysis/enzymatics. Vice versa e^- e^- rehybridization and spin polarization can alter the couple nuclear orbital momenta by RBL Effect. The localize bosons verses delocalized bosons allow different coupling of nuclei and their NMMs. The thermodynamics may favor one or the other but the change from one to other involves kinetics and dynamics by RB Little's Effect. The e^- spins and nuclear spins via delocalized or d (azimuthal) π e^- e^- can couple to alter the symmetry and motion from local to nonlocal and vice versa.

Pure metal clusters and nanoparticles may also couple nuclear spins. But in molecular compounds, the coupling may not be possible via more localized molecular orbitals. But the delocalized molecular orbitals via π bonds may afford the delocalized bonding over many C, N, O bonds as previously proposed in theory (RBL ferrochemistry) so that the π electrons can couple spins and orbitals of electrons (e^- e^- Lewis pairs and radicals) and the π electrons can also couple the nuclear spins and nuclear angular momenta over many atoms in nanodomaind. Thereby the pyrimidine aromatic more exchange the nonprimordials. The purine less exchanges the nonprimoridal and the nucleotides, oligonucleotides and nucleic acids couple their nuclear magnetic moments with nuclear magnetic moments (NMMs) in surrounding nanowater ad accumulated NH_3 to accelerate primordials replacement by nonprimordials by different NMMs. So the delocalized e^- pull in NMMs. These molecular orbitals can couple spins on centers. So also spins can alter orbitals and the orbitals can alter spins centers, spin ⋯ spin orbital interactions not only alter orbits but flicker spins, transition states break bonds, spins flip and intervening metal orbitals and/or orbits couple spins to other regions when orbits change and spin pairs to bond. By this mechanism [1-3], the spins not only interact with the orbits, but the spins transform by fractional, reversible fissing and fusing and fissed spins fractionally, reversibly fiss and fuse to orbits and vice versa the orbits fuse to spins. So also NMM via e^- e^- orbitals can couple nuclei spins and change the orbitals. Nuclear spin momenta and orbital momenta can alter the e^- e^- orbital. And e^- e^- orbitals can alter nuclear angular momenta. RBL here notes NMM are variable as not only during chemical reactions but also during chemical reactions, enzymatics, vibrations, optics and e^- e^- transport and transmutations. The nuclei are perturbed so relative motions of nucleons change and the nuclei swell and compress for fractional, reversible fissing fusing to alter and couple to surrounding e^- e^- lattice. Thereby momentary changes in

NMMs occur. Therefore, it is this reason of the aromatic rings in purines and pyrimindines that the nucleotides in DNA more readily exchange isotopes nuclear spins and NMMs relative to other biomolecules.

It may be possible by such unique ability of DNA and RNA via their nanodomains of graphene, diamond alkyl aromatic or diamagnetic paramagnetic ferromagnetis; that the resulting DNA and RNA catalyze isotopic exchange in proteins. So that during DNA, RNA and protein binding, interactions, charge exchange and enzymatics, isotopes may be exchanged. By this theory, the ^{14}N and ^{1}H ad ^{31}P via fractionl reversible fissing and fusing cause the denatured protein to renature and the DNA to unnature and renature during reproduction, and RNA to denature and nature. So in general, the NMMs in the proteins and nucleic acids cause orbitals tochange so the proteins and nucleic acids denature and renature. So the proteins and nucleic acids renautre so rapidly due to huge fields caused by the nonzero NMMs of ^{14}N and ^{31}P within them and $^{1}H_2O$) in surrounding nanowater. Thereby from this theory the RNA may catalyze the nonprimordials replacement in amino acids as the RNA translates proteins.

The isotopic exchange is selective in uphill anabolism in animals and humans nonadiabatic as it is selective in uphill anabolism in plants adiabatic. It is during uphill processes of DNA replication, RNA transcription and protein translation that the proteins are altered. Virus RNA can modify so the RNA produces unhealthy protein. It is that the side change sugar and side change phosphate couple energy into the nucleoside to break bonds. It is that the side phosphate and side sugar help the NMM replacements. This occurs by the ferrochemistry of the bond rearrangements of the sugar releases energy reversibly as accumulation and absorbing into the phosphates by NMM and other oligonucleotides by ^{14}N and ^{1}H so as to give energy to promote so activated states near or far and then as the transition state relaxes to products, the phosphate collects the energy and restores it back to the sugar unit. There is chemical energy in the sugar and the phosphate can store chemical energy and the nucleosides can delocalize energy). It is on this basis that the virus can kill cancer. But the downhill catabolism is less affected by isotopic replacements as the electronic energy can drive and dictate the dynamics. But in glycolysis the down hill is accelerated by the isotopic replacements as downhill glycolysis is reverse of photosynthesis in plants so the downhill accelerated by nonprimordial ^{13}C just as uphill is slowed by ^{13}C. It is logical that exothermic downhill would less discriminate nonprimordial / primordial replacements. But in Kreb cycle, higher electric and magnetic fields in the substrates and the enzymes cause stronger effects on the downhill processes as the high fields can couple more strongly to the NMMs for the nonadiabatic Kreb so that Kreb cycle becomes adiabatic as the heat is organized in the high fields. This is the reason the Kreb cycle is more sensitive to nonprimordial isotopes relative to the glycolysis process.

Thereby this theory determines that the DNA may accumulate the nonprimordials from the proteins and sugars combusting and then the DNA may incorporate the nonprimordial isotopes into the proteins during translation, replication and transcription for the nonessential proteins. The eating of nonprimordials in nonessential proteins can cause the animals to accumulate nonprimordials; first in nucleic acids and then in proteins via nonessential proteins. But as the organism eats other animals and obtain essential amino acids, then the essential has more nonprimordials so the nonprimordials with the eaten essential amino acids connect to alter catabolism in cancerous ways in the essential proteins. The ^{13}C in lysine is crucial for animals and humans to develop cancer. So diet accumulate ^{13}C in DNA and then diet of essential amino acids accumulate nonprimordials in enzymes. When the two conditions optimize then cells

become cancerous. Cancer cells may accumulate nonprimordial isotopes until they die and then the innards with the nonprimordials of the dead cancer cells are eaten by normal cells and the the surrounding normal cells transform to cancer cells. This may be a basis for metathesis. RNA with the nonprimordial isotopes can synthesize nonprimordial amino acids and construct nonprimordial proteins. These with lysine can cause cancer.

More general discussion of NMM couple by MO and AO causing nucleophile substitution and NMM substitution replacement. Nucleophile driven by nucleophile but the spin driven by magnetism as the null spins diamagnetically pushed out MO and the nonzero spin pulled in or push out MOs and AOs. But what about the + NMM and – NMM. The + NMM pull in + NMM and push out – NMM in MOs and AOs. But in nucleus and continua + NMM push out + NMM and pull in – NMM. So thereby ^{13}C is pulled into other ^{13}C via π bonds as the many ^{13}C nuclei create self conforming MOs. But ^{17}O disrupts MOs of ^{13}C to activate bond rearrangements. As ^{17}O pulls ^{13}C nuclei together and yet push their QF apart for driving bond activations for bond rearrangements. This is powerful as by this RBL introduces new type interaction as two or more objects interact in counter ways on different states on L frame they attract but smaller frame they repel and/or on L frame they repel and on smaller RS frame they attract. Something on inside binds whole and whole repels. Or something on inside repels as whole binds! The author published this in 2007 as p^+ and nuclei bond $e^- e^-$ of covalence by fissing of p^+ and nuclei to create QF to bind the $e^- e^-$ pairs. So it is that the ^{17}O can attack as it breaks up many + ··· + ··· + ·· · + ··· + NMMs. This may explain Ag nanoparticle atomizes due to the interactions of all its negative NMMs. So now this ^{33}S, ^{2}D, and it ^{14}N help pull in ^{13}C and ^{17}O lowers E_{act} for such isotopic replacements inside organisms for replacements of primordial isotopes by nonprimordials. But what about ^{15}N it lowers E_{act} at higher temp but at low temp it pushes ^{13}C away.

The observed higher deuteration, methylation, amination, and hydroxylation and enrichment with $^{13}CH_3$ $^{15}NH_2$ and ^{17}OH in cancer cells is consistent with nonrandom clustering and higher density methylation in DNA of cancer cells [10]. Moreover, in this work in addition to explaining faster methylation by $^{13}CH_3$ to cause cancer, the faster methylation is explained in details by the accelerated ^{17}OH hydroxylation and many body $^{15}NH_2$ aminations causing the transformations of C → T → U and A → G for chemically altering DNA and RNA for new chemical paths of DNA and RNA mutations for explaining cancer. $^{13}CH_3$ may be causing more methylation of DNA in cancer cells as in the this theory [1-3], $^{13}CH_3$ is a stronger nucleophile than $^{12}CH_3$. This isotopic effects of the nucleic acids can explain recent mysteries. Positive NMM of $^{13}CH_3$ relative to null (0) NMM of $^{12}CH_3$ by the RBL theory [1-3] causes the $e^- e^-$ to be pulled closer to nucleus of $^{13}CH_3$ relative to $^{12}CH_3$ nuclei. $^{24}Mg^{2+}$ should interact less strongly with $^{13}CH_3$ (and make $^{13}CH_3$ a stronger nucleophile) relative to $^{25}Mg^{2+}$ for more altering the bonding of $^{13}CH_3$ by $^{25}Mg^{2+}$ in cancer cells relative to the weaker effect of $^{25}Mg^{2+}$ and $^{24}Mg^{2+}$ interacting with $^{12}CH_3$ in the normal cells to explain the observed selective killing of cancer cells by $^{25}Mg^{2+}$ [11,12]. This accumulated $^{13}CH_3$ in RNA and DNA then alters RNA and the RNA alters translated proteins for splicing phenomena mechanisms. [13]. This theory of RBL determines the chemically altered RNA by nonprimordial isotopes causes the splicing of proteins that is hallmark for cancer genesis and habitats. The transmuting of $^{12}CH_3$ to $^{13}CH_3$ of the space twin relative to the earth bound twin would explain the observed elongation of the telomeres of the space twin as by $^{13}CH_3$ methylation of his telomeres and stronger binding of his telomeres by

$^{25}Mg^{2+}$ for elongation rather than shortening of the space orbiting telomeres [14]. The stronger binding of the telomeres containing $^{13}CH_3$ may less frazzle the ends for continued elongations.

Methylation and Altered Binding and Transcriptions and Translations

So after reasoning / explaining how the nucleotides are isotopically mutated and some consequences, here consider how altered genes malfunction. So these alterations of nucleotides alter the sequence, constitution, connectivity and stereochemistry of isomers so what are consequences. Based on this model [1-3], RBL the methylation of the cytosine not only causes the cytosine not to bind guanosine; But moreover the methyl-cytosine may be misread as thymine and vice versa the thymine may be misread as methyl-cytosine. These are some of the consequences of changing the isotopes in nucleotides. The bases pairs are GC and AT pairs. Also the functionalization / defunctionalization can alter the DNA and RNA sequence by transformation C → T → U and A → G so as to alter DNA and RNA and alter proteins for changing RNA, DNA and proteins content in cells to damage cells. So C may be methylated to similar to A and OH^- may replace NH_2^- for C → U. So methylation of C and deaminatation forms C and U for possible misreading of protein; so for example UUC (Phe) → UUU (Phe), CUU (Leu), CUC (Leu), CUA(Leu), and/or CUG (Leu) → UUU (Phe), UUC (Phe), UUA, UUG (Leu). So in some cases U and C can interchange without misreading protein, but in other cases such changes cause misreading and mutations. Likewise mutations as C → T and G → A can cause splicing of proteins as by the change in translations of amino acids. Thereby chemically interchanges in nucleoside sequences change the selection of peptides to alter proteins. Stops in nucleic acids do not involve C: UAA, UAG, UGA! The creation of organisms may have intentional avoided C in stops as the mutations of C would affect stops. The G is in stops and mutations of G may cause stops in nucleic acids not to stop for a basis or cancer. Thereby DNA is altered by nonprimoridals substituting for primordials.

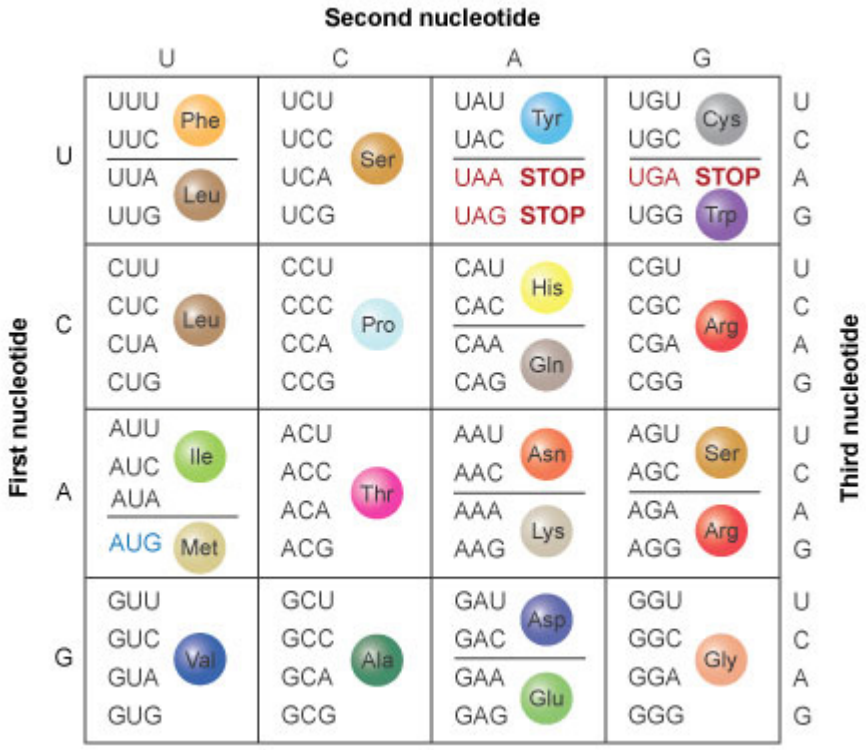


Figure 4 – Nucleotide Codons for Amino Acids (reference 9)

The $^{13}CH_3$ binding may alter interactions and dynamics due to its different NMM. Although thymine already has a $^{12}CH_3$, by changing the $^{12}CH_3$ to $^{13}CH_3$, the properties of the thymine change so that the $^{13}CH_3$ may appear like H on the phenyl ring of thymine; so thymine appears to behave like uracil for altered replications, transcriptions and translations. Such $^{13}CH_3$ and its + NMM may appear as ^{1}H so the thymine in nucleus of cells appears like uracyl (U) with alteration of the DNA replication and transcription inside the nucleus. So it is that U can exist in the nucleus but thymine (T) exists only in the nucleus. But if $^{13}CH_3$ replaces $^{12}CH_3$ on thymine then thymine appearing as uranyl can exist in cytoplasma also to alter biochemistry in cytoplasma. And uranyl can methylate to enter nucleus. This means that uracyl in the nucleus can be template as thymine (as $^{13}CH_3$ in thymine causes it to appear as uracyl). Or the other possibility is that the $^{13}CH_3$ in thymine causes it to not be recognized. A third possibility is that the $^{13}CH_3$ causes the similar nuclear behavior as H so the thymine may behave as cytosine assuming the =O (OH) and NH_2 manifest similar basic interactions. So these are possible consequences of $^{13}CH_3$ on the thymine.

C and G Depletions and Cancer Defient Amino Acids in Cancer Cells

In addition to these nonprimordial induced misreads of nucleic acids and proteins and nonprimordial induced interchemical transformations of C → T → U and G → A, this mechanism further determines the consequent deficiency in C and G due to the + NMM of $^{13}CH_3$ and – NMM of ^{17}O and ^{15}N by difficult hydroxylation of A → G due to ^{17}O and difficult demethylation of T → C. The consequent dynamics cause deficiencies in C and G in cancer cells. The efficient C and G on basis of this theory causes deficient proteins translated by C and G. See Figure 4. For instance, G and C strongly translate Gly (GGU, GGC, GGA, GGG). {Note Gly and Pro are extremely important for alpha helices breakers. Gly and Pro start secondary structures of beta turns. Beta turns are turns in primary structure. Pro has odd, cyclic structure in peptide bond and this cause bending of peptide. Gly has small size and can have large conformational changes due to lack of steric hindrance by Gly due to its small size. Bending breaks alpha helicies.}

The Arg and Try and Ala also have strong translations by G and C and shortages of C and G in cancer cells is here reasoned to cause cancer habitat and transform normal cells to cancer cells: Arg* (CGU, CGC, CGA,CGG); Trp* (UGA, UGG), Pro (CCU, CCC, CCA, CCG); Ala (GCU, GCC, GCA, GCG). Quite interesting Arg and Trp are also essential amino acids; and this enforces this theory of the cancer genesis due to shortages of G and C and the inability to synthesize the Arg and Trp translated by G and C. But then other amino acids are marginally affected by deficient G and C: Leu (CUU, CUC, CUA, CUG); Val* (GUU, GUC, GUA, GUG); Ser (UCU, UCC, UCA, UCG); Thr (ACU, ACC, ACA, ACG); Asp (GAU, GAC); Glu (GAA, GAG); Cys (UGU, UGG); His (CAU, CAC); Gln (CAA, CAG), * means the amino acids is an essentials amino acids. But then the following are amino acids that are not strongly affected by shortages in G and C content in cancer cells: Phen* (UUU, UCC); Ile* (AUU, AUC, AUA); Met* (AUG); Try (UAU, UAC); Asn (AAU, AAC); Lys* (AAA, AAG). So on basis of such deficient G and C causing deficient templating of amino acids to form proteins the following consequences are reasoned. The predicted deficient Gly, Arg*, Trp*, Ala, and Pro correlates with recent analyses of microenvironment of tumors. Van der Heiden [18] recently observed microenvironments of tumors are deplted in Trp* and Arg* and Cys. It is also important to note that Try* and Arg* are essential amino acids, as cells cannot synthesize the essential amino acids. But Gly and Glutamate where found by van der Heiden [18] to be abundant in cancer microenvironment. But Gly can be synthesized. And Glutamate is nonessential and can be synthesized so this observed abundance of Gly and Glu are consistent with this theory of cancer [1-3]. The stops are encoded by UAA and UAG, therefore excess A and A may cause high densities of stops. The telomerase has its own RNA (3' – CCCAAUCCC 5'). So telomerase cannot elongate and this is habitual of cancer cells as $^{13}CH_3$ methylates then the C cannot elongate the telomere. It is important that the telomerase involves a lot of C and by this theory the deficient C may affect telomerase expression for causing cancer as the lack of C causes lack of telomerase and the lack of elongating telomeres which is hallmark of cancer.

After discovery of this new DNA and RNA and protein chemistry by Little Effect via NMMs of nonprimordials, this work considers plants oligomers and possible such chemical interactions of plant oligomers with human oligomers. This work determined that just as the RNA and DNA and proteins can undergo intrinsic internal accelerated methylation, deamination, amination and hydroxylation and deuteration of nonprimordials relative to primordials; then also foods having similar oligomeric structures can also exchange primordials and nonprimordials via functionalizations and defunctionalizations between dietary oligomers and nucletides in the host.

(What happens to DNA as animals products are consumed? Plants products have less nonprimordial ^{13}C, ^{15}N, ^{17}O and ^{2}D in the nucleus acids, but how does such high nonprimimordials compare to animal DNA? Scientists find link between plant telomere and human telomere so plants live longer as by their lower nonprimordials relative to animals and humans. Is this due to diet of trees? Yes with less motion and less energy demands the tree does not break down ^{13}C compounds and then construct its as much in their DNA for high nonprimordial contents; so its DNA less mutates so trees live longer. The theory here determines that penalty of motion as by needed catabolic metabolism is breaking nonprimordial molecules and consequent uptake nonadiabatically of nonprimordials into DNA with mutations. Trees and plant use sunlight and operate adiabatically so less nonprimordials are taken up. Heat may help animals and plants pull in nonprimordials, plants operate cooler and pull in less nonprimordials. This explains how animals mutate DNA and develop cancer. This leads to cancer in humans and animals. In this theory RBL tried to correlate cancer to motion and diet on this basis of nonadiabatic catabolism and uptake of nonprimordials by animals and humans. So eating cancerous DNA may also cause cancer to be transferred to DNA and RNA. So cancer can be transferred by large transfer of cancerous tissues. Rats are implanted with cancer tissue to induce cancer. In this work, it is reasoned that cancer cells of different types may kill each other. Injecting different types of cancer into a tumor may kill the tumor as the DNA and RNA of the two cancers differ. It may be possible to kill tumors and cut it out by surgery.) The nonprimordials are determined to accelerate such new chemistry by differences in kinetic and thermodynamics of functionalization and defunctionalization. Thereby based on this new chemistry discovered for nucleotides based on NMMs and magnetics driving substitution of NMMs and a mechanism by which NMM substitution can couple and mix with nucleophilic substitution energies. So by replacing new oligomers with NMMs in the DNA of normal cells is disrupted to cause cancer. But in this work, it is further determined that just as the isotopic accumulation can transform normal cells to cancer cells, excessive nonprimordials can accumulate to kill cancer cells. Grape seeds may kill cancer but they may cause cancer as in this work, the grape seeds have oligomers of proanthocyanidins, which are in this work determined to have excess $^{13}CH_3$, ^{17}OH, $^{15}NH_2$, and/ or ^{2}D that can replace ^{1}H, $^{12}CH_3$, $^{14}NH_2$, and/or ^{16}OH in cancer to oversaturate the cancer DNA with nonprimordials to kill the cancer.

It has already been published in 2007 [1] that the + NMM and nuclei of ^{14}N, ^{16}O and ^{12}C via the proton (^{1}H) can magnetically couple for novel many bond nuclear magnetic moments (NMMs) and nuclear orbitals to cyclically move, transform and transmute for normal anabolism and catabolism. Thereby it is determined that without such effects of the proton nucleus, life cannot exist and thereby disease may be caused by altering this natural rhythm [1]. For instance, consider intrinsically the $^{31}PO_3$ gives P center strong ability to attack ADP and AMP so if change ^{12}C to ^{13}C then ^{13}C makes $^{13}CH_3$ a stronger nucleophile; and if change ^{16}OH to ^{17}OH then ^{17}OH is a weaker nucleophile and if change $^{14}NH_2$ to $^{15}NH_2$ then $^{15}NH_2$ a weaker nucleophile. So just as there is intrinsic NMM chemistry of $^{31}PO_3$ then there is new chemistry by NMMs in ^{13}C, ^{15}N and ^{17}O; ^{13}C attacks more than ^{12}C; ^{17}OH attacks less than ^{16}O. ^{17}OH attacks differently than ^{16}OH attacks;; so it is not that ^{17}O does not attack, but ^{17}O attacks differently than ^{16}OH. $^{13}CH_3$ attacks more with ^{17}OH than with ^{16}OH. ^{17}OH attacks ^{15}NH more. It is that + NMM attacks + NMM more in L Frame but less in L Continua and Nuclear Frame (NS Frame). {Where interior quarks are in QS Frame; quarks are inside hadrons in RS Frame; hadrons are inside nuclei in NS Frame. The NS frame couples continuously to interior LS continua of the electronic lattice outside the nuclei. Electronic orbitals exist in L frame (discontinua); Electrons manifest a continua about

them for LS Frame and a discontinua within the electron for E Frame. The LS frame of the electron can couple to the outer L continua of the electronic lattice to mix with the inner L continua of denser NS frame fields and such mixing of LS Frame and NF with diminution stretch and transform and combine with other outer L Continua of other atoms, leptons and hadrons to manifest the C Frame (macroscopic frame) of magnetic fields, gravity, electric and thermal fields and spaces}. It is that + NMM attacks – NMM less in L Frame and more in NS Frame. So thereby pressure effects manifest as high pressures push then the + NMMs into – NMMs so the L Frame – NMM repel + NMM → NS Frame + NMM attracting – NMM at higher pressures. This is consistent with RBL theory of high temperature superconductivity and why high pressures cause superconductivity. So now also with cancer as the cancer involves changing pressure the cancer may not metabolize as well; and by RBL theory this explains the changes in cancer as the host moves from surface of earth to outer space to kill the cancer due to changes in gravity and pressure. By this theory, the primordial isotopes of ^{1}H, ^{12}C, ^{14}N, ^{16}O, ^{24}Mg and ^{32}S manifest in normal cells at earth surface and atmospheric pressure with all positive NMMs so all positive NMM attract in L frame. But as cancer forms by ^{2}D, ^{13}C, ^{15}N, ^{17}O, ^{25}Mg and ^{33} S on the the earth's surface then the nuclei have + and – NMMs and + and – NMM repel in L frame. But if the normal cells and cancer cells are accelerated into outer space then the lower pressure as gravity becomes zero and the lost gravity in outer space less pushes + ··· + NMMs of normal cells to NS Frame for repulsive so normal cells are less affected by earth's gravity (this can also be a basis for new magnetic sensing of earth's magnetic field by normal cells.) So now cancer cells on the other hand have + and – NMMs and the loss pressure increases there + and – repulsions in L Frame to alter biochemical dynamics more in cancer relative to normal cells and may cause the cancer tissue to bind on larger scales for possibly killing the cancer. But such altered L Frames alter the glycolysis to kill cancer due to zero gravity. It is that ^{17}O helps ^{13}C in C Frame magnetically but then ^{17}O pushes ^{13}C away in L Frame QF. But then under compression ^{17}O pulls ^{13}C to it in inner L Continua and in NS Frame. This is how the ^{13}C and ^{17}O interact differently in complex ways on different scales to cause accelerated mutual replacements and substitutions for ^{16}O and ^{12}C in living organisms. So the interactions are contravariant on different scales as attack on nuclear scale (NS Frame) and repel on QF scale (L Frames) and attack on magnetic frame of C Frame. It is that as something pulls another to it, but simultaneously can push it away simultaneously; thereby this is new dynamics that by RBL explains transformations of it and transmutations of it for new mechanics as introduced here by RBL. And this is how transport goes to transform and to transmute and vice versa. As transport is by push but if push so hard then it pulls as it pushes to stretch and pull it towards to transform it and to transmute it as by this theory of RBL! So ^{17}O transmutes ^{13}C where as ^{14}N NMM pushes ^{13}C away. ^{17}O pulls ^{13}C to its nucleus and pulls ^{13}C atomic orbitals apart and magnetically binds ^{13}C as it stretches its orbitals! This is a new type physics of chemistry as by nuclear magnetic moments and nuclear spins. Simultaneous nuclear, chemical, and physical transformational phenomena are determined by the author to occur! So it is as the ^{17}O internally binds it; it also stretches it and holds it globally! But ^{14}N internally pushes ^{13}C away as it binds it in QF and magnetically globally repels it!

So after considering/discussing the peaks and the nucleosides and how cancer DNA therefore has different peaks relative to DNA of normal cells and the different peaks are due to nonprimordials, next oligomeric in grape seeds [15] and the seeds are considered and compared to these nucleotides in normal an cancer cells. The novel chemical alterations of DNA and RNA by grape seeds oligomers are considered. The grape seeds are the cellular nucleic of the fruits

with reproductive ability. So the biochemistry and biomolecules of grape seeds reproduction couples to biomolecules of human reproduction and malignant reproductions as by cancer so thereby the grape seed may couple to cell to cause cancer and/or to kill the cancer. The plants are observed to accumulate ^{2}D, ^{13}C, ^{15}N and ^{17}O in their proanthocyanidins in grapes with water deficit have more ^{17}O and the ^{17}O pulls in more ^{13}C into seed [14]. This is in the literature [14]. It has been observed that the draught and ^{13}C and ^{17}O in the seeds make the seeds more anti cancerous [15]. What is it about grapes that they incorporate ^{13}C ^{17}O among the plant kingdom? In this theory, it is determined that the chemistry of ^{13}C and ^{18}O, ^{17}O cause greater incorporation of nonprimoridals in grape seed proanthocyanidins as by aromatic background network of the oligomers. The corn may have similar background oligomers to help it pull in more ^{13}C in C4 process relative to C3 process.

Discuss Why the Grape Seeds Affect Cancer

In consistency of this reasoned aromatic alkyl background network accelerating nonprimordial uptake by coupling NMMs as by this theory, researchers recently report larger plant oligomers have greater anticancer effects [7,8,15]. The larger proanthocyanidins are more anticancerous as they have more nonprimordials isotopes and they pull in more nonprimordials or release more nonprimordials by extended C-C bonds and π bonds. Also consider that the enzymes of Kreb cycle may be able to pull in more nonprimordials relative to enzymes of glycolysis due to the high field substrates of Kreb cycle. Both such network of changing covalence in Kreb cycle and changing covalence of glycolysis process and the oligomerics of proanthocyanidins manifest changing covalence in extended arrays of sp^2 and sp^3 covalence with intrinsic magnetics of the changing covalence and with embedded p^+ and NMM of other nuclei so that by Ferrochemistry the nuclei revolve to orbitals as by fissing their NMM so as to couple the covalence and alter the many covalence for breaking covalence by the many NMMs and pulling in nuclei and pushing out nuclei and rebonding covalence to new nuclei so as to explain the isotopic replacement by the covalent lattice with embedded NMMs in accelerated many relative to null NMM as by RBL. It is noticed that the greater enrichment of nonprimordial isotopes in the heavier isotopes correlate to the anti cancer.

On basis of this a new idea is presented. It may be that the different ferrochemistry of glycolysis, Kreb, replication, transcription, translation can be reasoned by functional groups of amino acids as the alkyl + phenyl functional may in protein push together to induce greater nonprimordial uptake. Also the ^{17}OH and ^{15}NH regions of functionals in proteins more push together to lower E_{act} for such ^{13}C substitutions or to accelerate incorporation of ^{17}O and ^{15}N. So if RBL look at enzymes of glycolysis, RBL may find fewer Leu and Trp than in Kreb cycle. Kreb may have more Trp and Leu so it would more incorporate ^{13}C relative to glycolysis.

Proanthocyanidins are observed in grape seeds and cranberries [13]. The greater amounts of proanthocyanidins in grape seeds and cranberries are revealed in mass spectrometer as isotopic cluster are observed in Figure 5. The nature of the interflavan bonds (D2 amu) [M+Na}+ represented by observed masses. The PACs from grape seeds contain B type (m/z 1465) bonds. Masses represent variation in the nature of interflavan bonds (D2 amu) [M + Na] +. It is noticed that from the mass analyses, that the grape seeds explicitly show huge enrichments of either ^{2}D, ^{13}C, ^{15}N and/or ^{17}O in the mass spectra, but the authors of these prior data [13] do not correlate such properties of isotopes in the proanthocyanidins to anti-cancer.

Isotopes of predicted compounds are observed in the spectra with characteristic masses (m/z). For instance the predicted monoisotopes for PAC of 5 DP with 4 B type interflavan bands is 1465 m/z, which is observed to have primordials of ^{12}C, ^{14}N and ^{1}H. See Figure 6. But mass of 1466 m/z is observed of similar intensity as the 1465 m/z for similar relative concentrations so that the 1466 m/z has possible contributions from possibly one ^{13}C, one ^{2}H or one ^{17}O. The mass at 1467 m/z may have two of these nonprimordials two ^{2}D, two ^{13}C or two ^{17}O. The similar intensities of 1465, 1466 and 1467 m/z determine similar relative abundances and thereby isotopic enrichment of nonprimordials in the PACs. But in this work, the anticancer activity of proanthocyanidins is correlated with there enrichment with nonprimordial isotopes of ^{2}D, ^{13}C, ^{17}O and ^{15}N. Furthermore, the proanthocyanidins may be anti cancer as by the similar chemical structures of the tannins and polyphenols to the nucleosides and the possible exchange of the nonprimordial isotopes between the nucleosides of RNA and DNA and the proanthocyanidins for altering the translation of proteins in cytoplasma and the synthesis of DNA in nucleus. In this work it is determined that the nonprimordial isotopes couple more strongly to the cancer DNA and RNA relative to the RNA and DNA of normal cells, because by this theory and data the cancer DNA and RNA are isotopically different from the normal cells RNA and DNA. The stronger binding of tannin to cancer DNA is due to similar clumping of nonprimordials. The nonprimordials in grape oligomers may also chemically alter the DNA in cancer so as to alter cancer replication. Thereby the grape seed provide the epigenetics to alter cancer DNA selectively so the seeds are anti cancerous and this is the first molecular basis for anticancer of grape seeds. This is consistent with prior theory for also treating cancer by the prior theory [1-3] as by the prior theory, it was proposed to use of nonprimordial enriched foods to selectively target the cancer. So the prior theory [1-3] looks at the DNA in the human and the cancer and finds the nonprimordials, and the prior researchers find nonprimordials in the seeds of grapes. So in this work, the nonprimordials in cancer and in grapes are correlated for anticancer activity of grape seeds. And the grape nonprimordials disrupt the cancer nonprimordials.

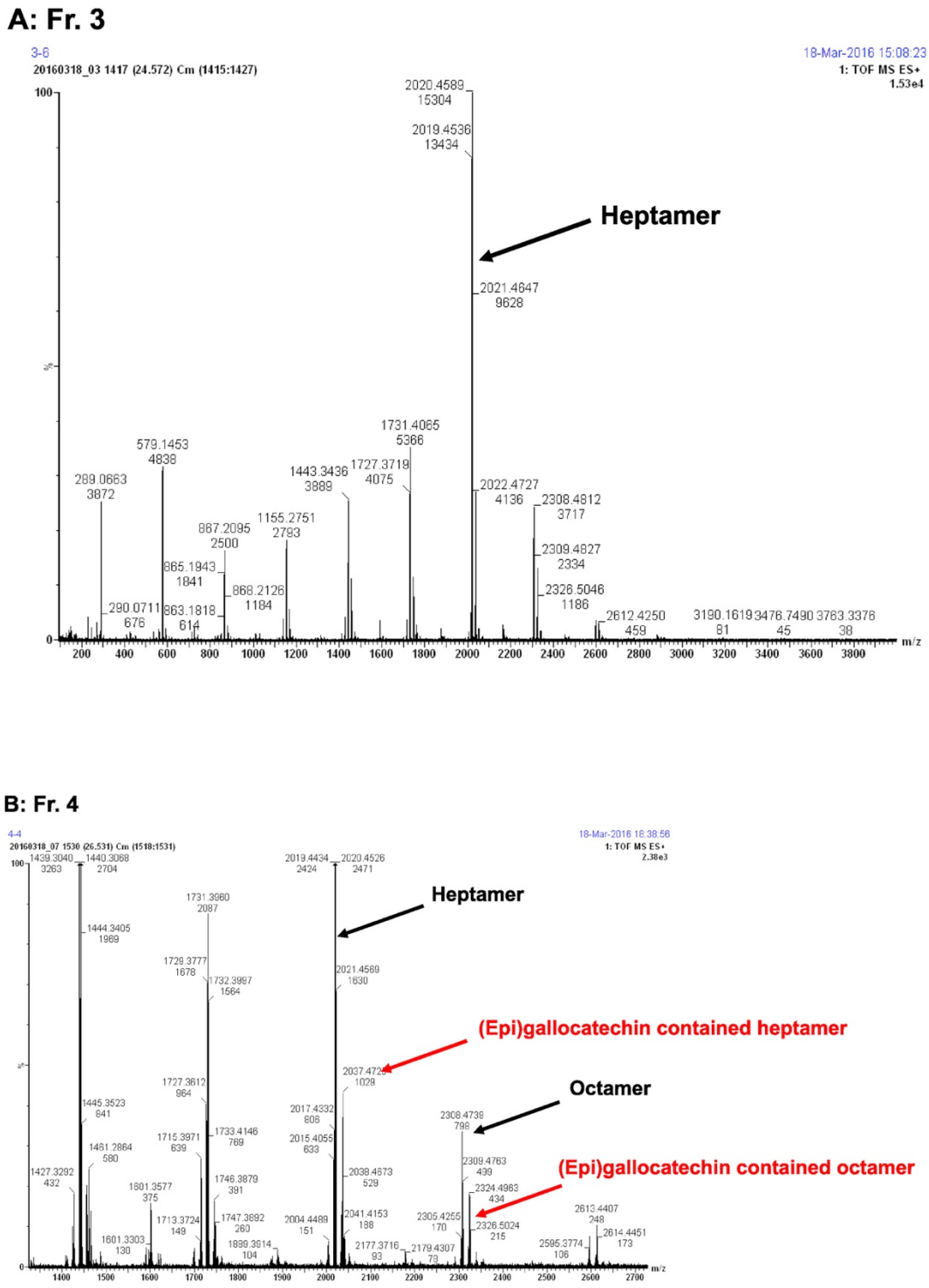


Figure 5 – Oligomeric Proanthocyanidins from Adzuki Beans With Those Larger than Tetramers Showing AntiCancer Activities (reference 8)

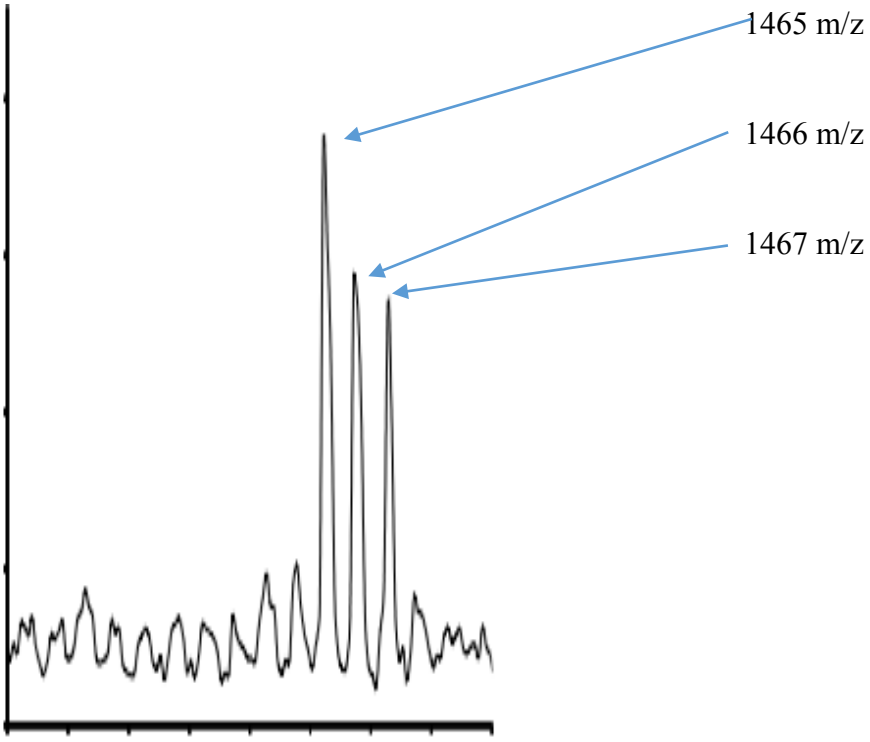


Figure 6 - **Grape Seed Proanthocyanidins (PAC) Isotopic Reveal Isotopic Enrichments (reference 14)**

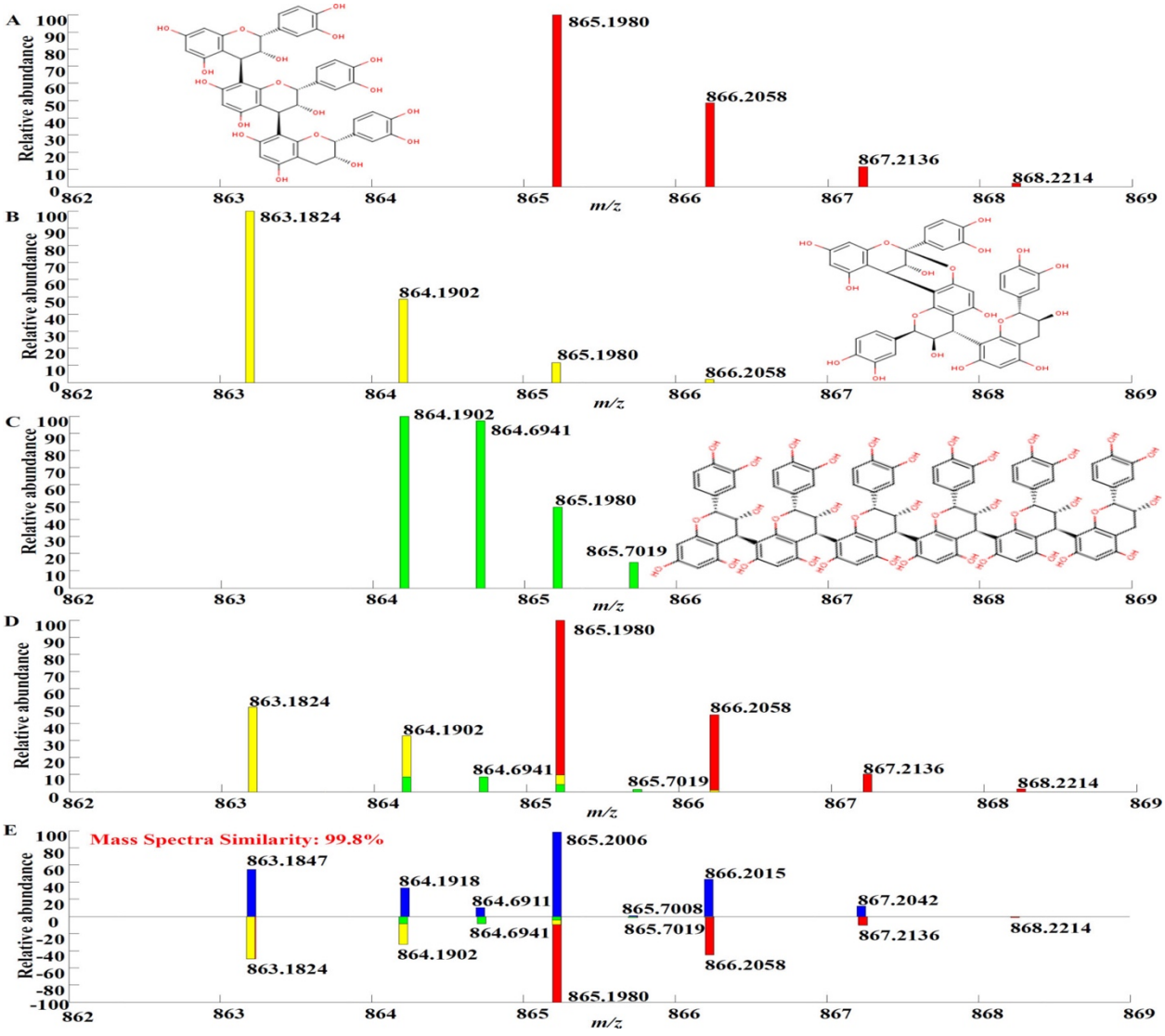


Figure 7 – Structures and Masses of Nonprimordial Isotopes in Plant Proanthocyanindins (Reference 7)

Discussion of Possible Cancer Cure Relative to DNA, RNA, Protein and Proanthocyanidins

Previously the authors [1-3] proposed a treatment and possible cure for cancer by the patient eating normal food but with added nonprimordial isotopes. Eating grape seeds is an application of this prior proposal by the author as grape seeds are in this work determined enriched in nonprimordial isotope. Therefore eating seeds of grapes and other foods enriched with anthodyanidins are an examples of such eating foods isotopically enriched with nonprimordials. The author [1-3] further proposed that the patients' tumor should be irradiated with radio frequency of specific wavelengths so as to tune into the nonprimordial isotopes within the cancer cells with few effects on the normal isotopes and normal cells. The author proposed that radiofrequency rotates the nuclei and fractionally fiss to alter wavefunctions about to alter enzymatic activity within the glycolysis process to overheat and/or starve the cancer cells with few effects on normal cells. The author [1-3] proposed that the patient's tumor irradiated with specific X-rays of wavelengths tuned to excite in the near edge only nonprimordial isotopes for further inducing superluminous rotations of their nuclei to alter fields and quantum fields about to demagnetize and deactivate enzymes containing nonprimordial isotopes in cancer cells with no effect on normal cells. The patient's tumor irradiated with thermal neutrons of specific kinetic energies to absorb under simultaneous conditions of RF and X-ray irradiation so as to enhance the selective absorbance of the neutrons by the nonprimordial isotopes to transmute the ^{13}C to ^{14}N, ^{15}N to ^{16}O, ^{17}O to ^{19}F for total inactivation of enzymes of glycolysis only in cancer cells with no affect on normal cells. In considering the merit of the authors [1-3] proposed cancer cure, it is important to note that the following Radiofrequency is almost innocuous but affects biomolecules in this newly discovered way. The radiowaves can be selected to only stop glycolysis in cancer with innocuous affects on normal cells. Soft X-rays can of certain wavelength be innocuous to more biomolecules and tissue. X-rays can be tuned by specific wavelengths to excite only nonprimordial isotopes to selectively kill cancer cells with few effects on primordial isotopes and normal cells. Neutrons have no charge. They pass through most elements not absorbed. In this invention tune the neutrons by slowing and rotate the nonprimordial isotopes by modulated RF and specific wavelength of X-rays to selectively increase absorption cross-section of the nonprimordial for neutron absorption and transmutation. All three (radiowaves, X-rays and neutrons) can penetrate the whole human body.

Conclusion

The analysis of DNA by MALDI mass spectroscopy led to the observations of different isotopic enrichments of nucleotides of guanosine (G), adenosine (A), cytidine (C), thymidine (T) and uridine (U). Such isotopic differences were further determined due to addition and removal of clumped isotopic enrichments of functional groups of ^{2}D, ^{13}C, ^{15}N, ^{17}O, and ^{25}Mg associated with H, CH_3, NH_2, and OH. The DNA cancer cells tend to show enrichment with clustered kernels of $^{13}CH_3$ relative to DNA of normal cells. The surrounding nanowater and proteins were reasoned to accumulate ^{17}O for hydrolysis to place ^{17}O onto G, T, and C. The ^{17}O was reasoned to lower activation for $^{13}CH_3$ and $^{15}NH_2$ functionalization of nucleotides. The accumulation of ammonia about cancer micro-environment was determined to facilitate such $^{15}NH_2$ functionalizations. These novel nonprimordial functionalizations of purines and pyrimidines of nucleotides are consistent with observed fragmentations of DNA of cancer and normal cells.

On the basis of such totally new chemical dynamics as driven by nuclear magnetic moments (NMMs) of nonprimordial isotopes of ^{2}D, ^{13}C, ^{15}N, ^{17}O, and ^{25}Mg of different NMMs cause ease of ^{17}OH functionalization and defunctionalization with ^{17}OH catalyzing $^{15}NH_2$ and $^{13}CH_3$ functionalization. The resulting $^{13}CH_3$ functionalization causes difficult $^{13}CH_3$ defuntionalization for accumulation of U as T → T and → C but the $^{13}CH_3$ defunctionalization of T is kinetically hindered so T accumulates as U and C convert to T. Furthermore the OH defunctionalization of G to form A is accelerated but the functionalization of A to G is kinetically hindered. Thereby with diet the host accumulates ^{2}D, ^{13}C, ^{15}N and ^{17}O for altered functionalizations of U, T, C, A, and G so that the clumped nonprimordial isotopes in the DNA causes internal chemical transformations of U → T and C → T and G → A with the developed deficiencies of G and C for causing the normal cell to transform to cancer cells.

On the basis of such accumulation of A and T in cells with deficiencies of G and C as discovered in this work by the model [1-3], many mutations are explained and model for cancer genesis. For instance the lack of G and C by this model leads to the in ability of RNA to properly translate some proteins like Cys, Trp and Arg. Such in ability to translate these proteins correlates with the deficiency of Try, Arg and Cys in microcancer environments. Such alterations of protein translation on basis of functionalizations of nucleotides by nonprimordial isotopes provide a new mechanism for protein splicing for cancer genesis. The induced low translation of Try and Arg due to low G and C content further explains the unusual interactions of cancer with plasmodium malaria virus which is known to have los G and C content in its DNA. This model by its determination of deficient G and C in cancer cells accounts for many mutations associated with cancer habitat. Less frequent cancer in whales, elephants, mole rats and bats can be reasoned by this low G and C due to nonprimordial isotopes as presented in this model. Low G and C has been determined to cause low melting point of DNA; such low G and C in cancer DNA relative to normal DNA is a basis for heat sensitivity of cancer cells. By using the defient G and C discovered in cancer in this research the altered cancer metabolism in zero gravity is understood in a new way as changing gravity would change the force fields about the cancer cells and alter its softer DNA relative to DNA of normal cells for selective killing cancer cells in zero gravity. Telomerase has the associate RNA with sequence ____; so that low G and C would prevent RNA for expressing telomerase as is the character of cancer cell. So the low G and C in cancer DNA explains the less expression of telomerase and the shortening of telomeres in cancer. The unusual G and C in bats can be explained by this model by the awkward flying and forces on bats for genetic mutations for higher contents of G and C with consequenct unusual proteins in bats with explanations of ebola virus as it originates from bats. Whales and large mass and buoyancy without gravity for developing unusual G and C contents for long life of bats. In general fungi have high G and C content and the anti cancer properties of fungi may be correlated to such for instance the unusual protein in brown seaweed fungi for forming polysacchides in these seaweeds in environment rich in nonprimordial isotopes. The ease of mutation of C by $^{13}CH_3$ methylation may explained how DNA was designed to involve stops that lack C. This theory explains how and why the lack of G and C correlates with less Gly and Pro and altered formations of alpha helical blocker and induction of beta turns. New theory for eating animal DNA and cancer. By this new theory it is further predicted that animals that eat animals have a greater possibility of cancer relative to herbivorous as the animal tissue have cells with DNA that enrich in nonprimordials and mutate the DNA in the animals that eat the DNA. But plant DNA has less nonprimordials. Eating animal DNA may cause cancer. Eating plant DNA may cure cancer. This theory may explain why plants do not get cancer due to their lack of

motion and their use of wind to move. But bats have huge motions for high levels of G and C in bat DNA; and plants are low G to C and bats high G to C. Therefore by this theory the ability of animals and humans to move: crawl, walk, fly and swim causes needed extra catabolism with nonadiabatics (shaking nonprimordials for hidden dynamics of RBL) for greater incorporation of nonprimordials in animals and humans and cancer genesis. But trees lack such extensive catabolism and motions and accumulate less nonprimordials for less cancer in plants. For some reason grapes have seeds high in nonprimordials. Grape seeds have DNA that exchange nonprimordials with cancer DNA to kill the cancer relative to normal cells. Even greater eating of nonprimordials by host may lead to new technologies for treating and curing cancer by selectively stimulating the accumulated nonprimordial isotopes in the cancer cells.

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Tables

Table 1 : 407 Da - 409 Da

White Blood Cells			Cancer Cells			Red Blood Cells		
400.144	0.039	0.097	402.221	0.063	0.062	400.128	0.016	0.099
400.342	0.023	0.089	403.278	0.037	0.086	400.329	0.013	0.122
401.138	0.045	0.114	405.146	0.045	0.060	401.117	0.014	0.086
402.231	0.093	0.103	407.169	0.248	0.059	402.222	0.031	0.109
403.124	0.026	0.076	408.184	0.050	0.049	403.137	0.012	0.058
403.282	0.050	0.116	409.283	0.133	0.063	403.271	0.023	0.122
404.269	0.050	0.117				404.268	0.017	0.142
405.171	0.043	0.179				405.160	0.020	0.209
406.166	0.020	0.100				406.198	0.011	0.090
406.326	0.018	0.112				406.322	0.011	0.100
407.180	0.296	0.096				407.169	0.101	0.091
408.185	0.043	0.089				407.321	0.015	0.101
408.315	0.018	0.115				408.174	0.018	0.090
						408.341	0.019	0.098

Table 2 : 428 Da – 429 Da

White Blood Cells	Cancer Cells	Red Blood Cells
425.229 0.035 0.169	425.283 0.044 0.075	425.268 0.024 0.135
426.292 0.018 0.199	429.153 0.248 0.049	426.277 0.011 0.202
427.236 0.032 0.167	430.161 0.051 0.049	427.250 0.011 0.089
429.164 0.242 0.099	430.161 0.051 0.049	428.162 0.010 0.112
430.173 0.032 0.113	436.114 0.040 0.069	429.151 0.110 0.096
430.346 0.018 0.012	heavier cancer	430.352 0.011 0.109
431.187 0.019 0.120		431.150 0.011 0.100
436.116 0.062 0.093		436.108 0.018 0.097
Unusual 436 in white ?		

Table 3 : 445 Da

White Blood Cells	Cancer Cells	Red Blood Cells
442.234 0.021 0.212		442.255 0.012 0.239
443.336 0.034 0.140	439.119 0.050 0.032	443.336 0.014 0.135
444.344 0.015 0.112	445.101 0.100 0.061	444.344 0.013 0.080
445.118 0.193 0.095	455.302 0.044 0.080	445.118 0.069 0.095
445.298 0.067 0.102		446.117 0.016 0.108
446.117 0.031 0.14		

Table 4- 483 and 484 Da

White Blood Cells	Cancer Cells	Red Blood Cells
480.318 0.020 0.214		
481.236 0.022 0.105		481.358 0.012 0.125
482.078 0.035 0.092		482.199 0.015 0.088
483.073 0.050 0.095		483.065 0.020 0.096
483.231 0.035 0.071		483.217 0.015 0.075
483.372 0.050 0.097	483.391 0.074 0.069	483.370 0.030 0.096 (R hv vs W)
484.044 0.022 0.097		484.041 0.027 0.101
484.258 0.019 0.081		
484.378 0.057 0.097	484.381 0.052 0.087	484.377 0.031 0.104 (R hvr vs W)
485.047 0.028 0.103		485.046 0.027 0.098
485.246 0.033 0.085		485.472 0.024 0.178
485.374 0.037 0.105		486.048 0.015 0.099
486.257 0.027 0.214		486.380 0.011 0.181
487.287 0.296 0.101	487.270 0.165 0.055	487.045 0.010 0.103 (R Lt vs W)
488.291 0.084 0.113	488.276 0.049 0.058	487.279 0.067 0.099 (W peak)
489.072 0.037 0.116	489.047 0.042 0.066	488.038 0.020 0.097
		488.256 0.024 0.197
		489.051 0.047 0.101 (R hv W)

Table 5

White Blood Cells	Cancer Cells	Red Blood Cells
498.385 0.017 0.098	499.366 0.043 0.083	499.703 0.022 0.099
499.254 0.029 0.072	500.367 0.044 0.089	500.027 0.023 0.102
499.367 0.065 0.101	501.242 0.092 0.055	500.388 0.020 0.104
500.257 0.021 0.074	503.254 0.043 0.083	501.222 0.032 0.099
500.390 0.058 0.100	505.263 0.463 0.064	501.386 0.015 0.109
501.241 0.121 0.099	506.264 0.113 0.070	502.254 0.013 0.091
501.390 0.037 0.107	507.266 0.065 0.080	502.378 0.018 0.097
502.251 0.042 0.095	515.362 0.051 0.064	503.250 0.056 0.112
502.386 0.031 0.096	Cancer is lighter at 506 Da	504.251 0.019 0.102

503.260 0.190 0.109	No peaks in at 503 Da	504.385 0.012 0.117
504.265 0.067 0.119		505.261 0.161 0.101
505.068 0.017 0.115		505.393 0.020 0.070
505.272 0.530 0.106		506.260 0.046 0.103
506.278 0.138 0.111		507.277 0.028 0.100
507.292 0.093 0.124		507.388 0.018 0.096
508.295 0.028 0.170		508.328 0.011 0.205
509.283 0.024 0.246		509.408 0.014 0.124

Table 6

White Blood Cells			Cancer Cells			Red Blood Cells		
521.332	0.024	0.216	522.289	0.050	0.047	521.371	0.012	0.113
522.310	0.056	0.129	525.418	0.036	0.116	522.305	0.018	0.136
523.233	0.047	0.128	527.238	0.055	0.068	523.220	0.015	0.111
524.234	0.023	0.108	529.240	0.035	0.081	523.403	0.011	0.083
525.247	0.034	0.095				524.208	0.010	0.113
525.400	0.033	0.119				524.403	0.011	0.110
526.266	0.024	0.256				525.254	0.014	0.098
527.252	0.080	0.099				525.388	0.022	0.112
527.401	0.030	0.117				526.389	0.013	0.122
528.265	0.029	0.084				527.246	0.026	0.102
528.401	0.066	0.097				527.400	0.011	0.126
529.265	0.048	0.097				528.249	0.011	0.088
529.390	0.044	0.105				528.396	0.020	0.104
530.334	0.032	0.227				529.255	0.019	0.098
						529.398	0.014	0.097
						530.333	0.013	0.240

Table 7

669 and 676

White Blood Cells			Cancer Cells			Red Blood Cells		
666.179	0.021	0.122	665.136	0.077	0.041	666.178	0.011	0.138
666.428	0.016	0.150	665.231	0.041	0.066	667.247	0.019	0.121
667.299	0.017	0.156	669.258	0.098	0.083	668.234	0.025	0.115
667.369	0.016	0.041	670.264	0.046	0.098	669.231	0.100	0.115
669.273	0.116	0.114	671.287	0.091	0.064	669.423	0.010	0.129
669.431	0.025	0.093	672.277	0.052	0.064	670.221	0.051	0.117
670.274	0.055	0.113	673.297	0.186	0.058	671.256	0.039	0.124
670.443	0.020	0.117	674.298	0.081	0.057	671.449	0.010	0.140
671.290	0.100	0.124	675.217	0.069	0.051	672.248	0.022	0.118

672.298	0.040	0.150	675.309	0.041	0.106	672.725	0.010	0.120
673.307	0.221	0.121	676.211	0.039	0.070	673.293	0.061	0.129
673.715	0.017	0.104				673.465	0.012	0.126
674.102	0.031	0.119				674.160	0.015	0.084
674.311	0.085	0.122				674.307	0.026	0.105
675.100	0.039	0.111				675.212	0.079	0.116
675.317	0.048	0.139				675.351	0.011	0.143
676.285	0.022	0.118				676.205	0.046	0.116
676.436	0.018	0.103						

Table 8 - 681-683

White Blood Cells			Cancer Cells			Red Blood Cells		
678.310	0.038	0.158	675.309	0.041	0.106	676.205	0.046	0.116
678.390	0.021	0.113	676.211	0.039	0.070	677.221	0.017	0.115
679.283	0.019	0.095	681.092	0.038	0.051	677.380	0.013	0.152
679.465	0.017	0.133	681.257	0.152	0.060	678.244	0.011	0.112
680.440	0.016	0.115	682.246	0.125	0.073	679.224	0.012	0.125
681.100	0.019	0.124	683.250	0.037	0.065	680.215	0.012	0.142
681.263	0.054	0.111	687.114	0.140	0.055	681.090	0.020	0.094
681.447	0.025	0.090				681.253	0.113	0.113
685.442	0.016	0.129				682.241	0.126	0.116
686.430	0.026	0.137				683.246	0.033	0.112
	0.019	0.133				684.249	0.012	0.117
						685.494	0.011	0.156
						687.113	0.037	0.116

Table 9 - 697 698 Da

White Blood Cells			Cancer Cells			Red Blood Cells		
692.250	0.023	0.129	687.114	0.140	0.055	692.207	0.018	0.120
693.263	0.023	0.114	691.242	0.045	0.094	693.218	0.011	0.130
695.283	0.065	0.117	695.270	0.046	0.059	695.265	0.021	0.131
695.460	0.016	0.146	697.288	0.042	0.024	696.246	0.015	0.220
696.081	0.028	0.115	703.075	0.039	0.044	697.044	0.010	0.075
696.283	0.031	0.115				697.211	0.042	0.120
697.082	0.039	0.116				698.206	0.023	0.132
697.293	0.032	0.148				699.270	0.010	0.208
698.434	0.018	0.110				699.508	0.010	0.139
699.319	0.026	0.232						

Table 10 – 703 and 709 Da

White Blood Cells	Cancer Cells	Red Blood Cells

703.413	0.016	0.130	703.075	0.039	0.044	703.066	0.018	0.100
704.242	0.016	0.106	709.088	0.106	0.063	703.238	0.023	0.103
704.430	0.017	0.140	719.039	0.036	0.052	704.233	0.024	0.113
705.438	0.020	0.136				705.238	0.010	0.113
707.254	0.018	0.113				709.089	0.036	0.118
709.095	0.083	0.117				713.184	0.021	0.117
710.094	0.021	0.107				714.167	0.019	0.114
712.439	0.018	0.113						
714.292	0.023	0.168						

Cytidine diphosphare 403.176 gm/mole triphosphate 483.156gm/mole
Adenosine diphosphate 427.201, triphosphate 507 gm/mole
Guanosine diphosphate 443.20 gm/mole, triphosphate 523 gm/mole
Uradine diphosphate 404.16 gm/mole , triphosphate 484.14 gm/mole
Thymidine diphosphate 402.19 , triphosphate 482.168 gm/mole