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REVIEW ARTICLE

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New Long Distance Quantum Entanglement Entropy's Effects in Cancer

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ABSTRACT

The theory of torsion fields has been formulated owing to the ideas of Eli Cartan who was the first to indicate clearly and definitely that there exist in the Nature the fields generated by the spin angular momentum density. The mentioned system of equations represents in itself the Cartan structural equations of the absolute parallelism geometry. The classical spins be the source of perturbation. We shall conjecture that the impact of the classical spin on the Physical Vacuum will consist of the following. Granted the source has a spin oriented as it is indicated in Fig. IF, then the spins of the phitons that match the orientation of the source spin, will be retaining their orientation. Those phiton spins, that are opposite to the source spin, will experience inversion under the effect of the source. As a result, the Physical Vacuum will transform into the state of a transverse spin polarization. This polarized state can be interpreted as a spin field (S-field), i.e. a field generated by the classical spin. The formulated approach is in tune with the ideas of spin fields as a condensate of fermion pairs.

Keywords: Cartan structural equations, phiton spins and fermion pairs.

INTRODUCTION

Spin polarization states SR and SL contradict the Pauli prohibition. According to the concept of M.A. Markov, under the densities in the neighborhood of the Planck ones, fundamental physics laws may have another aspect, different from the familiar ones. A waiver of the

Pauli prohibition for such a specific material medium as the Physical Vacuum is admittable, perhaps, in no lesser degree than in the quarks concept.

RESULTS

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 subjects 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. 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* $\Delta(p,\alpha_i)=\pi(p,\alpha_i)-p$.

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. (Raichle, 2001)

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 *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,* than to EU, and it will be easier to construct *an adaptationist account* of how *evolution* would have shaped brains to follow prospect-masking *rather* than EU. The prospect-masking follows from *psychophysics,* 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).* (Skopec III., 2015) Many great scientists have related how their most *original ideas* were repeatedly rejected by their peers.

Dichotomous correlations of 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 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 distinguish ability 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 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.*
Focus on cross-functional collaborations (Skopec I., 2017, Skopec IV., 2016)
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) 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 juveniles 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. In adition, activation patterns in *the periaqueductal gray* – involved in *autonomic control* – 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.* (Ramachandran, 2001, Gholipour, 2016)

New Long Distance Effects in Cancer

In congruity with the above stated approach it may be said that a unified medium - the Physical Vacuum - can exist in different phase polarization states. In the state of charge polarization the given medium manifests itself as an electromagnetic field (E). The very medium, while in the state of spin longitudinal polarization, shows itself as a gravitation field (G). Finally, the same medium (the Physical Vacuum) in the state of spin transverse polarization displays itself as a spin (torsion) field (S). All told, EGS-fields correspond to EGS-polarization states of the Physical Vacuum.

In the listed approach the polarization of the Vacuum in accordance with Ya. B. Zeldovich is interpreted as a charge polarization: electromagnetic field. The Vacuum polarization is interpreted by A.D. Sakharov as a spin longitudinal polarization: gravitational field. The polarization for the torsion fields is interpreted as a spin transverse polarization.

Since we cannot assert that other polarized states are impossible, apart from those three ones analyzed above, there are no basic reasons to negate a priori the possibility of other long-range actions. It is not inconceivable and impossible that the conception of A-fields and the polarized states of the Physical Vacuum (phase states of the Physical Vacuum) will usher in a breakthrough in the sphere of newer long-range actions. Torsion fields hold the properties that differ substantially from the known properties in electromagnetism and gravitation. K. Pribram in the Physical Vacuum possesses the property of a hologram.

CONCLUSIONS

Not only theoretical but also numerous experimental results testify to the fact that torsion

fields are an emphatic reality of the Nature. Aforementioned evidence reflects but a mite of the accomplished large-scale research work involving over half a hundred scientific establishments. The acquired results considerably change our ideas of the organization of the world. This indicates that the formulated scientific concepts form a new scientific Paradigm which is probably destined to play a more critical role than the breakthrough in the physics of the elapsing XX century. The already achieved results prompt a conclusion that the XXI century technologies will be torsion technologies.

The Ricci's torsion was introduced in geometry before Cartan's one by Ricci using the rotation coefficients. It is possible to show that the Ricci's torsion is the torsion of absolute parallelism geometry. Let us consider the manifold at each point Xa (a = 1, 2, 3) specified by orthonormalized references

e_A, A=1, 2, 3, (3)

where A is the number of the reference vector. Actually, any reference CA is the mathematical image of a three-dimensional accelerated reference frame.

Cartan and Schouten proceeding from the group properties of the space of constant curvature, introduced connection, in which the components of the Ricci rotation coefficients are constatnts. Suppose that in a n-dimensional differentiable manifold M with coordinates x^1, …x^n we have n contravariant vector fields

$\xi_a{}^j = \xi_a{}^j (x^k)$,

where

a,b,c,…1…n, are vector indices,

and i,j,k… = 1…n, are coordinate indices.

Suppose that that $(\xi_a{}^j) \neq 0$

And that satisfy the equations

$$\xi_a{}^j \xi_{b,j}{}^k - \xi_b{}^i \xi_{a,j}{}^k = - C_{ab}{}^f \xi_f{}^k$$

where the constants $C_{ab}{}^f$ have the following properties

$C_{ab}{}^f = - C_{ba}{}^f$,

$C_{fb}{}^a C_{cd}{}^f + C_{fc}{}^a C_{db}{}^f + C_{fd}{}^a C_{bc}{}^f = 0.$

$C_{fb}{}^a$ We have an n-parametric simple transitive group (group T_n) operation in the manifold

$C_{ab}{}^f$ are structural constants of the group that obey the Jacoby identity. The vector field $\xi_b{}^j$ is said to be infinitesimal generators of the group. We see that

$$\Omega_{jk}{}^i = \frac{1}{2} C_{jk}{}^i ,$$

the components of the anholonomity object (Ricci´s torsion) of a homogenous space of absolute parallelism are constant. It is easily seen that the connection possesses torsion. In this case

$$\Delta^k{}_{ij} = - \Omega_{ij}{}^k = T_{ij}{}^k = - \frac{1}{2} C_{jk}{}^i$$

where - $\frac{1}{2} C_{jk}^{\ i}$ is the torsion of absolute parallelism space.

It was exactly in this manner that Cartan and Schouten introduced connection with torsion

$\overset{*}{\Gamma}_{ijk} = \Gamma_{ijk} + (S_{ijk} - S_{jki} - S_{kij})$,

where S_{ijk} is the Cartan´s torsion of the Riemann-Cartan space. The Ricci´s and Cartan´s torsions have the same symmetry properties, but Ricci´s torsion depends on angular coordinates unlike Cartan´s one. Moreover, Ricci´s torsion defines the rotational Killing-Cartan metric, but Cartan´s not at all.

The first is background independence. This principle says that the geometry of spacetime is not fixed. To find the geometry, we have to solve certain equations that include all effects of matter and energy. The second principle is diffeomorphism invariance. This principle implies that, unlike theories prior to general relativity, we are free to choose any set of coordinates to map spacetime and express the equations. A point in spacetime is defined only by what physically happens at it, not by ots location according to some special set of coordinates. Diffeomorphism invariance is very powerful and is of fundamental importance. (Akimov, 1992)

Darwinian selection process promotes spreading of the new long 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. (Carhart-Harris & Friston, 2010)

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. (Haller *et al.,* 2017)

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 succeeded 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.

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