

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

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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-viral and anti-bacterial properties. Proanthocyanidins in grape seeds are observed to have high enrichment in heavy isotopes of ^2H , ^{13}C , ^{15}N and/or ^{17}O . Mass

analysis of DNA from human cancer cells are compared to normal human cells and cancer cells show bond specific enrichment of heavy isotopes in nucleotides G, A, T and C. On such basis, this study suggests possible stronger interactions of proanthocyanidins with DNA in cancer versus DNA in normal cells due to heavy isotope bond specific enrichments in both proanthocyanidins and the cancer DNA. 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 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 ^1H , ^{12}C , ^{14}N , ^{16}O , and ^{24}Mg isotopes by nonprimordial ^2D , ^{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 ^2D , ^{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 Krebs cycle (catabolism). Glycolysis is catabolic process of enzymatic conversion of glucose to pyruvate. The cellular transformations to cancer lead to accelerated glycolysis. Krebs cycle is catabolic enzymatic conversion of pyruvate to carbon dioxide. The cellular transformations to cancer lead to suppression of Krebs 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 also 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 Krebs cycle that suppress the Krebs cycle. A big mystery of cancer is the nature and mechanism of the DNA mutation, RNA mutation and altered protein translations. In this work, the prior theory [1-3] that nonprimordial isotopes drive, DNA, RNA and protein alterations for cancer is substantiated and the use of nonprimordials to alter cancer metabolism for new treatments of cancer is further stressed.

Isotopic DNA, RNA and Protein Alterations for Mechanism

In this work, the theory [1-3] of stable isotopic replacements and substitutions of primordial, stable ^1H , ^{12}C , ^{14}N , ^{16}O , ^{24}Mg and ^{32}S by nonprimordial, stable ^2D , ^{13}C , ^{15}N , ^{17}O , ^{25}Mg , and ^{33}S is further developed. This work focuses more on DNA, nucleotides and telomeres. Prior papers focused on glycolysis and Krebs cycle. In normal cells, the ends of DNA have unbounded, telomeric regions, which are shortened to terminate replications of genes, but in cancer the telomeres do not shorten and induce apoptosis. But in cancer, the telomeres mutate and involve telomerase with acceleration of replications [4]. Telomerase is a protein that is associated with elongations 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 (^2D , ^{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's 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 prevent the shortening of the telomeres for causing apoptosis for causing cancer. The nonprimordial isotopes cause such alternations by interfering with signaling to apoptosis by the nonprimordials binding of the telomeres for causing consequent continued replication of the DNA with more and more replications; such that the DNA becomes too bond specifically enriched in nonprimordial isotopes (^2D , ^{13}C , ^{15}N , ^{17}O , ^{25}Mg and ^{33}S) of different nuclear magnetic moments (NMMs) [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 bond-specifically enrich in the nonprimordial isotopes (^2D , ^{13}C , ^{15}N , ^{17}O , ^{25}Mg and ^{33}S) in specific bonds relative to the primordial isotopes (^1H , ^{12}C , ^{14}N , ^{16}O , ^{24}Mg and ^{32}S) for greater probability of simultaneous, multiple nonprimordial clumpings in specific bonds in both proteins and nucleic acids. On such basis, 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 interacting between the telomere and telomerase 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 specific bonds in the telomeres change the binding of the base pairs in the genes, so that

the shorter telomeres (and indeed for other genes and their expressions) do not express apoptosis as the telomeres are bound more tightly by the nonprimordial isotopes. The telomeric genes are bound more strongly to binding proteins for telomerase expression. So that the stronger bound nonprimordial, isotopic, shorter telomeres continue to allow the DNA to replicate and the resulting nonprimordial DNA to replicate further to transcribe nonprimordial RNA and the resulting nonprimordial RNA continues to produce nonprimordial proteins. In the DNA and RNA, the accumulations of nonprimordials by ^2D , $^{13}\text{C}^1\text{H}_3$, $^{15}\text{N}^1\text{H}_2$ and $^{17}\text{O}^1\text{H}$ (and $^{13}\text{C}^2\text{D}^1\text{H}_2$, $^{15}\text{N}^2\text{D}_2^1\text{H}$, $^{17}\text{O}^2\text{D}$) functional replacements on nucleotides of guanosine (G), adenosine (A), cytidine (C), uridine (U) and thymidine (T) rather than primordial ^1H , $^{12}\text{C}^1\text{H}_3$, $^{14}\text{N}^1\text{H}_2$, $^{16}\text{O}^1\text{H}$ 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 ^2D , $^{13}\text{C}^1\text{H}_3$, $^{15}\text{N}^1\text{H}_2$ and $^{17}\text{O}^1\text{H}$ (and $^{13}\text{C}^2\text{D}^1\text{H}_2$, $^{15}\text{N}^2\text{D}_2^1\text{H}$, $^{17}\text{O}^2\text{D}$) on guanosine, adenosine, cytidine, uridine and thymidine cause magnetic bondings in addition to the hydrogen bondings to reduce and hinder the separations of the DNA base pairs for causing normal cells to transform to cancer cells. But by the prior theory [1-3], such can cause greater nonprimordial uptakes by the cancer DNA; so new treatments are possible as here with proanthocyanidins as the overall nonprimordial bond-specific enriched cancer DNA becomes less separable with killing of the cancer cells by over isotopically enriching the nucleic acids and proteins in the cancer.

Theory for Mechanism of Cancer and Cure [1-3]

Atomic and Molecular Dynamics for Altered Biochemistry

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 (^2D , ^{13}C , ^{15}N , ^{17}O , ^{25}Mg and ^{33}S) relative to primordial isotopes (^1H , ^{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: ^1H {99.988%, 1 $\frac{1}{2}$ (I) spin, 2.79 (μ/μ_N) NMM} and ^2D {0.0115%, 0 (I) spin, (μ/μ_N) } NMM}. Carbon has 2 important stable isotopes with different NMMs, relative abundances, masses and spins: ^{12}C {98.9%, 0 (I) spin, 0 (μ/μ_N) NMM} and ^{13}C {1.1%, $\frac{1}{2}$ (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%, $\frac{1}{2}$ (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%, $\frac{1}{2}$ (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 food supplies of plants, animals and humans such that humans have increased levels of the nonprimordial stable isotopes (^2D , ^{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

enrichments, the radiowaves compound the clumping of non-primordial isotopes into specific chemical bonds in proteins, RNA and DNA. Technologies introduced all these new ingredients and conditions of nonprimordials, RF and microwaves and static magnetic fields (B_{ext}) to explain origin of cancer and acceleration of cancer.

Changes in Biomolecular Chemical Dynamics

These non-primordial isotopes reversibly, fractionally fission and fuse to momentarily transmute to different quantum fields about the nuclei in atoms and molecules relative to the reversible, fractional fission and fusing of primordial isotopes. Moreover, on the basis of this theory [1-3], the author has determined that the fractional, reversible fission 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 fission and fusing of the stable isotopes by the author's theory [1-3] alters the enzymatic dynamics along the reaction coordinates of protein, nucleic acid, lipid, and carbohydrate biochemical dynamics. The fractional, reversible fission and fusing of nuclei release NMMs to surrounding electrons for 'internal nuclear pressures' to alter surrounding atomic orbitals and such altered atomic orbitals 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 and nucleon orbitals also alter electronic orbitals for relativistic nuclear Little Effect as manifested by these nonzero NMMs of nonprimordials relative to more null NMMs of primordials.

For instance, the fractional, reversible fission 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 nuclear 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 fission 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 fission 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 ^2D , ^{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 Krebs cycle.

On the basis of the author's theory [1-3] the surrounding radiowaves and static magnetic fields of technologies accelerate such faster glycolysis and slower Krebs cycle. Alterations of DNA reproductions, RNA transcriptions and protein translations cause cancer. The author further notes that both technologies for more nonprimordials and technologies for more external RF, EM radiations and static magnetic fields in combination cause more cancer and continued acceleration of cancer during last 150 years. But the author notes here just as nonprimordials can affect biomolecules in normal cells to cause cancer these nonprimordials can also affect cancer itself to kill cancer and with B_{ext} and RF such use of nonprimordials to heat and kill cancer cells is enhanced. The author in this paper uses proanthocyanidins as in skin of fruit and grape seeds as natural sources of ^{13}C , ^{15}N and ^{17}O enriched polyphenols (nucleotides-like structures) to more strongly interact with cancer DNA in deadly ways (relative to normal DNA). Proanthocyanidins (PACs) are polyphenol compounds. Some foods are rich in PACs: blueberries, grapes, cranberries, cinnamon bark, hazelnuts and chocolate. Polyphenols are organic molecules having many phenol units. In this work the authors, determine similarity of polyphenol structures nucleoside structures in G, C, A, U and T nucleotides can cause favorable interactions and binding between the polyphenols of PACs and nucleotides in DNA. It is thought that the intrinsic

nonprimordial bond-specific enrichment in PACs and the bond specific enrichment of nonprimordials in cancer DNA may cause stronger interactions of PACs with cancer DNA to alter cancer genetics and metabolism for use of PACs for treating cancer. The author notes the cancer already has ^{13}C , ^{15}N , ^{17}O in its DNA and RNA and proteins. The proanthocyanidins have structures and ^{13}C , ^{15}N , and ^{17}O enriched in specific bonds to bind the cancer DNA/RNA in stronger ways.

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 shortened 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 ^2D , ^{13}C , ^{15}N , ^{17}O , ^{25}Mg and ^{33}S), then the interactions between the telomerases and the DNA change, becoming stronger due to magnetism of fractional, reversible fission and fusion of the nonprimordial isotopes; so that the telomeres of the DNA open and close like regions in normal telomeres 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. External electromagnetic waves and static magnetic fields can be factors affecting such processes for explaining the illusive effects of electric, magnetic and electromagnetic fields on organisms. Thereby cancer develops by random isotopic editing of DNA such that the nonpri-

mordial telomerase 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: ^1H , ^{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 ^2D , ^{13}C , ^{15}N , and ^{17}O that accelerate rather than stop replication due to stronger binding of the telomeres to proteins with nonprimordial isotopes: ^2D , ^{13}C , ^{15}N , and ^{17}O . In prior paper [4], 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. Finally, it is hypothesized that drugs and/or food having nonprimordial enriched isotopes like proanthocyanidins can more strongly bind cancer DNA to disrupt the cancer genetics and metabolism for treating cancer.

Method

In order to test some aspects of this hypothesis normal red and white cells and Leukemia cancer cells were obtained and 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 comparison 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 reasoned based on exchange of nonprimordial isotopes between the

proanthocyanidins and the oligonucleotides.

Results

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 versus 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 versus 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 fragmentations of the DNA into nucleosides, nucleotides and oligonucleotides during MALDI mass analysis process with varying characteristic isotopic compositions of $^2\text{D}/^1\text{H}$, $^{13}\text{C}/^{12}\text{C}$, $^{15}\text{N}/^{14}\text{N}$, $^{17}\text{O}/^{16}\text{O}$ and/or $^{25}\text{Mg}/^{24}\text{Mg}$ within the fragments. The C to T \rightarrow U and A \rightarrow

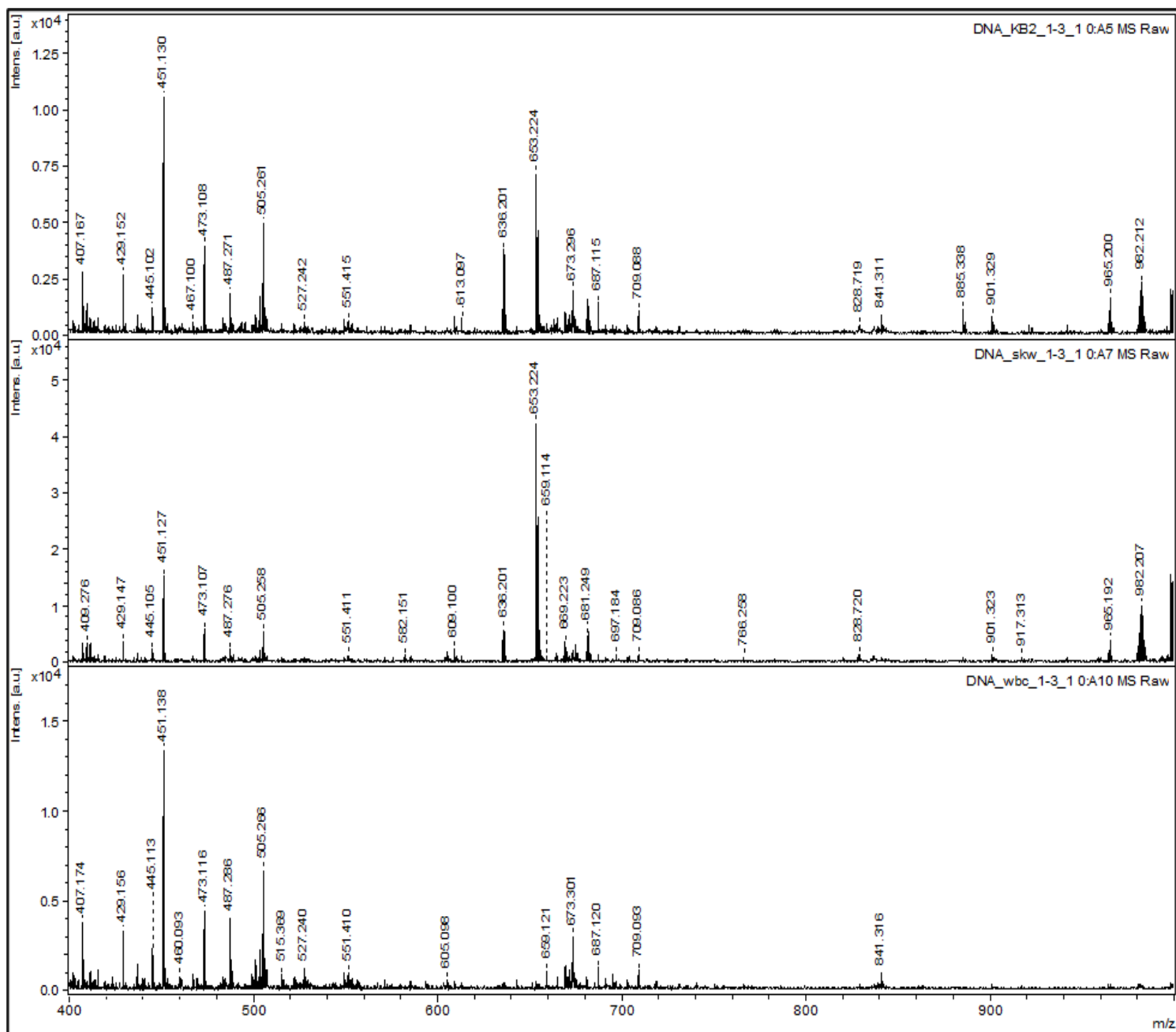


Figure 1. Mass Spectra (400 Da to 1000 Da) of DNA from Normal Cells and Cancer Cells

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	0.06	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	0.05	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				405.16	0.020	0.209
406.166	0.020	0.100				406.198	0.011	0.09
406.326	0.018	0.112				406.322	0.011	0.100
407.18	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	427.25	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				431.15	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	0.08
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	0.02	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	483.37	0.03	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 hv 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				486.38	0.011	0.181
487.287	0.296	0.101	487.27	0.165	0.055	487.045	0.01	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	0.02	0.097
						488.256	0.024	0.197
						489.051	0.047	0.101 (R hv W)

Table 5. 498-509 Da

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	500.0270	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			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	0.07
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. 521-530 Da

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						

G has methylations, dehydrations, deaminations and hydrations of rings of aromatics, purines, pyrimidines and ribose rings and phosphate groups as 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 versus cancer cells.

Discussion of $^{13}\text{CH}_3$ 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 \rightarrow T by dehydroxylation and methylation and vice versa. And the T can \rightarrow 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 difficulty rehybridization dynamics due to more electron $\times\times\times$ electron interactions about O nuclei and the attached p^+ . The ^{17}OH accelerates both the bond breakages for methylations and aminations. This theory determines important $^{17}\text{OH}_2$ and $^{15}\text{NH}_3$ nanosolutions [1-3] inside cancer cells such that the nanosolvent $^{17}\text{OH}_2$ and $^{15}\text{NH}_3$ weaken bonds in the nucleotides to accelerate $^{13}\text{CH}_3$ nucleophilic replacement in the aromatics for kinetics of the mechanism [1-3]. Typically, aromatics are so stable that they are more difficult for nucleophilic aromatic substitutions, but the negative nuclear magnetic moments (NMMs) of ^{17}O and ^{15}N lower the activation energies for accelerated substitutions on the nucleophiles [2,3]. The ^1H 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; strong electron $---$ electron interactions in valance of O further facilitate dynamics for lability. The $^{15}\text{NH}_3$ 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 [1-3] are consistent with prior experimental observations of NH_3 recycling by breast cancer cells. But researchers have not experimentally explained $^{17}\text{OH}_2$ in cancer cells [1-3]. But this theory predicts large anomalous dynamics of $^{17}\text{OH}_2$ in cancer cells [2,3]. The methylations are faster as ^{13}C has positive NMM. The negative NMM of ^{15}N may alter its amination of reaction centers. Dense nonzero NMMs may deaccelerate the $^{15}\text{NH}_2$ deamination.

The nonprimordials in U may cause it to manifest 409 Da peak in the cancer and the enrichments of nonprimordials and clumping in the cancer DNA. Such nonprimordials in cancer in U may accelerate its conversion to T by accelerating dehydroxylations of ribose and accelerating methylations of pyrimidine by $^{17}\text{OH}_2$ complexation and $^{15}\text{NH}_3$ complexation of U from surrounding nano- NH_3 and H_2O nanosolution. The ^{16}OH in the ribose of cancer's 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 ^{17}OH may be fragmenting from T nucleotides of cancer DNA to explain the spectra. The $^{13}\text{CH}_3$ is better, stronger nucleophile and more rapidly attacks the pyrimidine due to the positive NMM of ^{13}C of $^{13}\text{CH}_3$. 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 solubilities and greater complexations of $^{13}\text{CH}_3$ by nanowaters of $^{17}\text{OH}_2$ and $^{15}\text{NH}_3$ relative to complexations and solubilities of primordial $^{12}\text{CH}_3$. 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 methylations of the U and dehydroxylations are expected by prior theory [1-3] to have enriched nonprimordials in the T in cancer DNA. (So in general the $^{13}\text{CH}_3$ is a stronger better nucleophile and stronger base than $^{12}\text{CH}_3$ due the harder basicity $^{13}\text{CH}_3$ relative to $^{12}\text{CH}_3$ [2,3]. So now in general ^{17}OH ($^{15}\text{NH}_2$) is a better leaving group than ^{16}OH ($^{14}\text{NH}_2$) and a weaker base

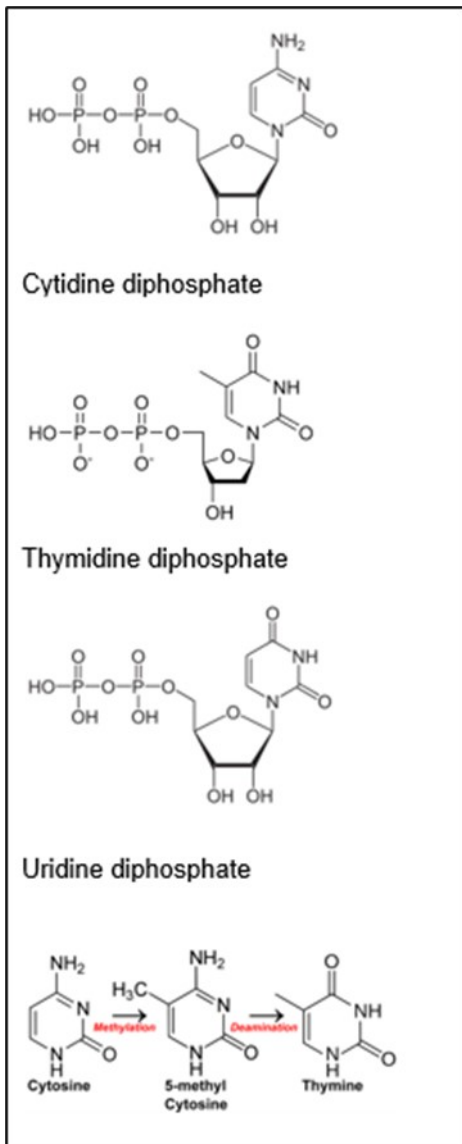


Figure 2. Pyrimidine Nucleotides

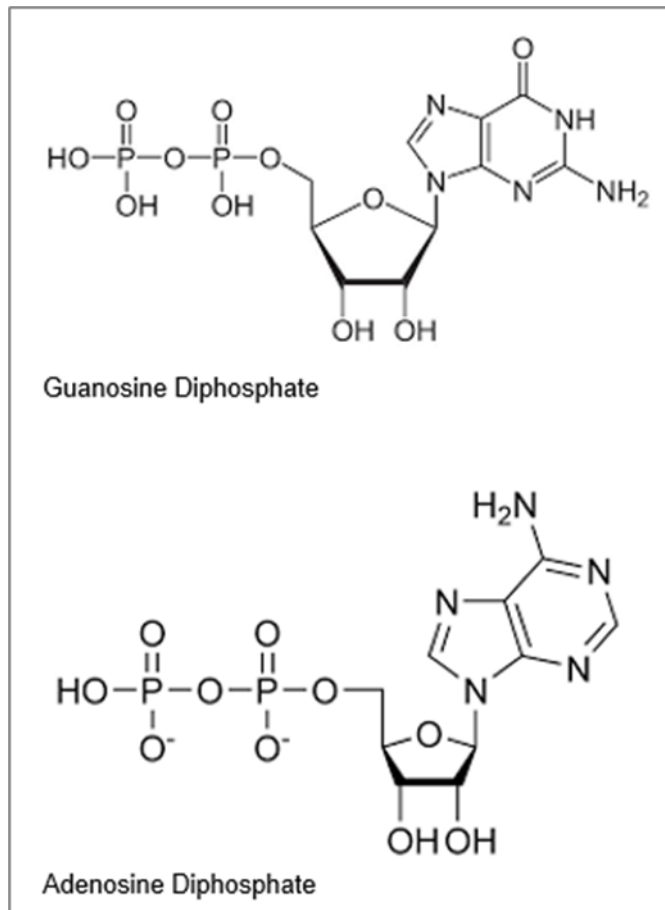


Figure 3. Purines

than ^{16}OH ($^{14}\text{NH}_2$) due to the greater polarizability of ^{17}OH ($^{15}\text{NH}_2$) to ^{16}OH ($^{14}\text{NH}_2$) [2,3].

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

The T may convert to C by deaminations and hydroxylations and demethylations. So in cancer the nonprimordials may accelerate the deaminations of T as $^{15}\text{NH}_2$ is a poor leaving group relative to $^{14}\text{NH}_2$. See Figure 2. But the hydroxylations may be more in cancer (as ^{17}OH is a better nucleophile than the ^{16}OH) relative to deaminations in cancer DNA for kinetics and thermodynamic reasons. But the $:\text{OH}_2$ is stronger base than $:\text{NH}_2$ and kinetically electronic rehybridizations are more labile in OH due to greater electron density about the O nucleus relative to N nucleus [2,3]. Thermodynamically the O-H bonds are stronger than N-H bond due to the larger effective nuclear charge of O. The $:\text{O}$ may be stronger base as its nucleus pulls $e^- e^-$ pair more strongly with denseness than $:\text{N}$. So denser $e^- e^-$ pull proton electrostatically. The zero NMM of ^{16}O and positive NMM of ^{14}N cause magnetic pull of $e^- e^-$ by $:\text{N}$, but the electrostatic extra p^+ in ^{16}O nucleus pulls more the $e^- e^-$ pair for its greater basicity. But as ^{16}O goes to ^{17}O the null NMM goes to negative NMM for larger more polarizable $e^- e^-$ cloud and also as ^{14}N goes to ^{15}N the positive NMM goes to negative NMM for even larger relative change in $e^- e^-$ cloud size and polarizability. So both ^{17}O and ^{15}N are weaker bases but softer bases relative to ^{16}O and ^{14}N . The nuclei magnetically polarize the $e^- e^-$ about ^{17}O and ^{15}N and the interactions with the p^+ is magnetic. And the interaction changes abruptly as temperature increases, pressure increases and electric and magnetic fields stimulate so depending on conditions stronger magnetic interactions can cause ^{15}N and ^{17}O to be stronger bases and change their nucleophilicities.

In cancer, the demethylations may be slower than in normal cells as $^{13}\text{CH}_3$ of the cancer may be 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 nonprimordial 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 formations of C^* (404 Da) are observed in the cancer as the $\text{U}^* \rightarrow \text{T}^*$. But T^* does not $\rightarrow \text{C}^*$ in cancer. The data thereby reveal depleted C^* in the cancer with 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 $^{13}\text{CH}_3$ relative to $^{12}\text{CH}_3$ during cancer genesis so that the cytidine may transform to uridine and thymidine for altering the ($^{13}\text{CH}_3$) 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). It may be that 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 with nonprimordial enrichment at 409 Da peak and enrichment of primordials at 407 Da peak. The $^{13}\text{CH}_3$ is a stronger nucleophile by this theory so it more rapidly attacks C in the $^{15}\text{NH}_3$, $^{17}\text{OH}_2$ solutes about DNA in nanosolutions in cancer cells [2,3]. This theory thereby predicts and explains 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 conversions in C^* (methylated) C and to then

accelerate the deaminations of pyrimidine of C* for loss of ^{14}N and replacements by OH to form U*. Thereby C is not only by this theory [1-3] 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 phosphate 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 of ^{17}O on $^{31}\text{PO}_3^-$ and ^{17}O and ^{15}N on the purines of guanosine diphosphate. Normal cells have ^{16}O on $^{31}\text{PO}_3$ with stronger protonation and ^{16}O and ^{14}N on purines of guanosine, but cancer DNA may accumulate ^{17}O in $^{31}\text{PO}_3$.

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 dehydrations and deaminations of 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 $^{13}\text{CH}_3$. 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 nonprimordial ^{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 . ^{17}O is more labile than ^{16}O . The formation of the 429 Da from the 445 Da for G \rightarrow A involves deaminations and dehydrations of G then aminations to A. The cancer cells have nanowater with $^{17}\text{OH}_2$ and $^{13}\text{NH}_3$ 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 substitutions by ^{14}N to form the A from the G by this theory [1-3] to explain the data. The clumping of nonprimordials in cancer DNA and G nucleotide may accelerate the deaminations and dehydroxylations of G as cancer has $^{15}\text{NH}_2$ and ^{17}OH , which are poor nucleophiles and good leaving groups due to their negative NMMs. The $^{15}\text{NH}_2$ is a stronger

nucleophile than ^{17}OH as ^{15}N has a less negative NMM than ^{17}O ; so $^{15}\text{NH}_2$ is expected to be harder than ^{17}OH . $^{15}\text{NH}_2$ is expected to be a better nucleophile than ^{17}OH for replacing ^{17}OH of G* to form $^{15}\text{NH}_2$ of A*, so the $^{15}\text{NH}_2$ is harder base and should attack the purine rings faster than ^{17}OH weaker nucleophile. Thereby cancer DNA should readily transform G* \rightarrow 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 transcriptions and protein translations 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 \rightarrow Adenosine Diphosphate

The unusual enrichments 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 deaminations, hydroxylations and aminations to form the G at 445 Da peaks. Thereby 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 nonprimordials 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 transform readily due to its clumped nonprimordial isotopes transform to A*. The dehydroxylations and deaminations and aminations of the G* in cancer DNA are accelerated due to the negative NMMs of the ^{15}N and ^{17}O for alterations of leaving ability in the dehydroxylations and deaminations. But the aminations to form the final A* is expected in cancer DNA as the $^{15}\text{NH}_2$ is a weaker base and weaker nucleophile than ^{17}OH due to the harder basicity of $^{15}\text{NH}_2$ relative to ^{17}OH and due to the less negative NMM of $^{15}\text{NH}_2$ relative to more negative NMM of ^{17}OH . The

hydrogens also help harden the $^{15}\text{NH}_2$ as there are more H in $^{15}\text{NH}_2$ relative to ^{17}OH . Stronger internal magnetism to stabilize $^{14}\text{NH}_3$ versus $^{15}\text{NH}_3$ has greater stability of $^{15}\text{NH}_3$ so $^{14}\text{NH}_3$ goes to $\text{N}_2 + \text{H}_2$ faster and stronger bonds. ^{15}N triple bond is less stable. ^{14}N triple bond may be easier to break. So breaking $^{15}\text{N}_2$ may be easier than breaking $^{14}\text{N}_2$ and the stability of ^{15}N -R in organic compounds may be greater than ^{14}N -R toward N_2 formations.

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 couple by chemical transformations to 502 Da of U* (methylated U) in cancer by methylations ($^{13}\text{CH}_3$, 16Da); by U* dehydroxylating to form T* (with $^{13}\text{CH}_3$, 16Da); by U* dehydroxylating (^{17}OH , 18Da) and aminating ($^{15}\text{NH}_2$, 17 Da) to form C*. See Figure 2. Such many possible dynamics manifest nonprimordial accelerated functionalizations 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 new mechanisms of mutations of DNA and RNA as here disclosed as by not only by changing isomeric connectivity along chains but also 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 nonprimordials clumping from 400 to 409 Da. The nonprimordials ($^{13}\text{CH}_3$) 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, the random methylations of C and random $^{13}\text{CH}_3$ in C, T, and U can lead to such fine peaks in the normal cells. But the cancer cells have nonrandom, clumping of $^{13}\text{CH}_3$ and $^{15}\text{NH}_2$ and ^{17}OH and the $^{13}\text{CH}_3$ causes stronger binding of the cancer DNA for less fragmentations under electromagnetic fields in mass spectrometer. There is more 483 Da in cancer DNA and there is more 484 Da in normal DNA, there is 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* conversions in cancer would involve the dehydroxylations of pyrimidine and hydroxylations of ribose and the aminations of the pyrimidine. The negative NMMs of the ^{17}OH and $^{15}\text{NH}_2$ in cancer make this less likely. As the negative NMM of ^{15}N and ^{17}O make $^{15}\text{NH}_2$ better nucleophiles for the conversions of the pyrimidine to C* in cancer DNA but instabilities. The cancer DNA thereby less expresses C* at 483 Da.

Dehydration of Adenosine Triphosphate and Suppression by Clumping $^{13}\text{CH}_3$

The 487 Da is from the dehydroxylations (^{17}OH or ^{18}OH $^{17}\text{O}^{2-}$ or $^{18}\text{O}^{2-}$ of 18 Da to 20 Da) of ribose in adenosine triphosphate at 507 Da. See Figure 3. In normal cells, the dehydroxylations are 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 dehydroxylations. So 523 Da 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 $^{15}\text{NH}_3$), so the 507 Da peak in cancer lacks heavier nonprimordials as they are loss of ^{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 deaminations, hydroxylations and aminations of the purine. The cancer having clumped nonprimordials may accelerate this as the $^{15}\text{NH}_2$ and ^{17}OH in the cancer DNA are weaker nucleophiles (due to their negative NMMs) relative to $^{14}\text{NH}_2$ and ^{16}OH . But in principle $^{15}\text{NH}_2$ and ^{17}OH should be poorer entering groups due to their negative NMMs but ring ^{15}N can pull in the $^{15}\text{NH}_2$ 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 $^{13}\text{CH}_3$ 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 $^{13}\text{CH}_3$ is active in the fragmenting the daughter peaks are enriched in nonprimordials as the $^{13}\text{CH}_3$ fragments stabilize by $^{13}\text{CH}_3$ with consequent heavier daughter peaks. The nonprimordials in the ^{17}OH destabilize and the $^{13}\text{CH}_3$ stabilizes primordials. So the enrichments in the daughter by primordials are actually due to lack of instability or less fragmenting of nonprimordial than primordials.

All + or - NMMs activate bond breaking. All - NMMs have faster kinetics of bond breaking to cause new effects relative to all + NMMs. Faster kinetics can lead to different product distributions and breaking stronger bonds. All negative NMMs may break C-C, C-O, C-H, O-H bonds and all positive break $\text{C}=\text{C} \leftrightarrow \text{C}-\text{C}$. The activated state may then better bond back together by + NMMs + +NMMs with faster rates and with more thermodynamic stability. Such may be more discerned under conditions of high temperature, strong electric fields, strong magnetic fields and/or high pressures. The π bonds in DNA and RNA makes it easier to alter by NMMs, explaining why reproductions, transcriptions and translations are more affected by nonprimordials relative to glycolysis and Krebs cycle. Amino acids having π bonds like tyrosine and phenyl alanine ($\text{C}=\text{C}$) may more easily be affected by nonprimordials. Aspartate and glutamate have carbonyl side groups with resonating pi bonds. But amino acids have $\text{C}=\text{N}$ and $\text{C}=\text{O}$ and $\text{C}=\text{C}$ but not aromatic. Carbonyls have resonating $\text{C}=\text{O}$.

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. $\text{G} \rightarrow \text{A}$. See Figure 3. The nonprimordial G at 525 Da peak more rapidly loses ^{17}O to

produce more than 50% greater loss 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 causing $\text{G} \rightarrow \text{A}$. The 523 Da peak may involve transformations between A and G with a surrounding peak; so that in cancer there is peak enrich in primordial isotopes. $\text{A} \rightarrow \text{G}$ by hydroxylations, deaminations, and aminations. $\text{G} \rightarrow \text{A}$ by dehydroxyations, deaminations and aminations. Ammonia in tumor can encourage aminating and deaminating G and A, and also induce $\text{C} \rightarrow \text{T}$.

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 $^{13}\text{CH}_3$ methylations of cytosine and the cytosine may undergo deaminations and dehydroxylations or the C may $\rightarrow \text{U}$ by deaminations and hydroxylations under acidic conditions as in altered nucleuses (isotopic replacements) as nucleuses are more basic than cytoplasm. The more basic nucleus in cells stabilize T and U as T is more basic and nonpolar relative to U. So in cytoplasm, the $\text{T} \rightarrow \text{U}$ as the more acidic cytoplasm can push out $^{13}\text{CH}_3$. It is 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 accumulations of nonprimordial T^* are observed as the T^* cannot (due to clumped nonprimordials) convert to C^* as the conversion of T^* would require demethylations (loss of $^{13}\text{CH}_3$). The $^{13}\text{CH}_3$ is a strong base and good nucleophile and the cancer cells cannot as well lose $^{13}\text{CH}_3$. 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 having more $^{12}\text{CH}_3$ and $^{14}\text{N}_2$; 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 $^{13}\text{CH}_3$ 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 enrichments of $^{13}\text{CH}_3$ in T* in nonprimordial cancer is detected and the inability to convert T* to C* in the cancer increases T*. 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 $^{17}\text{O}_2$ and H_2^{17}O and $^{13}\text{C}^{17}\text{O}$. 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 by 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 $695 \text{ Da} + 14 \text{ Da} = 709 \text{ Da}$ peak or the $695 \text{ Da} + 9 \text{ Da} = 703 \text{ Da}$. 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 $^{15}\text{NH}_3$; and NH_3 is less abundant in normal cells! It seems in general ^{17}O helps decompositions and fragmentations. The $^{17}\text{OH}_2$ and $^{15}\text{NH}_3$ in surrounding nano-water in cancer cells may accelerate

exchange of $^{12}\text{NH}_2$ and ^{16}OH by $^{13}\text{NH}_2$ 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 complexations of this biomolecules by ^{17}OH and $^{15}\text{NH}_2$ cause 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 $^{13}\text{CH}_3$ reactions are accelerated in cancer, the methylation consistently shows heavier peaks in cancer DNA and its pieces. But where ^{17}OH and $^{15}\text{NH}_2$ are involved the aminations and hydroxylations consistently show smaller masses in the mass spectra of cancer DNA and its pieces. The larger massive pieces during methylations result and are explained by the addition of more massive $^{13}\text{CH}_3$ into the functional of DNA nucleotides. The less massive pieces during aminations and hydroxylations 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 $^{13}\text{CH}_3$ and its positive NMMs strengthen the covalent bonds in cancer DNA for binding $^{13}\text{CH}_3$ is a stronger nucleophile for more rapid replacements in DNA and its nucleotides. But the $^{15}\text{NH}_2$ and ^{17}OH and their negative NMMs weaken the covalent bonds in cancer DNA for bond breakages and ^{17}OH and $^{15}\text{NH}_2$ are 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 + NMMs \rightarrow classical or all – NMMs \rightarrow classical, but balanced + NMMs \rightarrow and – NMMs \rightarrow 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 at higher temperatures, in electric fields and magnetic fields. But

¹⁵N can help ¹³C at higher temperature, in electric fields and magnetic fields. ¹⁷O disrupts ¹⁵N quantum mechanically, but together they help pull in ¹³C and less ¹⁴N causes loss of protein nuclear perturbation. On such basis the author notes tumors may be killed by enriching ¹⁷O, ¹⁵N, and/or ¹³C in their biomolecules and exposing them to strong electric fields and/or strong magnetic fields. ¹³C may overdrive classical mechanics of protein with ¹H and ¹⁴N. ¹³C causes accelerated glycolysis as driven fragmentation of glucose. But the combining of C to O is opposed by ¹³C and ¹⁴N in the Krebs cycle or they oppose $sp^3 \rightarrow sp, sp^2$. + NMMs favor sp^3 , - NMMs favor sp and sp^2 for ¹³C but not for ¹⁷O. So ¹³C favor sp^3 and ¹⁷O favor sp^3 (for different reasons) as higher $e^- e^-$ density for ¹³C increase electron density on C and less $e^- e^-$ repulsions for negative NMMs of ¹⁷O reduces electron repulsions about O. So ¹³C and ¹⁷O accelerate glycolysis by one environment. But ¹³C and ¹⁷O suppress the Krebs cycle as in the Krebs cycle the sp^2 and sp hybridizations are catalyzed about C and O and the ¹³C and ¹⁷O oppose such sp and sp^2 hybridizations but favor sp^3 hybridizations. But ¹⁷O and ¹³C decelerate Krebs cycle by different environments.

In this work, the author proposes a new way to alter functional groups of uridine, thymine, cytosine, adenine and guanine (by isotopic substitutions/replacements of ¹H, ¹⁶OH, ¹⁴NH₃, ¹²CH₃, and ²⁴Mg by nonprimordials of ¹⁷OH, ¹⁵NH₃, ¹³CH₃, ²D and ²⁵Mg) as nonprimordial, functional groups entering 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 NMMs 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 transportations, 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 (NMMs) are in analog to fermionic electrons in atoms. By such the atoms in the domains have a wave natures and they exchange and correlate to move and alter their wave natures and they exchange and correlate to move and alter motions and positions in the nanosolution so as to lower energies. But for biomolecules such waves are quantum waves and differ from larger classical waves as by the theory [1-3], the nano, subnano waves can superposition to focus intensities in to specific bonds for quantum activations and this explains novel bond activations by enzymes. 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 nano-ammonia. So that the biochemical transformations have been previously described by the author as 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. Methylations (¹³CH₃) of U cause T*. So isotopic effects in cytoplasm get into nuclei by $U + ^{13}CH_3 \rightarrow T^*$ in cytoplasm and transfer of T* into nucleus. So ¹³CH₃ 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 ¹³CH₃. So ¹³CH₃ seems like H (by their positive NMMs); so T* becomes as U; and U in nucleus alters genes. Normally U is in cytoplasm and T is in the 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 ¹³CH₃ (methyl) on the thymine alters biochemical dynamics. Also ¹³CH₃ in T* may accelerate T* \rightarrow C* by dehydroxylations, deaminations, and aminations. So this causes mixing of nucleotides and mutations by chemically interconverting of nucleotides. T \rightarrow U. U \rightarrow C. Such chemical transformations of nucleotides al-

ter 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 replications 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. 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. 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 + NMMs but many ^{14}N , ^1H and ^{33}S and other ^{13}C can induce, new bond formations, but as excess + NMMs cleave + ... + NMMs bonds and excess - NMMs cleaves - ... - NMMs bonds in quantum fields. So quantum fields + ... - NMMs globally bond and + ... + NMMs locally agitate bonds and - ... - NMMs locally agitate bonding and as the nonprimordial isotopes clump they manifest new enzymatics of the DNA and RNA. . So this theory [1-3] introduces totally new chemical dynamics as here it is determined novel non-local chemical bonding but local chemical decomposition and/or nonlocal chemical decomposition but local chemical bonding.

The patterns of null, + and - NMMs (needles in haystack) can cause local bonding while globally the fermions are unbound. So the theory [1-3] determines that

systems of + and - NMMs (Nuclear Frames) bind the atoms globally on large scales as they locally 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 substitutions of isotopes. But the theory [1-3], the + and - NMMs as are 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 - NMMs interacting with - NMMs repel locally in nuclei but bind to attract globally as in Ag nanoparticles and other rare elements having all - NMMs. But such considerations, RBL gives a totally new model for transportations (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 Frames with less chemistry and transport possibilities. Such manifest in primordial nanosolutions in cells having + NMMs of ^{14}N , ^1H and ^{31}P and null nuclear magnetic moments (NMMs) of ^{12}C , ^{14}N , and ^{16}O and normal primordial biology manifest on such basis of repulsions on NS Frames and motions and biochemistry of binding on L frames of wavefunctions. But RBL introduces totally new effects of - NMMs + ... + NMMs binding locally in NS Frames and repelling globally in L frames. So bonds are broken globally to isolate the $e^- e^-$ but locally the $e^- e^-$ bind by the + NMMs and - NMMs to manifest a Reggie Pair bond by NMMs of + and - NMMs as this occurs in nanosolutions in cells as $^{17}\text{OH}_2$ and $^{15}\text{NH}_3$ enrich with $^{13}\text{CH}_3$ in the nanosolutions, proteins and nucleic acids. So the nanosolutions bind on NS Frames 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 causing wavefunctions to repel. But the nuclei still pin the atoms together for cancer habitat.

It is important to consider that by such model of theory [1-3], in normal cells the ^{14}N and ^1H can modulate the bond cleavages and bond formations of PO_3^- and the

ribose as the compressions may induce bond cleavages of $^{31}\text{PO}_3^-$ to release energy and the chemical composition of ribose (of null NMMs). As compressions 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 fissings and fusings decompose ribose and these can couple to pull apart base pairs or also such dynamics couple to surrounding proteins to bind or decompose the proteins to pull in or push out proteins. And such can explain DNA replications quantum mechanically as bases recognize quantum mechanically by patterns of NMMs and compress/rarefy with pulling in and pushing out. And likewise for transcriptions. And in ribosomes such act vice versa as pulling in amino acids under conditions whereby the oligonucleotides, RNAs are stable.

The clumping may help ^{15}N incorporations 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 clumpings of dense regions of nonprimordials isotopes alter nuclei acid bindings, bond strengths and chemical stabilities 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 better nucleophile than the $^{15}\text{NH}_3$ and $^{15}\text{NH}_3$ or $^{14}\text{NH}_3$ is less abundant in normal cells! It could be that the presence of $^{14}\text{NH}_3$ causes the genetic alterations of normal cells to cancer cells and the $^{15}\text{NH}_3$ 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 deuterons, hydroxyls, amines, and methyls. Such clumpings of nonprimordials lead to sharper distinct fragmentations during the mass analysis of DNAs for sharper peaks relative to broader peaks in fragmenting of the primordial regions of normal DNA. By the theory, the incorporation of nonprimordials of ^2D , ^{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. It is important to note that the easier fragmenting of these pieces having nonprimordial isotopes in cancer cells relative to less sharp fragmenting in normal cells is evidence of altered interactions of nonprimordial isotopes in the DNA and RNA for altering the replications, transcriptions and translations.

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 by the theory [1-3] previously modelled such biomolecules on the basis of Na^+ and K^+ interactions with graphene oxides. It was determined that Na^+ and K^+ NMMs interact favorably with graphene oxides with their sp^2 and sp^3 mixed hybridizations and magnetics via the nonzero NMM of K^+ and Na^+ . Thereby, likewise, RBL reasoned similar NMMs interact with sp^2 and sp^3 networks but now in biomolecules like DNA. So that the theory [1-3] introduced changes in interactions in the DNA as primordials of ^1H , ^{12}C , ^{14}N , ^{16}O , ^{24}Mg , and ^{32}S are replaced by nonprimordials of ^2D , ^{13}C , ^{15}N , ^{17}O , ^{25}Mg and/or ^{33}S of different NMMs. Such manifest as the purines and pyrimidines in nucleic acids regions with sp^2 aromatic and regions with sp^3 nonaromatic in analog to prior different regions in graphene oxide.

Why Do Nucleotides Transform on Atomic Scale

Thereby the theory [1-3] realized nuclear spins could couple to carbon covalent dynamics in prior graphene and in biomolecules. But even before the experiment with graphene oxide the spin interactions and NMMs

of p^+ interacting with biomolecules had been published in a book Chapter [2]. So by considering graphene an analog for proteins and other biomolecules. The theory [1-3] proved 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 induce transports, exchanges and replacements of the different NMMs. These extended π electrons and orbital exchange and bonding about many atoms may be mechanism for more strongly coupling the nuclear spins and NMMs {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 exchange by π electrons. The nuclear spins and the nuclear orbital angular momenta are thereby exchanged and coupled via delocalized $\pi e^- e^-$ in the phenyls, polyphenols, and polyphenylamines. Also by this model [1-3], such spins are not limited to e^- spins; nuclear spins are also coupled, transformed, transported and transmuted by $\pi e^- e^-$ and d orbitals of transition metals. By the model [1-3], the substrates couple and quantum mechanically exchange the NMMs in the enzymes and macromolecules and vice versa.

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, antiferromagnetism and paramagnetism 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 delocalized to rehybridized to localized 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 transportations 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 fissions and fusions of nuclei seep QF into electronic shells as by the theory [1-3], 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 $e^- e^-$ rehybridizations and spin polarizations can alter the couple nuclear orbital momenta by RBL Effect. The localized 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 Little's Effect. The e^- spins and nuclear spins via delocalized or d (azimuthal) $\pi e^- e^-$ can couple to alter the symmetries and motions from locals to nonlocals and vice versa.

Why Purines, Pyrimidines, Polyphenols and Polyphenylamines More Strongly Couple NMMs?

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, and 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 nanodomains. Thereby the pyrimidine's aromaticity more exchange the nonprimordials. The purine's aromaticity less exchanges

the nonprimordials. Just as for the pi electrons in purines and pyrimidines delocalize the NMMs of ^{13}C , ^{15}N and ^{17}O , likewise the pi electrons in polyphenols and polyphenylamines delocalize these nonprimordial isotopes and their NMMs. The nucleotides, oligonucleotides and nucleic acids couple their nuclear magnetic moments (NMMs) with NMMs in surrounding nanowater and accumulated NH_3 to accelerate primordials replacements 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 spin 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 change polarizations to change bonds. By this mechanism [1-3], the spins not only interact with the orbits, but the spins transform by fractional, reversible fission and fusion. Fissioned spins fractionally, reversibly fission and fuse to orbits and vice versa the orbits fuse to spins. So also NMMs via e^- orbitals can couple nuclear spins and change the orbitals. Nuclear spin momenta and orbital momenta can alter the e^- orbital. And e^- orbitals can alter nuclear angular momenta. RBL here notes NMMs are variable by not only during chemical reactions but also during chemical reactions, enzymatics, vibrations, optics and e^- transportations and transmutations. The nuclei are perturbed so relative motions of nucleons change and the nuclei swell and compress for fractional, reversible fission and fusion to alter and to couple to surrounding e^- lattice. Thereby momentary changes in NMMs occur. The DNA and RNA have more pi bonds for easier activations by breaking pi bonds for easier replacing isotopes relative to other biomolecules. Therefore, it is this reason of the aromatic rings in purines and pyrimidines 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 and/or diamagnetic, paramagnetic ferromagnetic functional substances that the resulting

DNA and RNA catalyze isotopic exchange in proteins. So that during DNA, RNA and protein bindings, interactions, charge exchanges and enzymatics, isotopes may be exchanged. By this theory, the ^{14}N , ^1H and ^{31}P via fractional, reversible fission and fusion cause the denatured proteins to renature and the DNA to un-nature and renature during reproduction, and RNA to denature and nature. So in general, the NMMs in the proteins and nucleic acids cause orbitals to change. So the proteins and nucleic acids denature and renature. So the proteins and nucleic acids renature so rapidly due to huge fields caused by the nonzero NMMs of ^{14}N and ^{31}P within them (and $^1\text{H}_2\text{O}$ in surrounding nanowater). Thereby from this theory the RNA may catalyze the nonprimordial replacements in amino acids as the RNA translates proteins.

The isotopic exchange is selective in uphill anabolism in animals and humans nonadiabatically as it is selective in uphill anabolism in plants adiabatically. It is during uphill processes of DNA replications, RNA transcriptions and protein translations that the proteins are isotopically altered. Virus RNA can modify so the RNA produces unhealthy proteins. It is that the side chain sugar and side chain phosphate couple energy into the nucleoside to break bonds. It is that the side phosphates and side sugars help the NMM replacements. This occurs by the ferrochemistry of the bond rearrangements of the sugar releasing energy reversibly as accumulations and absorbing into the phosphates by NMMs and other oligonucleotides by ^{14}N and ^1H so as to give energy to promote the dynamics. So activated states near or far are involved and then as the transition states relax to products, the phosphates collect the energy and restore 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 some viruses can kill cancer cells. But the downhill catabolism (relative to uphill anabolism) is less affected by isotopic replacements as the electronic energy can drive and dictate the dynamics. But in glycolysis the downhill 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 is less discriminating nonprimordial / primordial replacements. But in Krebs 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 Krebs cycle so that Krebs cycle becomes adiabatic as the heat is organized in the high fields. This is the reason the Krebs 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 translations, replications and transcriptions 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 organisms eat other animals and obtain essential amino acids, then the essential amino acids have more nonprimordials. So the nonprimordials within 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 is given here of NMMs coupling by MOs and AOs causing nucleophile substitutions and NMMs undergo substitutions and replacements.

Nucleophiles driven by nucleophiles but the spin driven by magnetism as the null spins diamagnetically pushed out MO and the nonzero spins pull in or push out MOs and AOs. But what about the + NMMs and - NMMs. The + NMMs pull in + NMMs and push out - NMMs in MOs and AOs. But in nuclei and continua + NMMs push out + NMMs and pull in - NMMs. 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 QFs apart for driving bond activations for bond rearrangements. This is powerful as by this theory [1-3] introduces new types of interactions as two or more objects interact in counter ways on different states on L frames they attract but smaller frames they repel and/or on L frames they repel and on smaller RS frames they attract. Something on inside binds whole and whole repels. Or something on inside repels as whole binds! This is new by author for how particle \leftrightarrow wave. This is new basis for composite forces. The author published this in 2007 as p^+ and nuclei bond $e^- e^-$ of covalence by fissing of p^+ and nuclei to create QFs 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 , ^2D , and it ^{14}N help pull in ^{13}C and ^{17}O as such 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 lower temp it pushes ^{13}C away.

The observed higher deuterations, methylations, aminations, hydroxylations and enrichments with $^{13}\text{CH}_3$, $^{15}\text{NH}_2$ and ^{17}OH in cancer cells is consistent with nonrandom clustering and higher density methylations in DNA of cancer cells [10]. Moreover, in this work in addition to explaining faster methylations by $^{13}\text{CH}_3$ to cause cancer, the faster methylations are explained in details by the accelerated ^{17}OH hydroxylations and many body $^{15}\text{NH}_2$ aminations, causing the transformations of $\text{C} \rightarrow \text{T} \rightarrow \text{U}$ and $\text{A} \rightarrow \text{G}$ for chemically altering DNA and RNA for new chemical paths of DNA and RNA mutations for explaining

cancer. $^{13}\text{CH}_3$ may be causing more methylations of DNA in cancer cells as in this theory [1-3], $^{13}\text{CH}_3$ is a stronger nucleophile than $^{12}\text{CH}_3$. This isotopic effects of the nucleic acids can explain recent mysteries. Positive NMMs of $^{13}\text{CH}_3$ relative to null (0) NMMs of $^{12}\text{CH}_3$ by the theory [1-3] cause the e^- to be pulled closer to nucleus of $^{13}\text{CH}_3$ relative to $^{12}\text{CH}_3$ nuclei. $^{24}\text{Mg}^{2+}$ should interact less strongly with $^{13}\text{CH}_3$ (and make $^{13}\text{CH}_3$ a stronger nucleophile) relative to $^{25}\text{Mg}^{2+}$ for more altering the bonding of $^{13}\text{CH}_3$ by $^{25}\text{Mg}^{2+}$ in cancer cells relative to the weaker effect of $^{25}\text{Mg}^{2+}$ and/or $^{24}\text{Mg}^{2+}$ interacting with $^{12}\text{CH}_3$ in the normal cells to explain the observed selective killing of cancer cells by $^{25}\text{Mg}^{2+}$ [11]. This accumulated $^{13}\text{CH}_3$ in RNA and DNA then alters RNA and the RNA alters translated proteins for mechanism of splicing phenomena. [12]. 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 $^{12}\text{CH}_3$ to $^{13}\text{CH}_3$ of the space twin relative to the earth bound twin would explain the observed elongation of the telomeres of the space twin as by $^{13}\text{CH}_3$ methylations of his telomeres and stronger binding of his telomeres by $^{25}\text{Mg}^{2+}$ for elongation rather than shortening of the telomeres of space orbiting twin [13]. The stronger binding of the telomeres containing $^{13}\text{CH}_3$ may less frazzle the ends for continued elongations.

Methylation and Altered Binding and Transcriptions and Translations

So after reasoning and explaining how the nucleotides are isotopically mutated and some consequences, here it is considered how altered genes malfunction. So these alterations of nucleotides alter the sequencings, constitutions, connectivities and stereochemistry of isomers so what are consequences? Based on this model [1-3], the methylations 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 normal base pairs are GC and

AT pairs. Also the functionalizations / defunctionalizations can alter the DNA and RNA sequencing transformation $C \rightarrow T \rightarrow U$ and $A \rightarrow G$ 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 similar to A and OH^- may replace NH_2^- for $C \rightarrow U$. So methylation of C and deamination forms C and U for mutations and for consequent possible misreading of protein; so for example $\text{UUC (Phe)} \rightarrow \text{UUU (Phe)}$, CUU (Leu) , CUC (Leu) , CUA (Leu) , and/or $\text{CUG (Leu)} \rightarrow \text{UUU (Phe)}$, 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 $C \rightarrow T$ and $\rightarrow G \rightarrow A$ 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 nonprimordials substituting for primordials.

The $^{13}\text{CH}_3$ binding may alter interactions and dynamics due to its different NMMs. Although thymine already has a $^{12}\text{CH}_3$, by changing the $^{12}\text{CH}_3$ to $^{13}\text{CH}_3$, the properties of the thymine change so that the $^{13}\text{CH}_3$ may appear like H on the phenyl ring of thymine; so thymine appears to behave like uracil for altered replications, transcriptions and translations. Such $^{13}\text{CH}_3$ and its + NMM may appear as ^1H so the thymine in nucleus of cells appears like uracyl (U) with alterations of the DNA replications and transcriptions inside the nucleus. So it is that U can exist in the nucleus but thymine (T) exists only in the nucleus. But if $^{13}\text{CH}_3$ replaces $^{12}\text{CH}_3$ on thymine then thymine appearing as uranyl can exist in cytoplasm also to alter biochemistry in cytoplasm. And uranyl can methylate to enter nucleus. This means that uracyl in the nucleus can be template as thymine (as $^{13}\text{CH}_3$ in thymine causes it to appear as uracyl). Or the other possibility is

that the ¹³CH₃ in thymine causes it to not be recognized. A third possibility is that the ¹³CH₃ causes the similar nuclear behavior as H so the thymine may behave as cytosine assuming the =O (OH) and NH₂ manifest similar basic interactions. So these are possible consequences of ¹³CH₃ on the thymine.

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

In addition to these nonprimordial, induced misreads of nucleic acids and proteins and nonprimordial, induced, inter-chemical transformations of C → T → U and G → A, this mechanism [1-3] further determines the consequent deficiency in C and G due to the + NMMs of ¹³CH₃ and - NMMs of ¹⁷O and ¹⁵N by difficult hydroxylations of A → G due to ¹⁷O and difficult demethylations of T → C. The consequent dynamics cause deficiencies in C and G [14-18] in cancer cells. The deficient C and G [14-18] on basis of this theory [1-3] causes deficient proteins translated by C and G [14-18]. See Figure 4. For instance, G and C strongly translate Gly (GGU, GGC, GGA, GGG) [21]. {Note Gly and Pro are extremely important for alpha

helical breakers. Gly and Pro start secondary structures of beta turns. Beta turns are turns in primary structure. Pro [19] has odd, cyclic structures in peptide bonds and these cause bendings of peptides. 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 [23], Try [22] and Ala [20] also have strong translations by G and C and shortages of C and G in cancer cells are 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); and Ala (GCU, GCC, GCA, GCG). [19-20] 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, UGC); His (CAU, CAC); and Gln (CAA, CAG). * means the amino acids are essential amino

		Second nucleotide					
		U	C	A	G		
First nucleotide	U	UUU Phe UUC UUA Leu UUG	UCU UCC Ser UCA UCG	UAU Tyr UAC UAA STOP UAG STOP	UGU Cys UGC UGA STOP UGG Trp	U C A G	
	C	CUU Leu CUC CUA CUG	CCU CCC Pro CCA CCG	CAU His CAC CAA Gln CAG	CGU CGC Arg CGA CGG	U C A G	
	A	AUU Ile AUC AUA AUG Met	ACU ACC Thr ACA ACG	AAU Asn AAC AAA Lys AAG	AGU Ser AGC AGA Arg AGG	U C A G	
	G	GUU Val GUC GUA GUG	GCU GCC Ala GCA GCG	GAU Asp GAC GAA Glu GAG	GGU GGC Gly GGA GGG	U C A G	
						Third nucleotide	

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

acids. But then, the following amino acids are not strongly affected by shortages in G and C contents in cancer cells: Phen* (UUU, UCC); Ile* (AUU, AUC, AUA); Met* (AUG); Try (UAU, UAC); Asn (AAU, AAC); and 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 [24] recently observed microenvironments of tumors are depleted in Trp*, Arg* and Cys. It is also important to note that Try* and Arg* are essential amino acids, as cells cannot synthesize these essential amino acids. But Gly and Glutamate were found by van der Heiden [18] to be abundant in cancer microenvironments. 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 may cause high densities of stops. The telomerase has its own RNA (3' – CCCAAUCCC 5') for translating telomerase. So telomeres cannot elongate and this is habitual of cancer cells as 13CH₃ methylates C on telomerase, then the C of telomere cannot help elongate the telomerase. It is important that the telomerase translation involves a lot of C and by this theory the deficient C may affect telomerase formation, length and stability for causing cancer as the lack of C causes lack of telomerase and the lack of elongating telomeres which is one hallmark of cancer.

After discovery of this new DNA, RNA and protein chemistry by Little Effect via NMMs of nonprimordials, this work considers plants oligomers and possibly such chemical interactions of plant oligomers with human oligomers. This work determined that just as the RNA, DNA and proteins can undergo intrinsic internal accelerated methylations, deaminations, aminations, hydroxylations 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 nucleotides in the host.

But what happens to DNA as animal products are consumed? Plant products have less nonprimordial ¹³C, ¹⁵N, ¹⁷O and ²D in their nucleus acids. But how does such low nonprimordials compare to animal DNA? Animals tend to have in general greater amounts of ¹³C and ¹⁵N relative to plants. Scientists find link between plant telomere and human telomere so plants live longer as by their lower nonprimordials relative to animals and humans. The diet and metabolism of tree differs from animals and trees have less motion and less energy demands so trees do not break down ¹³C compounds and then construct ¹³C as much in their DNA for high nonprimordial contents as occurs in animals and humans; so tree 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. Muscles and lysine cause ¹³C and cancer. Trees and plants 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 host DNA and RNA. So cancer can be transferred by large transfer of cancerous tissues. Rats are implanted with cancer tissue with induction of 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 functionalizations and defunctionalizations. Thereby a new chemistry is described based on discovered for nucleotides based on NMMs and magnetics driving substitutions of NMMs and a mechanism [1-3] by

which NMM substitutions can couple and mix with nucleophilic substitution energies. So by replacing new oligomers with nonzero NMMs in heavier isotope, the DNA of normal cells is disrupted to cause cancer. But in this work, it is further determined that just as the isotopic accumulations 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 $^{13}\text{CH}_3$, ^{17}OH , $^{15}\text{NH}_2$, and/ or ^2D that can replace ^1H , $^{12}\text{CH}_3$, $^{14}\text{NH}_2$, 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 + NMMs and nuclei of ^{14}N , ^{16}O and ^{12}C via the proton (^1H) can magnetically couple for novel many-body 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 protonic nuclei, life cannot exist and thereby disease may be caused by altering this natural rhythm [1]. For instance, consider intrinsically the $^{31}\text{PO}_3$ gives P center strong ability to attack ADP and AMP. So if change ^{12}C to ^{13}C then ^{13}C makes $^{13}\text{CH}_3$ a stronger nucleophile; and if change ^{16}OH to ^{17}OH then ^{17}OH is a weaker nucleophile and if change $^{14}\text{NH}_2$ to $^{15}\text{NH}_2$ then $^{15}\text{NH}_2$ a weaker nucleophile. So just as there is intrinsic NMM chemistry of $^{31}\text{PO}_3$ 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 . $^{13}\text{CH}_3$ attacks more with ^{17}OH than with ^{16}OH . ^{17}OH attacks ^{15}NH more. It is that + NMMs attack + NMMs more in L Frame but less in L Continua and Nuclear Frames (NS Frames). {Where interior quarks are in QS Frames; quarks are inside hadrons in RS Frames; hadrons are inside nuclei in NS Frames.} The NS frames couple continuously to interior LS continua of the electronic lattice outside the nuclei. Electronic orbitals exist in L frames (discontinua); Electrons manifest continua about them for ES Frames and discontinua within the electron for E Frames. The ES frames of the

electron can couple to the outer L continua of the electronic lattices to mix with the inner L continua of denser NS frame fields and such mixing of ES Frames and NF frames with diminution of stretch; 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 + NMMs attack – NMMs less in L Frames and more in NS Frames. So thereby pressure effects manifest as high pressures push then the + NMMs into – NMMs; so the L Frames – NMMs repel + NMMs → NS Frames + NMMs attracting – NMMs 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 theory [1-3] 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 ^1H , ^{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 NMMs attract in L frames. But as cancer forms by ^2D , ^{13}C , ^{15}N , ^{17}O , ^{25}Mg and ^{33}S on the earth's surface, then the nuclei have + and – NMMs and + and – NMM repel in L frames. 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 Frames for repulsion, 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 Frames 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 Frames' QFs. But then under compression ^{17}O pulls ^{13}C to it in

inner L Continua and in NS Frames. 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 attract on nuclear scale (NS Frame) and repel on QF scale (L Frames) and attract 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 the theory [1-3] 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 (NMMs) and nuclear spins under very high temperatures, strong electric fields and strong magnetic fields [1-3]. Simultaneous nuclear, chemical, and physical transformational phenomena are determined by the theory [1-3] 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 [25] and the seeds are considered and compared to these nucleotides in normal and 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 couple to biomolecules of human reproductions 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 having more ^{17}O and the ^{17}O pulls in more ^{13}C into seed [26]. This is in the literature [26]. It has been observed that the draught and ^{13}C and ^{17}O in the seeds make the seeds more anti-cancerous [25]. What is it about grapes that they incorporate ^{13}C and ^{17}O among the plant kingdom? In this theory, it is determined that the chemistry of ^{13}C , ^{18}O and ^{17}O cause greater incorporation of nonprimordials 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 [27].

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; release more nonprimordials by extended C-C bonds and π bonds and/or bind nonprimordial isotopes existing in DNA, RNA and proteins and causing cancer and other diseases. Also consider that the enzymes of Krebs cycle may be able to pull in more nonprimordials relative to enzymes of glycolysis due to the high field substrates of Krebs cycle. Both such networks of changing covalence in Krebs cycle and changing covalence of glycolysis process and the oligomeric 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 NMMs of other nuclei. By such Ferrochemistry the nuclei revolve to orbitals as by fractionally fissioning their NMMs so as to couple the covalence and to 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. Such explains the isotopic replacements by the covalent lattices with embedded NMMs in accelerated many-bodies relative to null NMMs as by the theory[1-3]. It is noticed

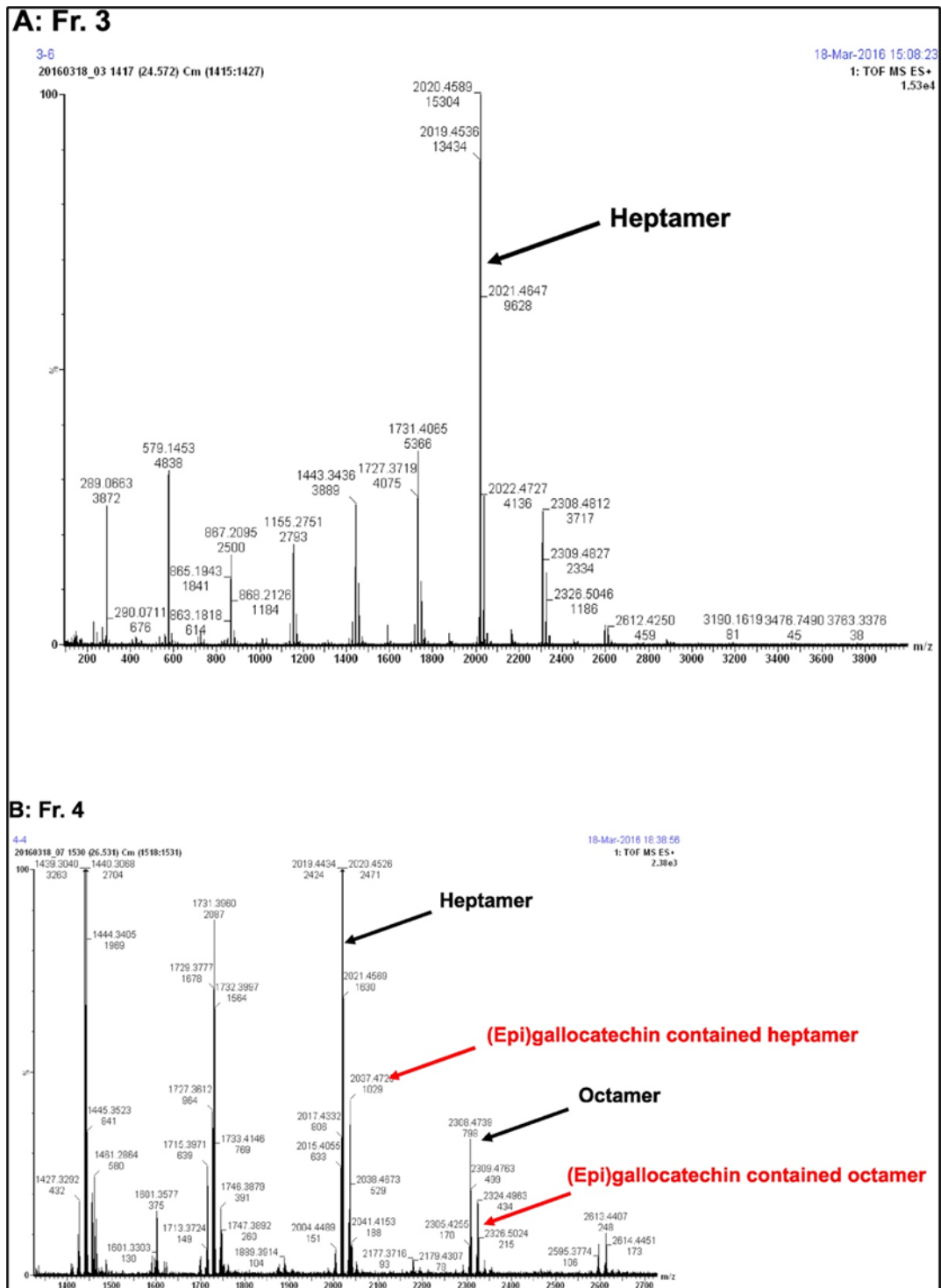


Figure 5. Oligomeric Proanthocyanidins from Adzuki Beans With Those Larger than Tetramers Showing AntiCancer Activities (reference 8)

that the greater enrichments of nonprimordial isotopes in the heavier isotopes correlate to the anti-cancer.

On this basis, a new idea [1-3] is presented. It may be that the different ferrochemistry of glycolysis, Krebs, replication, transcription, and translation can be reasoned by functional groups of amino acids as the alkyl + phenyl functionals may in proteins push together to induce greater nonprimordial uptakes. 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 incorporations of ^{17}O and ^{15}N . So if the theory [1-3] looks at enzymes of glycolysis, it may find fewer Leu and Trp than in Krebs cycle. Krebs cycle may have more Trp and Leu so it would more incorporate ^{13}C relative to glycolysis.

Proanthocyanidins are observed in grape seeds, cranberries and other fruits and vegetables [13]. The greater amounts of proanthocyanidins (PACs) in grape seeds and cranberries are revealed in mass spectrometer as isotopic clusters are observed in Figure 5. The nature of the interflavan bonds (D2 amu) $[\text{M}+\text{Na}]^+$ represented by observed masses. The PACs from grape seeds contain B type (m/z 1465) bonds. Masses represent variations in the nature of interflavan bonds (D2 amu) $[\text{M} + \text{Na}]^+$. It is

noticed that from the mass analyses, that the grape seeds explicitly show huge enrichments of either ^2D , ^{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 (PACs) 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 are observed at 1465 m/z, which are observed to have primordials of ^{12}C , ^{14}N and ^1H . See Figure 6-7. 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 ^2H or one ^{17}O . The mass at 1467 m/z may have two of these nonprimordials ^2D , 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 their enrichments with nonprimordial isotopes of ^2D , ^{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

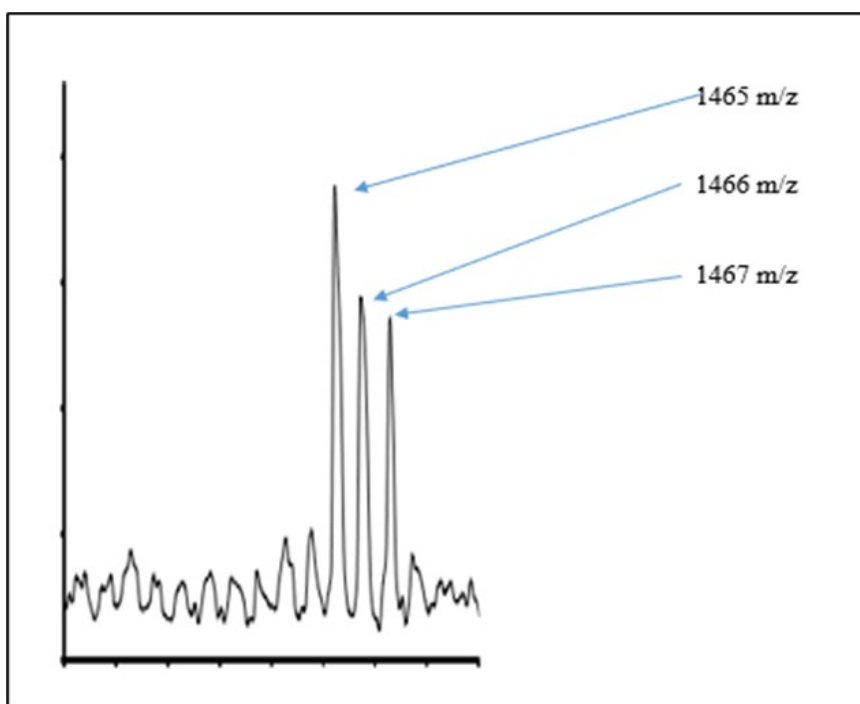


Figure 6. Grape Seed Proanthocyanidins (PAC) Isotopic Reveal Isotopic Enrichments (reference 14)

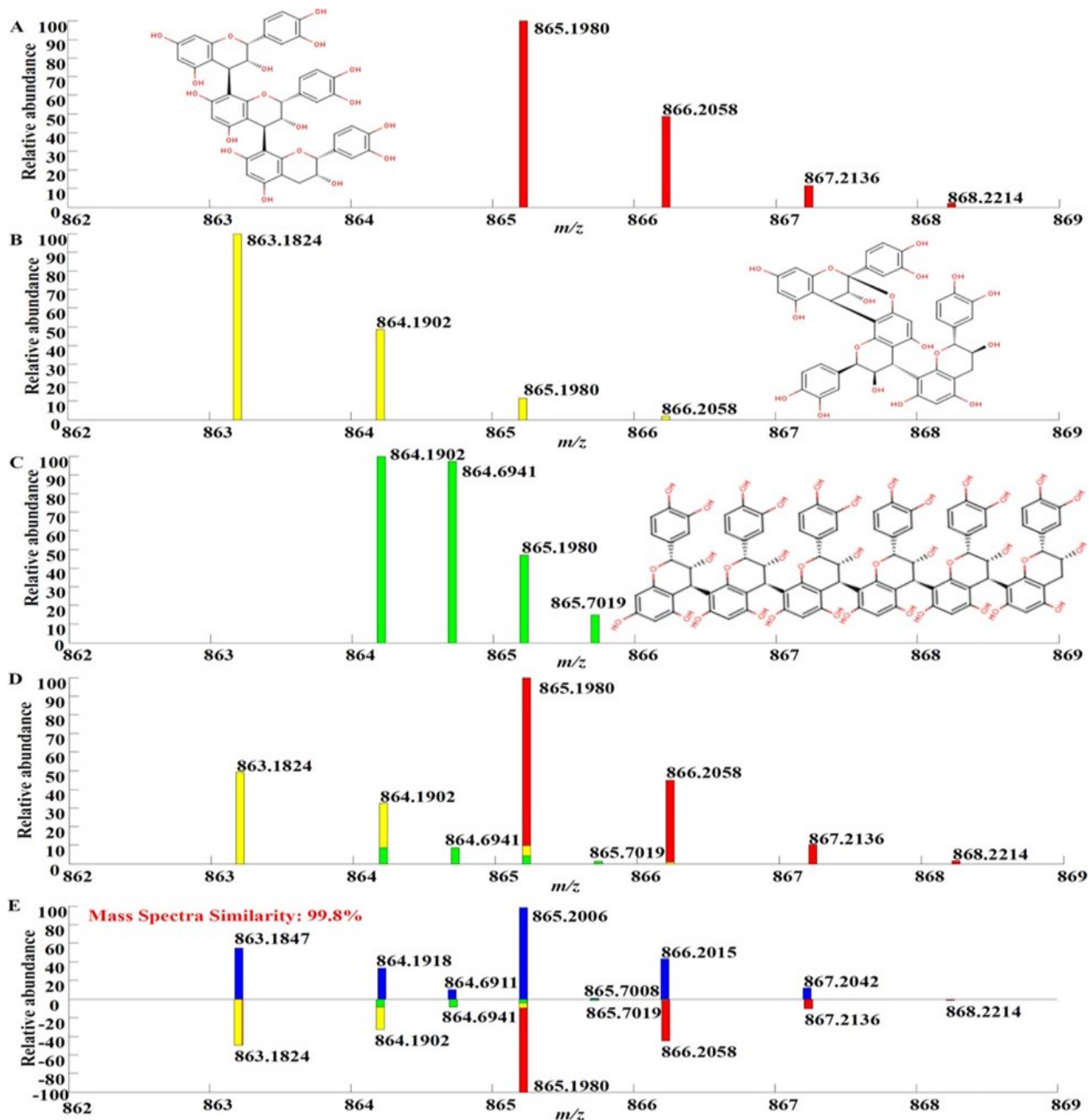


Figure 7. Structures and Masses of Nonprimordial Isotopes in Plant Proanthocyanindins (Reference 7)

nucleosides and the possible exchange of the nonprimordial isotopes between the nucleosides of RNA and DNA; and possibly more favorable binding of the nonprimordial enriched proanthocyanidins with nonprimordial enriched DNA and RNA in cancer cells. The proanthocyanidins may also alter the translations of proteins in cytoplasm and the synthesis of DNA in nucleus. In this work, it is determined that the nonprimordial isotopes couple (bind) more strongly to the cancer DNA and RNA relative to the RNA and DNA of normal cells, because by this theory [1-3] 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's replication. Thereby the grape seeds provide the epigenetics to alter cancer DNA selectively so the seeds are anti-cancerous and this is the first molecular basis for anticancer properties of grape seeds. This is consistent with prior theory [1-3] 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.

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

Previously the theory [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 theory [1-3] as grape seeds are in this work determined enriched in nonprimordial isotope. Therefore, eating seeds of grapes and other foods enriched with anthocyanidins are an examples of such eating foods isotopically enriched with nonprimordials. The theory [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 theory [1-3] proposed that radiofrequency rotates the nuclei and fractionally fissions 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 theory [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 superluminal 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 irradiations 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 effect on normal cells. In considering the merit of the theory [1-3] proposed for cancer cure, it is important to note that the 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 effects on normal cells. Soft X-rays can of certain wavelengths 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 with no absorption. In this invention, tuning the neutrons by slowing and rotating the nonprimordial isotopes by modulated RF and specific wavelength of X-rays can selectively increase absorption cross-sections of the nonprimordials for neutron absorptions and transmutations. All three (radiowaves, X-rays and neutrons) can penetrate the whole human body for effective treatment of the whole 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 ^2D , ^{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 enrichments with clustered kernels of $^{13}\text{CH}_3$ relative to DNA of normal cells. The surrounding nanowaters 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 $^{13}\text{CH}_3$ and $^{15}\text{NH}_2$ functionalization of nucleotides. The accumulations of ammonia about cancer micro-environment were determined to facilitate such $^{15}\text{NH}_2$ 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 ^2D , ^{13}C , ^{15}N , ^{17}O , and ^{25}Mg of different NMMs cause ease of ^{17}OH functionalizations and defunctionalizations with ^{17}OH catalyzing $^{15}\text{NH}_2$ and $^{13}\text{CH}_3$ functionalizations. The resulting $^{13}\text{CH}_3$ functionalizations cause difficult $^{13}\text{CH}_3$ defunctionalizations for accumulations of U as $\text{T} \rightarrow \text{T}$ and $\rightarrow \text{C}$ but the $^{13}\text{CH}_3$ defunctionalizations of T is kinetically hindered so T accumulates as U and C convert to T. Furthermore, the OH defunctionalizations of G to form A are accelerated but the functionalizations of A to G, which are kinetically hindered. Thereby with diet the host accumulates ^2D , ^{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 cause internal chemical transformations of $\text{U} \rightarrow \text{T}$ and $\text{C} \rightarrow \text{T}$ and $\text{G} \rightarrow \text{A}$ with the developed deficiencies of G and C for causing the normal cell to transform to cancer cells.

On the basis of such accumulations 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 inability of RNA to

properly translate some proteins like Cys, Trp and Arg. Such inability to translate these proteins correlates with the deficiency of Try, Arg and Cys in microcancer environments. Such alterations of protein translations 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 bacteria, which is known to have low G and C content in its DNA. Thereby, it is explained how bacteria of malaria may treat cancer. But other bacteria may cause cancer.

This model [1-3] 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. Weaker interactions by less G and C in cancer makes less rigid nucleus in cancer cells. By using the deficient 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 repeating sequences TTAGGG; 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 expressions 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 consequent 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 and whales. 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 polysaccharides in these seaweeds in environment rich in nonprimordial isotopes. The ease of mutation of C by $^{13}\text{CH}_3$ methylation may explained how DNA was designed to involve stops that lack C. This theory [1-3] 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 [1-3] for eating animal DNA and cancer is given. By this new theory [1-3] 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 [1-3] 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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