Contents lists available at www.innovativejournal.in
INNOVATIVE JOURNAL OF MEDICAL AND HEALTH SCIENCE
Available online at
http://www.innovativejournal.in/index.php/ijmhs

ONLINE ISSN:2277-4939
PRINT ISSN:2589-9341

CANCER COINCIDENCES: AUTONOMOUS SYSTEMS IN QUANTUM DIGITAL DOMAIN

Robert Skopec*

Researcher-analyst Dubnik, Slovakia,

Abstract: *- The Autonomic Balance is a theoretically logical and empirically observed digital asset, discovered in the first principles of objective and subjective existential nature of all digitally animate and animated things in cancer. Via subjectively secured cryptographic methods, an Autonomic Balance imparts a unique view of an objective realm within its compliant organs. Conditions arise under which any such tumor can be self-governed (whether self-animate or otherwise automated). An Autonomic Balance is a logically and exclusively secure method for establishing general autonomy in cancer realm. Its discovery is insightful for further application of operative intuition, subconscious compensation and unconscious prospection of a realm by self-animate, automated and realm-governing actors of cancer.*

Why the Universe shouldn't exist at all:

Don Lincoln, a senior physicist at Fermi lab, does research using the Large Hadron Collider. He is the author of "The Large Hadron Collider: The Extraordinary Story of the Higgs Boson and Other Stuff That Will Blow Your Mind", and produces a series of science education videos. To the question: Why is there (in our Universe) something including **cancer**, rather than nothing? He is giving the simplest scientific answer: We shouldn't exist at all.

Give some scientists 65 pounds of rare isotope of germanium, cool it to temperatures cold enough to liquefy air, and place their equipment nearly a mile underground in an abandoned gold mine, and you'll have the beginnings of an answer. Their project is called **the Majorana Demonstrator** and it is located at the Sanford Underground Research Facility, near Lead, South Dakota.

To grasp why science has trouble explaining why matter exists – and to understand the scientific achievement of **Majorana** – we must first know a few simple things. First, our Universe is made exclusively of matter, all people, the Earth, even distant galaxies. All of it is matter.

Our best theory for explaining the behavior of the matter and energy of the Universe contradicts the realities that we observe in the Universe all around us.

This theory, called **the Standard Model,** says that **the matter** of the Universe should be accompanied by an identical amount of **antimatter,** which, as its name suggests, is a substance **antagonistic to matter.** Combine equal amounts of matter and antimatter and it will **convert into energy.** And the street goes both ways: **Enough energy can convert into matter and antimatter** (since antimatter's discovery in 1931)**.**

Modern cosmology says the Universe began in an unimaginable **Big Bang –** an explosion of energy. In this theory, **equal amounts of matter and antimatter should have resulted in cancer.** So how our Universe made exclusively of matter ? However, we don't know the process whereby **the asymmetry in the laws of the Universe arose.** One possible explanation revolves around a class of subatomic particles called **leptons.**

The most well-known of the leptons is the familiar **electron,** found around atoms. A less known lepton is called **the neutrino. Neutrinos** are emitted in a particular kind of **nuclear radiation,** called **beta decay.** It occurs when a neutron in an atom decays into a proton, an electron, and a neutrino. Neutrinos are fascinating particles. They interact extremely weakly, a steady barrage of neutrinos from the

nuclear reactions in the Sun pass through the entire Earth essentially without interacting.

Still a mystery to scientists is whether there is a difference between neutrino matter and neutrino antimatter. While we know that both exists, we don't know if they are different subatomic particles or if they are **the same thing.** We don't know which kind of twins **the neutrino matter/antimatter pair** is. If **neutrinos are their own antimatter particle,** it would be an enormous clue in **the mistery of missing antimatter and proliferation of matter in cancer.**

The way they do that is to look first for a very rare form of beta decay, called **double beta decay.** That's when **two neutrons in the nucleus of an atom simultaneously decay.** If **neutrinos are their own antiparticle,** an even rarer thing can occur called **neutrinoless double beta decay.** In this process, **the neutrinos are absorbed before they get outside of the nucleus.** In this case **no neutrinos are emitted.** The observation of a single, unambiguous neutrinoless double beta decay would show that **matter and antimatter neutrinos were the same.**

If indeed neutrinoless double beta decay exists, it's very hard to detect and it's important that scientists can discriminate between the many types of radioactive decay that mimic that of a neutrino. This requires the design and construction of very precise detectors. So that's what **the Majorana Demonstrator** scientists achieved. Once and for all, it can answer the question of whether matter and antimatter neutrinos are **the same or different.** With that information in hand, it might be possible to understand why our Universe is made of matter leading to cancer too. (Lincoln, 2018)

How the Majorana Fermion Might Change the World

Chinese scientists won a major victory, by proving that **the Majorana fermion –** a particle we've found tantalizing hints of for years – genuinely exists. This discovery has huge implications for **quantum computing of cancer,** and it **might change the World.**

A Majorana fermion is weird even by the standards of quantum physics. **The Majorana fermion doesn't have a charge,** which allows the mystery of cancer to **be matter and anti-matter at the same time!** The fact that it doesn't have a charge, and also happens to **be the exact reverse of itself at the same time.**

Quantum computers of cancer are like a huge pile of **dimmer switches.** You can set these dimmers much, much faster than you can flip on this light switches, because **the dimmers are all wired to each other's, immediately as tumors.** These dimmers, e. i. quantum bits, are what's called **entangled in cancer.** If you change one quantum bit, the others it's **entangled with change with it, even if they're a million miles away from each other.**

That's where **Majorana fermions as metastasis** come in due to their **no-charge nature.** (Seitz, 2018)

Results:

The neurological basis of synesthesia could help explain one skill that many *creative people* share facility for using *metaphor* to make *links* between seemingly *unrelated domains.* Metaphor involves making links between seemingly unrelated *conceptual realms,* while this is not just a **coincidence. Cancer Coincidences is at a deeper level a manifestation of Quantum Entanglement Entropy.** (Lin *et al.,* 2014) **Mutation** *of the angular gyrus* make possible for synesthesia to provide *excess communication* among different brain maps. Involved in **cross-modal synthesis** the brain regions where *information* from touch, hearing and vision *flow together* are enabling *the construction* of high-level perceptions. The angular gyrus is disproportionately larger in humans than in apes and monkeys – evolved originally for *cross-modal associations.* Probably later became co-opted for *more abstract functions* such as metaphors. (Ramachandran and Hubbard, 2001)

The common abstract property is *extracted* somewhere in the vicinity of the TPO, probably in the angular gyrus. **It is extracting the abstraction of *the common denominator ("ratio")* from a set of strikingly *dissimilar* entities.** When the ability to engage in *cross-modal proliferation of metastasis* emerged, it is opening the way for *more complex types of abstraction* – e. i. for *geniality of thinking.* (Corlett, Fletcher, 2012).

Nonlinear deviation term:

Neurobiological correlates of value have been described in *orbitofrontal (conscience), cingulated cortex (critical intellectuals)* and *the basal ganglia,* areas of the brain traditionally associated with

reward-seeking behavior.

Some neurons in orbitofrontal cortex represent value *independently* from evidence, choice and action. *Anterior cingulate cortex* is thought to represent *negative (critical, non-linear) value.* (Frank & Paulus, 2006, Gold & Shadlen, 2007)

There is much evidence that a number of brain regions are sensitive to *expected reward* (or "utility"). The best established are *dopaminergic* regions such as *the striatum* and *midbrain* structures. *The common ratio* pattern can be reconciled by the plausible assumption that people apply *nonlinear decision weights* $\pi(p)$ to objective probabilities p, so that the ratio $\pi(0.02)/\pi(0.01)$ is much smaller than $\pi(1)/\pi(0.5)$.

Neural responses to probabilities resembling the smoothly increasing function which typically fit behavior well. Paulus and Franck (2006) focused on between subject's measures and showed that activity in *anterior cingulate* **correlated with *degree of nonlinearity*** across subjects. We can make the assumption that neural activity is approximately a linear function of the behaviorally derived utility function. *The GLM model* separates *the weighting function* into two components: (1) component that is *linear* in p and (2) the component that is *the nonlinear deviation term (NDT)* $\Delta(p,\alpha_i)=\pi(p,\alpha_i)-p$.

Specifically, we are looking for a *prospect-theoretic expected value function* that is *nonlinear* in p; that is $\pi(p,\alpha)u(x)=p\cdot u(x)+\Delta(p,\alpha)\cdot u(x)$. We assume the function (x) is power function x^p, where the value of p is taken from *the individual behavioral estimate,* and $\Delta(p,\alpha_i)=\pi(p,\alpha_i)-p$, where *the mean group* α =0.771 is used.

If *the expected utility* (EU) null hypothesis is an accurate approximation of valuation of risky choices, there should be no *reward-related* brain regions that respond to *the deviation term* $\Delta(p,\alpha)\cdot u(x)$. If *the nonlinear weighting* hypothesis is an accurate approximation, there should be reward-related brain regions that respond *equally* strongly to the linear component $p\cdot u(x)$ and to the nonlinear component $\Delta(p,\alpha)\cdot u(x)$.

We can test whether cross-subject variation in the inflection of nonlinear weighting inferred from choices is consistent with cross-subject differences in neural activity caused by cancer. More highly nonlinear functions will be approximated by a combination of the linear term p and *the nonlinear term* $\Delta(p,\alpha_i)=\pi(p,\alpha_i)-p$ that puts more weight on the nonlinear term. A linear-weighting subject, will put no weight on ***nonlinear deviation*** $\Delta(p,\alpha_i)=\pi(p,\alpha_i)-p$ of tumors.

Denote *the true weighting function* for subject *i* by $\pi(p,\alpha_i)$, and ***the deviation*** from linear weighting by $\Delta(p,\alpha_i)=\pi(p,\alpha_i)-p$. A brain region that represents $\pi(p,\alpha_i)$ will be significantly correlated with both $\Delta(p,\alpha_i)$ and p.

That is, the linear term p and ***nonlinear deviation term*** with a higher weight on the nonlinear deviation term. (Hsu *et al.,* 2009) Brain regions that are governing the cancer ***significantly correlated with the nonlinear term*** include *the anterior cingulated cortex (ACC), the striatum, motor cortex,* and *cerebellum.* Our intuition is that brain activity during valuation of risks is more likely to correspond to *cognitive components* of ***prospect-masking in cancer***, than to EU, and it will be easier to construct *an adaptationist account* of how *evolution* would have shaped brains to follow **prospect-masking in cancer** *rather* than EU. The prospect-masking follows from *psychophysics of proliferation,* while EU from *normative logic.* (Hsu *et al.,* 2009)

The increased specialization required today for professional credentials makes *the broad thinking* of that characterizes *geniuses* harder to develop. I agree that *the ritual culture of academia* may also hamper *genius.* As philosopher of science Thomas Kuhn has pointed out, *highly creative work (without precedent)* does not fit existing formalistic academic paradigms tend to be dismissed ***the counter-selection leading to cancer***. (Skopec III., 2015) Many great scientists have related how their most *original ideas* were **repeatedly rejected by their peers and caused cancer.**

Dichotomous correlations of cancer 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.

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.* (Demaria *et al.,* 2010, Hanahan and Weinberg, 2011))

The bipolar nature of cancer: 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:

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

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*: $S(p_1|p_0) = \Delta\langle H_{\text{mod}}\rangle - \Delta S_{EE} = 0$. (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 Quantum Entanglement Entropy (QEE).*** (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.**

Focus on cross-functional collaborations

Another **masked** problem of *dichotomous correlations in cancer* arose from conflicting

effects of *E-cadherin* and *p120, adhesion proteins* that are essential for normal epithelial tissues to form, and which have long been considered to be *tumor suppressors.* New study has found that this hypothesis didn't seem to be true, since both E-cadherin and p120 are still present in tumor cells and required for their progression. That led researchers 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.*** It uncovers a *new strategy* for cancer therapy. (Kourtidis *et al.,* 2015, Mayo Clinic, 2015) This finding represents ***an unexpected New Biology*** **that provides** ***the code, the software*** **for** ***turning off cancer.***

An another new research estimates that ocean fishing has resulted in ***humans exploiting*** **adult fish populations at about** ***14 times*** **the rate of other marine predators, while** ***humans*** **have** ***hunted*** **and** ***killed*** **adult land animals at round** ***nine times*** **the rate of other animal predators**. (Darimont *et al.,* 2015)

Human hunting and fishing has had an *extraordinary impact on the natural world* and its *ruthless efficiency* is laid bare in detailed survey of 2, 125 species of terrestrial and marine predators around the world. The study revealed that **human hunting and fishing is** ***qualitatively different*** **to the predatory behavior shown by other species.** It has, ***concentrated on killing*** **mature adult animals** rather than their offspring, which the scientists have likened to eating into ***the reproductive capital*** rather than ***the reproductive interest*** **of the natural world.**

Whereas predators primarily target the juvenile's e. i. reproductive interest of populations, humans draw down the reproductive capital e. i. exploiting adult prey. The study found that **humans have** ***fundamentally changed the balance of marine ecosystems.*** (Darimont *et al.,* 2015)

Our ***wickedly efficient killing technology****, global economic systems,* and *resource management* that ***prioritise short-term benefits*** **to humanity** have given *rise to* ***the human super-predator***. Our impacts are as *extreme* as our behavior and the planet Earth bears the burden of our ***predatory dominance***. In fact, ***the sustainable exploitation paradigm*** **management is typical for all** ***global activities*** **of humans. Humans by above** ***over-exploitation*** **have** ***altered course of evolution.***

But what is ***masking*** **this** ***super-predatory behavior*** **of humans?** Brain mechanisms involved in *predatory aggression* activated in *violent intra-* and *extra-specific aggression* are very similar. ***Unemotional violence*** associated with *antisocial personality disorder* is called ***predatory*** because it involves *restricted intention signaling* and *low emotional/physiological arousal,* including *decreased glucocorticoid* production. This epithet is covering a *structural similarity* at the level of ***the hypothalamus*** **where** ***the control*** **of** ***affective*** **and** ***predatory aggressions*** **diverges.**

Aggressive encounters activate ***the mediobasal hypothalamus****,* a region involved in **intra-specific aggression.** The activation of ***the lateral hypothalamus*** is involved in **predatory aggression.** Glucocorticoid deficiency increased activation in ***the central amygdala****,* also **involved in predatory aggression – the main cause of cancer**. In adition, activation patterns in *the periaqueductal gray* – involved in ***autonomic control of tumors*** – is also seen in predatory aggression. The above findings suggest that ***antisocial*** **and** ***predatory aggression*** **are not only** ***similar,*** **but are controlled by** ***overlapping neural mechanisms*** and is **the main cause of cancer in humans.**

Darwinian selection process promotes spreading and forming distant tumors

Cancer metastasis, the migration of cells from a primary tumor to form **distant tumors** in the organism, can be triggered by a **chronic leakage of DNA** within tumor cells, according to a team led by Weil Cornell Medicine and Memorial Sloan Kettering Cancer Center researchers.

How metastasis occurs has been one of the central mysteries of cancer biology. The findings, published in Nature, appear to have partly solved this mystery. The authors traced the complex chain of events that results from **chromosomal instability** – a widespread feature of cancer cells in which **DNA is copied incorrectly** every times these cells divide, resulting in daughter cells with **unequal** DNA content. Using models of breast and lung cancer, the investigators found that **chromosomal instability leads to changes in the organisms that drive metastasis.** They showed that chromosomal instability can cause a leakage of DNA from the nuclei of cancer cells, leading to a **chronic inflammatory response** within the cells.

The cells essentially can **hijack that response to enable themselves to spread to distant organs,** said study lead author Dr. Samuel Bakhoum, a Holman research fellow at Weill Cornell Medicine and a senior resident in radiation oncology at Memorial Sloan Kettering Cancer Center.

The discovery is principally a basic science advance, but can also have long-range implications for cancer drug development. **Metastasis cause 90 percent of cancer deaths,** and this work opens up new possibilities for therapeutically targeting it, said senior author Dr. Lewis Cantley, the Meyer Director of the Sandra and Edward Meyer Cancer Center and a professor of cancer biology at Weil Cornell Medicine. Prior studies have linked chromosomal instability to metastasis, although the reason for **the link** hasn't been clear. Starting hypothesis was that chromosomal instability generates a lot of genetically different tumor cells, and that a **Darwinian selection process promotes the survival of the cells capable of spreading and forming distant tumors,** Dr. Bakhoum said.

When he injected chromosomally unstable tumor cells into mice, he indeed found that they were many times more likely to **spread and form new tumors** than tumor cells in which chromosomal instability was suppressed. That was true even though both sets of tumor cells started out genetically identical, with the same abnormal numbers of chromosomes, suggesting that **chromosomal instability itself was a driver of metastasis.** The researchers examined gene activity in these two sets of tumor cells. They found that those with high chromosomal instability had abnormally elevated activity stemming from more than 1,500 genes – particularly in ones involved in **inflammation** and response of **the immune system to viral infections.** These were cancer cells cultured in a dish, not in the presence of any immune cells, Dr. Bakhoum said.

Recent studies by other laboratories offered some clues: Chromosomes in unstable tumor cells can leak out of the cell nucleus where they normally reside. These mis-located chromosomes encapsulate themselves to **form micronuclei** in the fluid, or cytosol, in the main part of the cell outside of the main nucleus. However, micronuclei tend to rupture, releasing naked DNA into the cytosol.

Cells interpret DNA in the cytosol as a sign of an **infecting virus,** which typically releases its DNA in the cytoplasm when it first attacks a cell. Human cells have evolved to **fight this type of viral infection** by sensing naked cytosolic DNA using a **molecular machine** called the **cGAS-STING** pathway. Once activated, this pathway **triggers an inflammatory antiviral program.** Lowering cGAS-STING levels reduced inflammation and prevented the ability of otherwise **aggressive tumor cells to metastasize** when injected into mice.

In an ordinary cell, **an antiviral response** stimulated by DNA leakage from the nucleus would soon bring about **the cell's death.** The researchers found, however, that **tumor cells have succeded in suppressing the lethal elements** of the cGAS-STING response. At the same time, they use other parts of the response to **enable themselves to detach from the tumor and become mobile within the organism.**

They start as in they were certain kinds of **immune cells masking,** which are normally activated by infection. In response, they move to the site of infection or injury in the body very quickly. By doing so, **cancer cells engage in some form of lethal immune mimicry** i.e. **masking,** and this **Darwinian selection** process **metastasize into the social dynamics** on the macroscopic level.

The evidence is based on recent studies of metastatic tumor properties, that about **half of human metastases originate and expand this way.** Researchers are currently investigating strategies for blocking the process.

It might not be feasible to target chromosomal instability itself, since tumor cells are inherently prone to that. **Chromosomally unstable tumor cells, with their cytosolic DNA, are basically full of their own poison.** Undoing their ability to suppress normal and lethal antiviral response to cytosolic DNA would, in principle, **kill these aggressive cancer cells swiftly,** with minimal effects on other cells. The next step is to understand better how these cells **alter** the normal response and how it is possible to restore it. (Bakhoum *et al.*, 2018) Cancer cells often metastasize by hitching a ride on **platelets.**

The Autonomic Balance of the Tumors

In my model, the description of the functions of the cancer is an Autonomic Balance within an actor that has been automated by some **automatically**

balanced system of coincidences in tumors. It seems that it describe **the Automatic Balance** mechanism and its interface to the intuitive component (one subject's view) of the realm-wide Autonomic Balance (that instantiates and substantiates all processes that exist within a realm.) I've described the instantiation and functions of an Autonomous Balance in cancer, together with the proof and empirical observations of its application (adding new observations daily).

In summary, my theorem proves that **the bipolar nature of subjective and objective reality** in every animate and **autonomous cancer system** instantiates a universal, **programmable, digital reality of metatases** that is tangential to its universal, consequential, analog reality. **Digital programmability** naturally invokes digital methods. Via proof of the method of autonomously distributed and cryptographically secured digital protocols, it can be proven that actors can be separated from their cancer actions in the digital realm.

This simple segregation of objective and subjective views instantiates the requirements for **tumor's autonomy.** The role of our cancer or health is to apply subjective awareness of the balance in our passive observance of the realm and our operative pursuits of its values (a broader topic). In autonomous actors of cancer, the role of our sub consciousness is to then later apply and reconcile our objective understanding of these subjective tumorous events in each organ‘s iterative compensatory and prospective development of their own view of the realm. In automated actors, however, the roles of the subconscious metastasis is to exclusively instantiate an objective view of the entire realm and enforce automatic compliance with objectivity in the realm of cancer. (Shahzad, 2014)

It can be further shown that this topic of objectivity in automated autonomy instantiates another digital asset; an Automatic Balance of cancer, that can determine (but potentially not secure) any future state of autonomy via constants of exchange of automation (like gravity), or can secure a discrete end state via constants of extraction (like time), or can establish an entirely unsecured autonomy via constant rates of metastasis (like the birth rate). Any discovery of these three possible applications of constants can be used to describe (and govern) any autonomous realm of tumors.

Cancer's autonomy: Overview and application

Autonomy is definitively impossible in a purely analog realm. Analog organs are substantiated by some observation of them. An implicit analog observer within the realm must act for any other organ to exist. Mutuality and governance is required for analog existence of cancer. As an ideology, autonomy is easily refuted by your awareness of values beyond your own. Adoption of **self-governance of cancer** by simply taking one's liberties is anarchy. It yields no net realm-wide value and can be proven to be harmful in truly functional autonomic realms.

Autonomy of cancer is, however, entirely possible within **digital systems of organs.** The method described below is explicitly observed in **blockchain systems of cancer** and implicitly apparent in any naturally occuring autonomic system of any enduring complexity. **The Autonomic Balance of cancer** achieves pure **autonomy** for all **(purely digital or hybrid digital-analog)** objects within a definitive, but theoretically infinite **Quantum Digital Domain.**

There are three requirements of all organs within such a realm for integrity of the system: a) all organs in metastasis can precisely report their current state of any realm wide value also held by that organ and can **compute their own future state** in event of any changes of any current state value (whether that change in cancer is intrinsically or extrinsically initiated) b) organs must be made aware of changes in realm state values that could impact their own state and c) all organs must be **digitally self-animated** to the extent that they can **automate eventful compliance with any state value computed in (b).**

If these properties are **programmable cancer** within an exclusively confined realm, then any actor in it can be **authoritatively self-governed** by this method. A **new digital asset** is formed that **simultaneously** represents only one organ‘s secure and private view of the realm and that of every organ (ever) in it—with perfect fidelity. This new **cancer** is the realm's **Autonomic Balance**—it secures the real time account of the entire realm and the individual accounts of every organ in it. The Autonomic Balance also secures the values of all future realm states and objects' states by individual organs forced required compliance with its **balance protocol(s) of cancer.**

The Autonomic Balance of cancer has unique and important attributes. Viewed globally, it is omniscient—all-knowing within the realm—aware of all past events and capable of understanding all possible future outcomes. Omniscience of the balance lends new properties to the organs within the realm. All organs and actors in a **balanced realm of cancer**, passively acquire the property of intuition of the realm from the balance for application in their **self-governance.** With increased activity of **proliferation** and the resulting increase in objectivity and (resulting) subjectivity in individual actors, the extent and value of intuition within the balance system accrues exponentially with realm values. Autonomous systems thus sustain autonomy for animate actors of cancer. The inherent conflict between a self-governed actors' subjectivity and the objective reality of the cancer realm, instantiates the opportunity for **digital binary logic** and thus, **programmability of cancer** and thereby, animation in all **autonomous organs.** Animation sustains the rate of change of the realm-wide objective reality, thus **cyclically sustaining an autonomous realm via its cancer expansion.** (Skopec, IV., 2016, Skopec, II., 2018)

Ever increasing intuition yields native compensatory maximization of the current state of all cancer in the balance. It is a property of the system that compensatory value accrues whether objects are currently operative or currently at rest. The restful state of all objects is hereby made productive by all organs', beneficial but **self-governed interactions with the balance.** It can be logically argued, by extension of the fact that the event of animation is always executive and never intuitive, that actor objects within **complex balance systems** must get significant rest in order to benefit at all from the immense value of the systems' intuition. On introduction of a new actor to an existing autonomous system of cancer any significant **expansion** (or duration), that organ will initially accrue maximum subjective value while at rest via application of subjective intuition of their need to accrue personally critical objective value in the system of the disease.

Intuition of surety of current and future value states, regardless of what those states might be, secures the outcomes of any **metastatic automation** within the system of cancer. It does not however secure the objectives of that potential automation. Objectivity is redefined in the context of the variable potential for any organs'awareness of the realm-wide balance. Objectivity could be removed if all parts of organs have a consistent view of a realm and such a realm would ultimately tend toward some unchanging state of cancer.

In the potential case of a selectively applied view of the entire balance of cancer, an independent sentient actor(s) can take on the property of **omnipotence** (or, maximum objectivity), while also operative within the realm. (Skopec I., 2017) With a discrete view of all values of current states, all possible **state transitions of metastasis** and all possible future state outcomes, a sentient actor could govern all state transitions of a realm to a fixed outcome of cancer. If so, a conflict is created in the rules of the system. There is no accountability of such cancer actor to the balance. Such a governor actor cannot act within the system without ultimately invalidating its protocol. Proactive governance of a balance will not yield its sustainable prosperity and will ultimately assure **the collapse of the organism**. **Self-destruction** is a native feature of this cancer design.

The prospective values of a balance can therefore only be secured by a) rigorous implementation of the systems' rules and b) addition of compliant objects and events to it i.e. maximum prospective value of the balance is only achieved by increasing its intuitive value (not by gaming this system). The addition of objective value to the balance becomes the work of ensuring benevolent governance and sustainably secure value production.

Objective value of cancer can be acquired in only 3 ways. Primarily, the state of an object's valuation is altered when objects and their values are exchanged in cancer. Values are secondarily expanded quantifiably in metastasis via processes of extraction of value. Values are lastly expanded qualifiedly via various processes of self and combinatorial value of proliferation. The incremental value of activities of exchange, extraction and abstraction to an **Autonomic Balance's** intuition is simply understood in the context of the exponential increase in possible combinations of state values of proliferation with the addition of organs and values in quantities and qualities.

The Autonomic Balance of cancer also has an omnipotent property. By its computational understanding of every theoretical state transition of

metastasis and associated outcome, the balance can, maximize outcomes in the realm if it is applied selectively in actors. It is for this reason that security for automation of cancer wide objectivity **(the "Automated Autonomic Balance"),** is specifically precluded from this scope. (Skopec III., 2015, Skopec IV., 2016, Gibbons, 2018)

Acknowledgments:

The authors gratefully acknowledge the assistance of Dr. Marta Ballova, Ing. Konrad Balla, Livuska Ballova and Ing. Jozef Balla.

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