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All Humans are Pre-Programmed to Innate Carcinogenesis through the Co-Occurrence of Metastases Caused by Quantum Entanglement Entropy

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Abstract

Developmental biologists at Tufts University, using a tadpole model, that bioelectrical signals from distant cells control the incidence of tumors arising from cancer-causing genes and that this process is impacted by levels of a common fatty acid produced by bacteria found in the tadpole and also in humans. Bile acids and oxidative stress decrease DNA repair proteins, an increase in DNA damage and increased genomic instability through this mechanism of metastases caused by Quantum Entanglement Entropy. This process provides a mechanistic explanation for the important QEE link between a Western-style diet and associated increased levels of colon cancer.

Keywords: *CRISPR enzyme, CRISPR-Cas13d, indole-3-carbinol, lining of the intestines, the surface of the bowels, stomach acid, vegetables, cancer, gut inflammation, metastases, Quantum Entanglement Entropy*

Introduction

Tufts biologists showed bioelectrical signals control tumors arising from cancer-causing genes, fatty acid involved in process. Genetic information is often not enough to determine whether a cell will become cancerous, we also have to take into account the physiology of the cell and the bioelectrical signals it receives from other tissues. This has a huge implications for diagnostic technology as well as our basic understanding of the role of genetics and physiology in oncology.

Materials and Methods

These data also suggest a number of ways we might prevent, detect and treat cancer, said Michael Levin, Ph.D., Vannevar Bush Professor of Biology and corresponding author of the paper in the journal Oncotarget, that describes their research. By using ion channel drugs "electroceuticals" – to target the bioelectric state of distant sites in the body. Ion channel agents, such as anti-epileptic drugs, are already approved for human use.

Levin and Brook T. Chernet, Ph.D., injected xenopus laevis tadpoles with oncogenes associated with many human cancers. The oncogenes caused tumor-like structures to form in these locations. Levin and Chernet's study showed that the incidence of tumor formation could be significantly reduced through misexpression of hyperpolarizing ion channels, which control current flow across a cell membrane, even when these electrical signals originated far from the oncogene-expressing cells. These distant bioelectric signals suppressed tumor growth, despite the cells'continued high levels of oncogene protein.

Further investigation revealed that the tumor-suppressing effects of hyperpolarization were regulated by a mechanism involving the short chain fatty acid butyrate and its target, the enzyme histone deacetylase. In humans, butyrate is produced in the colon by natural bacterial fermentation of carbohydrates, and butyrate has been shown to protect against colorectal cancer. To confirm that bacterial butyrate was also involved in regulating distant tumor formation in tadpoles, the researchers administered

antibiotics. They found that the drugs indeed reduced butyrate production and thereby stopped membrane-voltage-based tumor suppression.

Above research uncovers a promising conncetion between the microbiome and cancer, that is controlled by alterations in bioelectric signaling and also opens possibilities for biomedicine. Bacteria that are metabolically programmed to produce butyrate levels may be appropriate to prevent tumors.

The distance over which carcinogenesis can be predicted and controlled has been addressed in a handful of earlier studies, including work of Lewin and colleagues. They have shown that aberrant bioelectrical properties of tissue revealed the location where tumors were likely to form and that melanoma-like growth could be triggered by bioelectrical signaling of instructor cells far from the melanocytes. More research is needed to determine whether such signaling occurs in mammalian cancer models and over what distance.

The Tufts biologists are also intrigued by the question of whether cancers emit bioelectrical information that could be detectable at a distance from tumors themselves. Probably the long-range signaling connections are bi-directional. (Chernet & Levin, 2014)

Colorectal Cancer Progression

Researchers at Nanjing University, China, found epigenetic markers for colorectal cancer (CRC) which may offer a new approach for treatment. Epigenetic alterations leading to colorectal carcinogenesis are thought to hold great promise for the development of novel, minimally invasive molecular biomarkers.

Scientists at the State Key Laboratory of Pharmaceutical Biotechnology, School of Life Sciences, Nanjing University, China, have demonstrated that heterochromatin protein HP1γ was critical for CRC cell proliferation and could be specially regulated by miR-30 at the protein level.

These studies have improved the chances that the epigenetic biomarkers will find a place in the clinical practices of screening, early diagnosis, therapeutic choice and recurrence surveillance for CRC patients, said professor Quan Zhao, who is one of the principal investigators at School of Life Sciences, Nanjing University, China.

In the future Professor Zhao and his collaborators want to further investigate the role of these new markers in individual steps of invasion-metastasis cascade of CRC.

Key Findings Included

HP1γ protein levels were significantly increased in primary CRC tissues compare to adjacent non-tumor tissues. Cancer correlation analysis indicated prognostic potential. Thus, HP1γ appears to be a promising biomarker for CRC and may provide an effective target for CRC therapy.

MiR-30a was down-regulated in CRC tissues, and exhibited a significant inverse correlation with the HP1γ protein level. Further, HP1γ could be post-transcriptionally regulated by miR-30, which suppressed colorectal cancer growth both in vitro and in vivo, suggesting that miR-30a can serve as another potential prognostic biomarker for CRC, may also have promise as therapeutics. (Liu *et al.,* 2015)

Explore further: Serum miR-21 putative biomarker for colorectal cancer

The Next Stage of CRISPR: RNA Editing

Researchers at the prestigious Salk Institute are reporting that they have managed to map the molecular structure of a **CRISPR enzyme** that could allow scientists to **more precisely manipulate functions within cells.** Over the past several years, **CRISPR-Cas9** has seized the public imagination for its ability to **edit genetic code** in a way that may **correct defects inside individual cells – potentially healing mutations** and preventing the advent of many illnesses.

Specifically, **Cas9 enzymes** act sort of like scissors, spinning away piececes of genetic code and swapping them out with a **replacement.** These enzymes target **DNA,** which is the fundamental building block for the development of an organism, and there are growing concerns that using the enzyme to **essentially reprogram the DNA of a cell** may cause more harm than good. Research suggests that's only the tip of a Titanic-sized iceberg: CRISPR-Cas9 can cause significantly greater genetic havoc than experts thought, the study concludes, perhaps enough to threaten the health of patients who would one day receive **CRISPR-based therapy.**

The results come hard on the heels of two studies that identified a related issue: some **CRISPR'd cells** might be **missing a key anti-cancer mechanism** and therefore be able to **initiate tumors.** The new findings from the Salk Institute, published in the journal Cell, provide **the detailed molecular structure of CRISPR-Cas13d,** an enzyme that can **target RNA instead of DNA.**

Once thought to just be the delivery mechanism for instructions **encoded in DNA** for cell operations, **RNA is known to carry out biochemical reactions like enzymes,** and serve their own regulatory functions in cells. By identifying an enzyme that can **target the mechanisms by which cells operate,** rather than the overall plan for cellular function, scientists should be able to come up with even more highly **refined** treatments with fewer risks.

Having **editing tools** can allow scientists to **modify a gene's** activity without making permanent – and **potentially dangerous – changes to the gene itself** seems like a good option to explore.

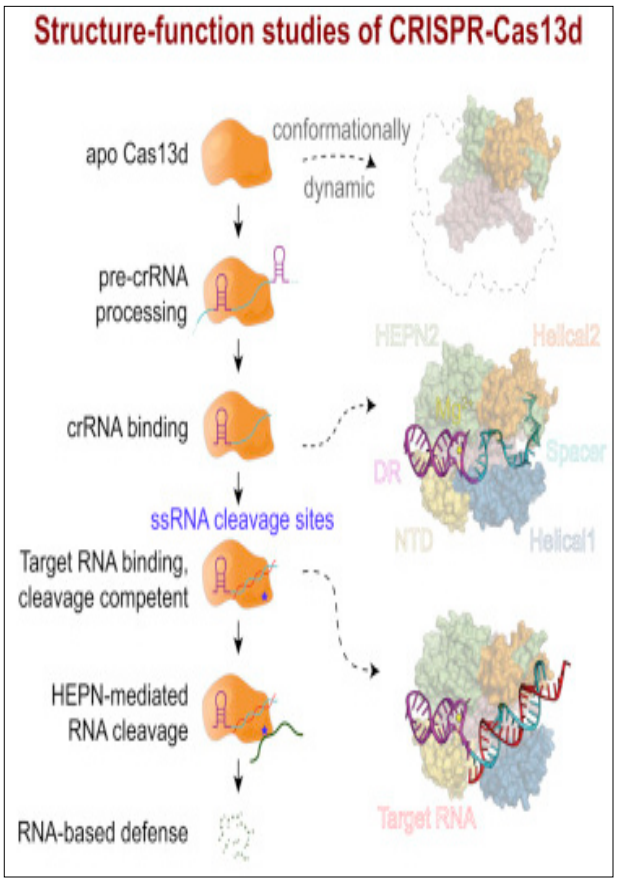
DNA is constant, but what's always **changing are the RNA messages copied from the DNA,** says Salk Research Associate Silvana Konermann, a Howard Hughes Medical Institute Hanna Gray Fellow and one of the study's first authors, in a statement. Being able **to modulate those messages by directly controlling the RNA has important implications for influencing a cell's fate.**

Researchers at Salk first identified the family of enzymes they're re calling CRISPR-CAS13d earlier this year and suggested that that **this alternate system could recognize and cut RNA.** Their first work was around dementia treatment, and the team showed that the tool could be used to **correct protein imbalances** in cells of dementia patients.

In their previous paper, they discovered a new CRISPR family that can be used **to engineer RNA directly inside of human cells,** said Helmsley-Salk Fellow Patrick Hsu, who is the other corresponding author of the new work. They have been able to visualize the structure of Cas13d, we can see in more detail how the enzyme is guided to the RNA and how it is able to **cut the RNA.** These insights are allowing us **to improve the system** and make the process more effective, paving the way for **new strategies to treat RNA-based diseases.** (Zhang & Konermann *et al.,* 2018)

CRISPR-Cas13d

CRISPR-Cas endonucleases directed against foreign nucleic acids mediate prokaryotic adaptive immunity and have been tailored for broad genetic engineering applications. Type VI-D CRISPR systems contain the smallest known family of single effector Cas enzymes, and their signature Cas13d ribonuclease employs guide RNAs to cleave matching target RNAs. To understand the molecular basis for Cas13d function and explain its compact molecular architecture, resolved cryoelectron microscopy structures of Cas13d-guide RNA binary complex and Cas13d-guide-target RNA ternary complex to 3.4 and 3.3 Å resolution, respectively. Furthermore, a 6.5 Å reconstruction of apo Cas13d combined with hydrogen-deuterium exchange revealed conformational dynamics that have implications for RNA scanning. These structures, together with biochemical and cellular characterization, provide insights into its RNA-guided, RNA-targeting mechanism and delineate a blueprint for the rational design of improved transcriptome engineering technologies. (Zhang & Konermann *et al.*,, 2018).



Results

Vegetables can Reduce the Risk of Bowel Cancers

Cruciferous vegs including cabbage, broccoli and kale are very good for the gut, but a detailed explanation has been elusive. The team at the Francis Crick Institute found **anti-cancer chemicals were produced as the vegetables were digested.** Cancer Research UK said there were plenty of reasons to **eat more veg.**

The work focused on **how vegetables alter the lining of the intestines,** by studying mice and miniature bowels growing in the lab. Like the skin, **the surface of the bowels is constantly being regenerated** in a process that takes four to five days. But this **constant renewal** needs to be tightly **controlled,** otherwise it could lead to **cancer** or **gut inflammation.** The work published in the journal Immunity, showed chemicals in cruciferous vegetables were vital.

The researchers investigated a chemical called **indole-3-carbinol,** which is produced by **chewing such vegetables.**

We must make only sure they're **not overcooked,** no soggy broccoli, said researcher Dr Gitta Stockinger. The chemical is modified by **stomach acid** as it continues its journey through the digestive system. In **the lower bowel,** it can change the behavior of **stem cells,** which **regenerate the bowel lining,** and of **immune cells** that **control inflammation.**

The study showed **diets high in indole-3-carbinol protected** the mice **from cancer.** Without **the protective diet,** the gut cells **divided uncontrollably.** Dr Stockinger added that even when the mice **started developing tumors** and researchers switched them to the appropriate **diet, it halted tumor progression.**

Signs of bowel cancer include persistent:

- blood in the stools,
- changes in bowel habits, such as going to the toilet more often,
- tummy pain, bloating or discomfort.

Dr Stockinger said the findings were a cause for optimism. She has **reduced the amount of meat** she eats and now consumes a **lot more vegetables.** With this study, we have **the molecular mechanism** about **how this system works.**

Prof. Tim Key, from Cancer Research UK, said that this study in mice suggests it's **not just the fibre contained in vegetables** like broccoli and cabbage that help **reduce the risk of bowel cancer,** but also molecules found in these vegetables too. Probably, further studies will show the molecules in these vegetables have **the same effects in humans.** Also today there are already plenty of good reasons to **eat more vegetables. (**Gallagher, 2018)

Genotoxic Effect of Bile Acids in Colon Cancer

Colorectal cancer is the second cause of death for tumors worldwide. Among the risk factors for this disease **the dietary habits** seem to have a **pivotal role.** An **elevated intake of fats** causes a **high release** in **the gut lumen of bile acids** that are **positively correlated** with **colorectal cancer,** since they act as **detergents** and **proliferation promoters.** It was evidenced that bile acids can also be able to **induce DNA damage.**

The genotoxicity of **deoxycholic acid (DCA)** and **chenodeoxycholic acid (CDCA)** has been evaluated in human normal **colonocytes** and in **tumor cells.** The involvement of **reactive oxygen species (ROS)** and **the oxidative DNA damage** assessed. In addition, **the protective effect** exerted by both two well-known **antioxidants** were commonly present in **the diet. Beta-carotene, alpha-tocopherol** and **butyrate,** which are known to be involved in the regulation of cellular functions.

The DNA damage can be evaluated by the "comet assay" or single cell gel electrophoresis (SCGE) both in its conventional use and by the EndonucleaseIII modified method, which allow to detect the presence of **oxidized pyrimidines.**

Bile acids (CDA and CDCA) resulted **genotoxic** on both normal and tumor human colon cells. The inclusion of the endonuclease III digestion step in the comet assay demonstrated that bile acids **induced an oxidative DNA damage.** In addition, treatment of colonocytes with bile acids in the presence of **the antioxidants**

(beta-carotene, alpha-tocopherol) and **Na-butyrate** caused a **reduction of DNA damage.**

Results suggest that **bile acids** are **involved in tumor initiation** by **inducing a DNA oxidative damage,** and so add further evidences to **the preventive properties of antioxidants** present in **the Mediterranean diet.** (Rosignoli *et al.,* 2008)

Colon Carcinogenesis through Mechanism of Metastases Caused by the QEE

Sporadic colon cancer is caused **predominantly by dietary factors.** We can select **bile acids** since **high levels of hydrophobic bile acids accompany a Western-style diet,** and play a key role in **colon carcinogenesis.** Bile acid-induced **stresses** cause **cell death** in susceptible cells, contribute to **genomic instability** in surviving cells, **impose Darwinian selection on survivors** and **enhance initiation** and **progression to colon cancer.** The most likely **major mechanism** by which hydrophobic bile acids **induce stresses on cells is the Quantum Entanglement Entropy (QEE) metastases through the DNA damage, endoplasmic reticulum stress, and mitochondrial damage.** Persistent exposure of colon epithelial cells to hydrophobic bile acids can result by **QEE** in the activation of **pro-survival stress-response pathways,** and **the modulation** of **genes/proteins** associated with **chromosome maintenance** and **mitosis. The mechanism** of **QEE** by which hydrophobic bile acids contribute to genomic instability include **oxidative DNA damage, p53** and other **mutations, micronuclei formation** and **aneuploidy.** Bile acids and oxidative stress **decrease DNA repair proteins,** an **increase in DNA damage** and **increased genomic instability** through this mechanism of **metastases** caused by **Quantum Entanglement Entropy.** This process provides a **mechanistic explanation** for **the important QEE link** between a **Western-style diet** and associated **increased levels of colon cancer.** (Skopec I., 2018, Skopec IV., Payne *et al.,* 2008)

Dichotomous Correlations of Cancer HYBRID Adaptation

One prevalent description of translational medicine, first introduced by the Institute of Medicine's Clinical Research Roundtable, highlights *two roadblocks* (i.e., distinct areas in need of improvement): *the first translational block (T1)* prevents basic research findings from being tested in a clinical setting; *the second translational block (T2)* prevents proven interventions from becoming standard practice.

An important role in the processes of *adaptation and masking* in humans is playing also *the immune system. The innate* immune system functions as an *interpreter* of tissue damage and provides a *first line of defense,* also *translates the information* to other repair and defense systems in the body by stimulating angiogenesis, wound repair, and activating *adaptive immunity.* It is appropriate to consider *autophagy* a means for *programmed cell survival* balancing and *counter-regulating apoptosis.* Autophagy seems to have a *dichotomous role* in *tumorigenesis* and *tumor progression.*

Two other attributes play a similarly *paradox* role. The first involves major *reprogramming* of cellular *energy metabolism* in order to support continuous cell growth and *proliferation replacing the metabolic program* that operates in most normal tissues. The second involves *active evasion* by cancer cells from attack and *elimination* by immune cells. This capability highlights *the dichotomous correlations* of an immune system that both *antagonizes* and *enhances* tumor development and progression. (Hsu *et al.,* 2009)

Evidence began to accumulate in the late 1990s confirming that *the infiltration of neoplastic tissues* by cells of the immune system serves *counter-intuitively* to *promote tumor progression.* (Skopec III., 2015)

The Bipolar Nature of Cancer: HYBRID, Twofaced New Main Law of Nature

The quantum entanglement is a basis of *twofaced reality* in which we are living our lives. From this reality are outgoing also *the science and healthcare too.* Altough **metastasis** is important for **systemic correlations expansion** (as **in tumors**), it is a *highly dichotomous process,* with millions of cells being required to **disseminate to allow for the selection of cells-correlates aggressive enough to survive the metastatic cascade.** To quantify the dynamics of **metastasis of correlations** development, we need

look at **the coincidences of metastases** in terms of ***co-occurrence*** at every point of time. To quantify co-occurrence we can use **the φ -correlation between *dichotomous variables*** defined as:

$$\frac{N_X(t)C_{ij}(t) - m_i(t)m_j(t)}{\sqrt{m_i(t)m_j(t)[N_X(t) - m_i(t)][N_X(t) - m_j(t)]}}$$

where $C_{ij}(t)$ is the number of co-occurrence at time t. Than i and j represent particular site of metastasis, X represents the primary correlations type. **The pair-wise correlations (coincidences) between metastasis network links** for every primary correlations types and lead to ***the correlation coefficient matrix.***

The dichotomous correlations of the adaptation may be caused also by ***the Quantum Entanglement Relative Entropy*** as a measure of distinguishability between two *quantum states* in the same Hilbert space. The relative entropy of two *density matrices* p_0 and p_1 is defined as.

$$S(p_1|p_0) = tr(p_1 \log p_1) - tr(p_1 \log p_0)$$

When p_0 and p_1 are reduced density matrices on a spatial domain *D* for two states of a *quantum field theory* (QFT), implies that $S(p_1|p_0)$ increases with the size of *D.* Than $\Delta S_{EE} = -tr(p_1 \log p_1) + tr(p_0 \log p_0)$ is ***the change in entanglement entropy*** **across *D* as one goes between the states.**

When the states under comparison are close, *the positivity* is saturated to *leading order*:

$S(p_1|p_0) = \Delta\langle H_{\mathrm{mod}}\rangle - \Delta S_{EE} = 0$. (Skopec II., 2018)

The problem of conventional adaptation may be given by a definition of static, deterministic world. **The proliferative correlations lead to *the resonances between the degrees of freedom.*** When we increase the value of energy, we **increase the regions where *randomness prevails.*** For some critical value of energy, chaos appears: over time we observe *the exponential divergence of neighboring trajectories.* For fully developed chaos, **the cloud of points** generated by a trajectory leads to *diffusion.* (Prigogine, 1997) Here we must as first formulate a **new *Main Natural Law: the HYBRID Quantum Entanglement Entropy (HQEE).*** (Skopec III., 2015) **Through above resonances the QEE is causing a *metastasis of correlations,* antagonistically intertwining (coincidences) all types of potentially conflicting interests in cancer.**

Conclusions

All Humans Are Pre-Programmed With Innate Code of Carcinogenesis

Cancer is a terrifying **Innate Disease** that researchers around the globe are obsessively working to cure. As it is known, scientists from the USA have made a breakthrough discovery related to how cells replicate in cancer patients and how to put a stop to the process, including how to reverse a tumor. The finding was published in Nature Cell Biology, represents an unexpected **new biology** that provides **the code, the software for turning off cancer,** said the study's senior investigator, Panos Anastasiadis, Ph.D., chair of the Department of Cancer Biology on Mayo Clinic's Florida Campus.

Evidences

The code was unrevealed by the discovery that **adhesion proteins – the glue** that keeps cells together – **interact** with **the microprocessor,** a **key player** in the production of molecules called **microRNAs (miRNAs). The miRNAs orchestrate** whole **cellular programs** by **simultaneously regulating expression of a group of genes (co-occurence).**

The investigators found that when normal cells come in contact with each other a specific subset of **miRNAs suppresses genes promoting cell growth. Adhesion can be disrupted in cancer cells,** these miRNAs becoming **misregulated** and cells **grow out of control.** Since, **restoring** the normal miRNA levels in cancer cells can **reverse the aberrant cell growth,** but under the law of **co-occurrence coded by the QEE,** aberrant cell growth **begin at the same time in some other place** regulated by the microprocessor miRNAs.

The study brings together two so-far unrelated research fields – **cell-to-cell adhesion** and **miRNA biology –** show a long-standing problem about the role of **adhesion proteins** in cell behavior, said the study's lead author Antonis Kourtidis, Ph.D., a researcher associate in Dr. Anastasiadis lab. It uncovers a new strategy for cancer therapy.

That problem arose from conflicting reports about **E-cadherin** and **p120 catenin – adhesion proteins**

essential for normal epithelial tissues to form and which have long been considered to be tumor suppressors. This hypothesis didn't seem to be valid today. **Both, E-cadherin** and **p120 catenin** are still **present in tumor cells** and **required** for their **progression.** That lead to believe that these molecules have **two faces:** a **good one**, maintaining **the normal behavior** of the cells, and a **bad one** that **drives tumorigenesis** (through the **effect of Majorana Fermions).**

The question is what was regulating this behavior ? To answer this, the researchers studied a new **protein** called **PLEKHA7,** which associates with E-cadherin and p120 catenin only **at the top,** of **the apical** part of normal polarized epithelial cells. The investigators discovered that **PLEKHA7 maintains the normal state** of the cells, via a set of **miRNAs,** by **tethering the microprocessor to E-cadherin** and **p120.** In this state, E-cadherin and p120 exert their **good tumor suppressor** sides.

When this **apical adhesion complex** was **disrupted** after **loss of PLEKHA7,** this set of **miRNAs** was **misregulated,** and the E-cadherin and p120 **switched (QEE)** sides to **become oncogenic.** (Mayo Clinic, 2015)

Loss of the apical PLEKHA7-microprocessor complex is an **early,** and **universal event in cancer (pre-programmed, encoded).** In the vast majority of human tumor samples this apical structure is **absent,** although E-cadherin and p120 are still **present.** This produces the equivalent of a speeding car that has a lot of gas (**the bad p120)** and no brakes (**the PLEKHA7-microprocessor complex).**

By administering the affected miRNAs in cancer cells **to repair** their normal levels, we should be able **to introduce the brakes** and achieve normal cell function. We must learn that **the universal code of carcinogenesis is in all humans pre-programmed, innate.**

Above finding means that **cancer is Not an Error, but is an Innate Systemic Disease. All humans are pre-programmed to carcinogenesis.** From this theorem follows that we must this **Innate Cancer not "restore", but to repair.**

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