Review Article

Exploring Quantum in Cancer Biology: A Comprehensive Review of Nontrivial Quantum Events

Ayesha Ali[1], Muhammad Yasir Naeem[2], Zeliha Selamoglu[3,4,5*], Muhammad Raza Naqvi[6]

1. *Department of Mathematics, University of Bari Aldo Moro, Bari, Italy.*
2. *Department of Plant Production and Technologies, Faculty of Agricultural Sciences and Technologies, Nigde Omer Halisdemir University, Nigde, Türkiye.*
3. *Department of Medical Biology, Medicine Faculty, Nigde Omer Halisdemir University, Nigde, Türkiye.*
4. *Western Caspian University, Baku, Azerbaijan.*
5. *Khoja Akhmet Yassawi International Kazakh-Turkish University, Faculty of Sciences, Department of Biology, Central Campus, Turkestan, Kazakhstan.*
6. *Medical Director (Hematologist/Oncologist) and Cancer Liason Physician Intermountain Health Care, Denver, USA.*

How to cite this article: Ayesha Ali, Muhammad Yasir Naeem, Zeliha Selamoglu, Muhammad Raza Naqvi. Exploring Quantum in Cancer Biology: A Comprehensive Review of Nontrivial Quantum Events. Archives of Razi Institute. 2025;80(2):395-400. DOI: 10.32592/ARI.2025.80.2.395

Article Info:
Received: 6 March 2024
Accepted: 24 April 2024
Published: 30 April 2025

Corresponding Author's E-Mail:
zselamoglu@ohu.edu.tr

ABSTRACT

This study explores the potential of quantum computing as an alternative information processing approach, utilizing quantum bits (qubits), superposition, and entanglement to significantly expand computational capabilities in the healthcare domain. It is evident that quantum mechanics has become a foundational component in the construction of our contemporary physical reality. This scientific field is distinguished by its rapid advancement and the potential to transform various aspects of our daily lives. In this era, quantum biology is of significant importance and has the potential to act as a transformative force, particularly in the field of medicine, specifically in addressing the challenges posed by cancer. Cancer is defined as a complex and abnormal alteration of cells, orchestrated through intricate signaling pathways. This transformation is characterized by the accumulation of deleterious mutations. The concept of phenocopy, representing genetic mutations influenced by the environment, challenges the linear process line of molecular biology involving DNA, RNA, and proteins. Notwithstanding the augmented focus on quantum biology in recent decades, a plethora of unresolved issues persist within the domain of cancer biology, thereby giving rise to unexplored avenues. Quantum theory has demonstrated its ability to explain models related to biological and biochemical processes, encompassing the effects of carcinogens on genes, the mechanism of interactions between chemotherapy drugs and DNA, and the understanding of DNA mutations and defective protein synthesis. Recent skepticism among quantum physicists regarding the fundamental role of quantum effects in biology has emerged, particularly with regard to open quantum systems and the impact of decoherence on the destruction of coherence necessary for significant quantum effects. The document under scrutiny herein undertakes an investigation of recent studies that are rooted in the principles of quantum physics, with a particular focus on the manner in which these principles apply to the domains of cancer biology and metabolism.

Keywords: Cancer biology, Quantum, Quantum Principles.

1. Introduction

Historically, medical professionals relied on their diagnostic expertise to interpret diseases and predict outcomes. However, as technology advances and populations grow, the process of examining patients has become arduous, and occasionally yielded inconsistent results from human efforts. The objective of ongoing healthcare research is to enhance visualization and accuracy by leveraging machine learning models. This study explores the potential of quantum computing as an alternative information processing approach, utilizing quantum bits (qubits), superposition, and entanglement to significantly expand computational capabilities in the healthcare domain. Quantum computing systems offer a range of advantages, including high-speed processing, accelerated and more efficient diagnostic assistance, substantial reductions in processing time, and more (1). The emerging domain of quantum biology has facilitated novel insights into biological processes at the quantum level, particularly in complex systems such as cancer biology. In this comprehensive review, the aim is to explore the nontrivial quantum events that occur within cancer cells and their potential implications for disease progression and treatment strategies. The primary objective of this research is to elucidate the intricate interplay between quantum phenomena and the fundamental processes underlying cancer biology. Synthesizing extant research findings and theoretical frameworks, the present study aims to provide a comprehensive overview of the role of quantum mechanics in shaping the molecular and cellular dynamics associated with carcinogenesis, tumor progression and therapeutic resistance. Moreover, the objective is to emphasize the challenges and opportunities intrinsic to the study of quantum phenomena within the context of cancer biology. By critically examining the current state of knowledge and identifying gaps in our understanding, it is hoped that further research efforts aimed at unravelling the quantum mysteries of cancer will be stimulated. The present review aims to illuminate the intriguing intersection of quantum physics and cancer biology, offering insights into the potential implications for diagnosis, prognosis, and treatment modalities. The objective of this project is to promote interdisciplinary dialogue and collaboration, facilitating the advancement of knowledge concerning the quantum aspects of cancer pathogenesis and therapy. This endeavor is approached through a multidisciplinary lens, integrating expertise from diverse scientific domains.

2. Need for Quantum Computing in Healthcare

The study proposes a novel approach in the field of quantum healthcare, contending that quantum computing possesses considerable potential to benefit all. The necessity for a system capable of analyzing patients' health data and offering personalized, practical recommendations to healthcare professionals is emphasized. In circumstances where a patient is reliant on life support, healthcare practitioners generally undertake an assessment of vital health parameters, with the objective of initiating measures that are intended to enhance the patient's well-being. It is evident that there is a requirement for a systematic framework that is capable of providing recommendations to healthcare practitioners with a view to enhancing a patient's health condition. In circumstances where a patient's health is in a state of decline, healthcare professionals are able to glean insights by conducting a thorough analysis of various parameters. However, the conventional approach is time-consuming, as practitioners must re-evaluate the patient's parameters to confirm the effectiveness of recent interventions. This iterative process, influenced by the practitioner's experience and knowledge, prolongs the time required to enhance the patient's health. In instances where patient responses are deemed to be unfavorable, practitioners are obliged to reconsider alternative actions and recommence the process. The challenge is compounded when the number of patients exceeds the number of healthcare professionals available, resulting in a significant increase in the time required to improve patients' health. The temporal dimension assumes particular significance, especially in cases involving patients reliant on life support systems. The recent emergence of the novel severe acute respiratory syndrome (SARS-CoV-2) virus, which causes the disease known as the 2019-nCoV pandemic, has highlighted the challenges faced by healthcare practitioners, who are overwhelmed by the sheer volume of critically ill patients requiring their attention (1, 2).

3. The Onset and Advancement of Cancer from a Quantum Perspective

Cancer is defined as a complex and abnormal alteration of cells, orchestrated through intricate signaling pathways. This transformation is characterized by the accumulation of deleterious mutations. A cancerous tumor is capable of harboring numerous mutated genes and of swiftly adapting to its surroundings (1). The presence of tumor heterogeneity emphasizes the necessity for a personalized pharmacogenomics approach, given that each cancer possesses a distinctive signaling identity. According to Hanahan and Weinberg (2), the hallmarks of cancer encompass a multitude of traits, including but not limited to unlimited replication, reprogrammed respirasome, evasion of apoptosis, angiogenesis, insensitivity to anti-growth signals, contact inhibition, rewired metabolic cascades, and manipulation of the immune system. The central inquiry guiding this study is to ascertain the factors that contribute to the activation of these driver genes and the progression of cancer. Furthermore, the potential for elucidating these factors through the framework of quantum mechanics is a subject of consideration. Moreover, recent research has emphasized the significance of quantum events in cancer biology, as outlined in Table 1.

Table 1. Nontrivial Quantum Events in Cancer Biology.

Quantum Event	Description
Quantum Tunneling	Spontaneous passage of particles through energy barriers
Quantum Coherence	Persistence of quantum superposition of states in biological systems
Entanglement	Correlation between quantum states of particles
Quantum Superposition	Ability of particles to exist in multiple states simultaneously

4. Quantum Mutation

Tomasetti et al. (3) found that approximately two-thirds of cancers result from random mutations. The question that arises from this is whether this randomness can be explained or even predicted through a mathematical framework rooted in the principles of quantum physics. Mutations are defined as perturbations in the deoxyribonucleic acid (DNA) that occur during cell division. These mutations can stem from various sources, including errors during the process of replication. The seminal work of Darwin (1859) posited the theory of natural selection, proposing that the fittest phenotype is adaptively selected in a specific environment. Within this paradigm, a mistaken mutation occurring during stem cell division can be conceptualized as a selective and adaptive mutation. A plethora of studies in the scientific realm have indicated that the progression of cancer is often attributable to a multitude of mutations, with the quantity of such mutations varying among different types of cancer. It has been established that the development of retinoblastoma tumors is contingent on a minimum of two mutations (Knudson, 4). It is suggested that a typical cancer comprises at least four or five driver gene mutations, and significant proportions of human cancers may possess more than nine relevant driver mutation genes. It is hypothesized that these mutations and their selection occur sequentially (5). The cell's capacity for precise replication of genetic material constitutes a complex facet of its inherent characteristics. Notwithstanding the high degree of accuracy exhibited in the replication of genetic material, the phenomenon of adaptive mutation poses a challenge to the Darwinian time-scale separation scheme. A plethora of "classical" hypotheses have been postulated to elucidate the phenomenon of adaptive mutation (4). Numerous research studies posit that adaptive mutation plays a role in the evolution of microbial pathogenesis, cancer, and drug resistance. This concept offers a promising outlook for potential therapeutic interventions in the future (6). Bielas and colleagues (7) observed elevated frequencies of random single-nucleotide substitutions in the genomes of cancer cells, suggesting a mutator phenotype. The question that arises from this is whether a cancer cell contains a random distribution of a few mutations that are not selected. Loeb and colleagues (8) proposed a hypothesis suggesting that malignant changes result from errors during DNA replication base pairing, analyzing the energy of interaction between deoxynucleotides. It has been indicated that this phenotype persists at a late stage in tumor evolution (4). Quantum theory has demonstrated its ability to explain models related to biological and biochemical processes, encompassing the effects of carcinogens on genes, the mechanism of interactions between chemotherapy drugs and DNA, and the understanding of DNA mutations and defective protein synthesis. From a quantum mechanical standpoint, gene mutations can be regarded as quantum wave functions characterized by a degree of uncertainty, which propagate as coherent superpositions of possibilities (8, 9). The concept of phenocopy, representing genetic mutations influenced by the environment, challenges the linear process line of molecular biology involving DNA, RNA, and proteins. Goldschmidt's research demonstrated that exposure of Drosophila embryos to elevated temperatures, ether, and X-rays could induce changes in the organism's phenotype that resemble those produced by gene mutations (10). Conversely, some biologists consider phenocopies to be conclusive evidence that epigenetic mechanisms contribute to evolution. The phenomenon can be explained by reference to the super-orbital or unified energetic quantum state of cells, in which all proteins, genes, and other molecules exist within the same quantum system (11). Consequently, both genes and proteins are simultaneously affected by alterations in the quantum state. The subsequent manifestation of protein modifications as phenocopies is a key feature of this process. As postulated by Rahman et al. (9), the interaction of the cellular quantum system with the genetic apparatus, influenced by the environment, results in gene mutations – a quantum process. The mutated genes persist as uncollapsed coherent superpositions as such mutations accumulate in possibility (10). Bordonaro's (11) exploration focuses on directed adaptive mutation influenced by quantum mechanical effects as a potential contributor to carcinogenesis. The model posits that mutations that promote cell growth occur in environments conducive to such growth. Each microenvironment is characterized by a distinct set of potential cell states, including mutant DNA sequences. This non-random occurrence is selected by the cellular microenvironment. Quantum coherence, termed "quantum selection" by Bordonaro (11), is proposed to play a role in the development of these directed adaptive mutations (12).

5. Quantum Tunnelling

The model proposed by Watson and Crick posits that the genetic code is stored through hydrogen bonds between the purine and pyrimidine nucleic acid bases: adenine–thymine (A–T) and cytosine–guanine (C–G). Lowdin (2023) defines a hydrogen bond as a proton H shared between two electron pairs on nitrogen or oxygen atoms. The attraction of each electron lone pair onto a proton in a hydrogen bond is represented by a deep single-well potential. The superposition of two such potentials forms a highly asymmetric double-well potential with a barrier in the middle (13). Chan (14) asserts that these bonds pair the bases of the two DNA strands – guanine with cytosine (G–C) and adenine with thymine (A–T). In ordinary circumstances, DNA replication produces replicas of the genetic coding of the molecules in question. Improper pairing during replication has been demonstrated to result in alterations to the genetic coding, which in turn can lead to mutations (14). Zhao's (15) model offers a justification for the incidence of cancer data by proposing that the duplex structure of DNA evolved to provide an optimum rate of point mutation variation. This phenomenon fulfils two functions: firstly, it enables species to respond positively to changing environmental conditions, and secondly, it protects the species from the detrimental consequences of accumulating excessive mutations. The model evaluates the consequences of "tunneling-sensitive" DNA codes in diploid and haploid human genomes resulting from evolutionary lesions (15). Zhao (15) posits the hypothesis that spontaneous DNA mutation occurs during normal DNA replication when a proton tunnels through a potential barrier via the quantum tunneling process, thus disrupting the hydrogen bond within the base sequence. Zhao et al. (15) developed a computational quantum mechanical model with the objective of identifying the lowest potential path connecting the centers of two wells, assuming the proton undergoes tunneling. As Godbeer et al. (16) reported, quantum processes such as tunneling can be enhanced or thermally assisted when the system couples to its environment, thereby allowing transitions to higher-energy eigenstates. It is asserted that increasing the temperature of the heat bath over a specific range encourages thermally assisted tunneling, which is analogous to increasing the frequency of a von Neumann-type measurement on the system by the environment. As posited by Godbeer et al. (16), the correlation between quantum measurements and decoherence can be substantiated through numerical simulations (17).

6. Quantum Superposition

Ogryzko (17) was a pioneering figure in the field of quantum evolution, proposing the Quantum Evolution Hypothesis. This hypothesis asserts that cell growth, under specific environmental conditions, functions as a quantum operator. The hypothesis advanced was that cells might manifest behavior analogous to that exhibited by a quantum wave function, existing in a superposition of eigenfunctions of the operator. The process of measurement consequently gives rise to the selection of directed mutants. This suggests that diverse growth conditions correspond to various superpositions, leading to the quantum selection of distinct mutants (6). In a biomolecular adaptation of the Schrödinger cat paradox, Chattopadhyay and colleagues proposed the concept that DNA can be perceived as a superposition of mutational states. This proposition suggests that the elements within living cells could maintain an organized structure while preserving quantum coherence, even at higher temperatures, which would typically destroy the quantum state in insensate systems (17, 18). Patel (19) contributed to the discourse by suggesting that nucleotide bases might persist in a quantum superposition for an extended duration, actively participating in the replication process. Nonetheless, it was observed that this quantum superposition is vulnerable to disruption when DNA interacts with its environment (19).

7. Quantum Metastasis

Quantum has been demonstrated to endow living organisms with the capacity to instigate particular actions, encompassing the production of novel mutations. The distinction between benign and malignant tumors is determined by their characteristics, with benign tumours exhibiting a lack of invasion patterns and metastatic potential. Metastasis, responsible for almost 90% of cancer-related deaths, is dependent on the coordinated morphogenetic differentiation of cells within the microenvironment (20). The cytoskeleton, composed of actin, microtubules, and intermediate filaments, and the Extracellular Matrix (ECM), consisting of protein complexes, cell adhesion molecules (CAMs), fibroblasts, collagen, fibronectin, and laminin, play crucial roles. It is via ECM signaling pathways that the cytoskeleton establishes the structural framework of cells and coordinates their movements during cell division. Microtubules, with their ordered dipolar structure, are considered prime cellular nanostructures supporting coherent functional dynamics (21). In previous research, the impact of anti-tubulin drugs on cancer cells and their environment highlighted the significant effect of microtubule alterations on cell properties. It has been demonstrated that these alterations also influence cellular responses to chemotherapy and micro environmental stressors. This, in turn, contributes to chemotherapy resistance, tumor development, cell survival and cancer metastasis (22). The electric polar vibrations of microtubules are influenced by water molecules within living cells. In cancer cells exhibiting mitochondrial dysfunction, disrupted water ordering and diminished vibrations influence cell interactions, thereby generating altered electromagnetic field (EMG) spectra. These conditions are conducive to local cancer invasion and metastasis (23). In their seminal work, Timofte et al. (24) introduced a novel concept of carcinogenesis and tumor progression, exploring the phenomenon of tumor self-

seeding by circulating cancer cells (CTCs). The movement of metastatic tumor cells along the systemic circulation is conceptualized as a coherent wave with oxygen. The ECM and the TME are regarded as non-differential media that possess holographic properties. The hypothesis that this tumor self-seeding is a mathematical possibility is one that is currently under investigation, with a view to determining whether it contributes to tumor growth or the formation of new tumors. The findings of the study propose new opportunities for targeted therapies to inhibit tumor progression (24).

8. Cancer Cells Survival: A Quantum Mechanical Point of View

As Zink et al. (25) demonstrate, there are marked discrepancies in the nuclear architecture of cancer cells. This indicates accelerated aging and disruption of the body's orderliness. The body becomes susceptible to a state of "maximum entropy," which accelerates patient death in comparison to normal conditions. Interactions between the quantum system and its environment induce decoherence, thereby altering the wave function. Quantum fluctuations are regarded as a potential origin of random biological information, which may offer an explanation for carcinogenic effects through bio-molecular instability (25).
Telomerase has been shown to contribute to the survival of cancer cells by lengthening telomeres, thereby enabling uninterrupted replication. The relationship between shortened telomeres and cancer proliferation remains to be definitively established (10). Telomere uncapping and the subsequent DNA damage may result in the suppression of cancer cell cycle checkpoints, which can lead to chromosome alterations, a hallmark of cancer (Jones et al., 2022). It is further hypothesized that quantum entropic conditions may influence transitions in altered cell cycle checkpoints, thereby allowing cells to meet increased energy demands. In rare cases, the phenomenon of terminally shortened telomeres may not prevent cell divisions. This is influenced by quantum phenomena, thereby enabling cancer cells to survive and replicate (26).

9. Conclusion

Despite extensive knowledge of genetic changes, cancer remains inadequately explained, defying simple mathematical principles and linear molecular pathways. Quantum mechanics has emerged as a potential key to comprehensively understanding cancer, thereby addressing fundamental flaws in molecular biology. While this represents a modest advance in the extensive domain of cancer research and the intricate realm of quantum mechanics, it introduces a non-trivial element into the functioning of life. Recent skepticism among quantum physicists regarding the fundamental role of quantum effects in biology has emerged, particularly with regard to open quantum systems and the impact of decoherence on the destruction of coherence necessary for significant quantum effects. Microscopic biological systems, such as proteins within cells, are to be regarded as open quantum systems. In order to maintain a low entropy, out-of-equilibrium state, they must receive energy from their surroundings. It is anticipated that quantum effects, including superposition and coherence, will dissipate rapidly or decohere as a consequence of this interaction, thereby suppressing quantum dynamics. However, there is an emerging consensus that living systems may depend on the dynamics of small numbers of molecules, which are localized and operate over brief timescales. This relative isolation in space, complexity, and time suggests that substantial quantum mechanical processes could play a significant role in living systems before decoherence induced by the environment eradicates them. In conclusion, the exploration of non-trivial quantum events in cancer biology presents a promising avenue for future research endeavors. Whilst the present review has furnished insights into the potential implications of quantum mechanics in understanding cancer progression and therapeutic interventions, several avenues for further investigation remain to be explored. Firstly, it is imperative that future studies seek to elucidate the specific mechanisms by which quantum phenomena influence fundamental processes in cancer biology, including cell proliferation, metastasis, and drug resistance. The integration of experimental approaches with computational modelling techniques will be crucial in unravelling the complex interplay between quantum dynamics and cellular behavior. Moreover, there is an imperative for interdisciplinary collaborations between physicists, biologists, and clinicians to bridge the gap between quantum theory and clinical practice. It is vital to acknowledge the importance of fostering dialogue and exchange of expertise across disciplines in order to accelerate the translation of fundamental insights from quantum biology into novel diagnostic and therapeutic strategies for cancer patients. Moreover, it is imperative to recognize the limitations of our present comprehension of quantum phenomena in the context of cancer biology. The inherent complexity of biological systems, in conjunction with technical challenges in experimental validation, poses significant obstacles to the complete unravelling of the quantum mysteries of cancer. The present review paper seeks to explore the correlation between quantum mechanical principles and cancer biological processes. The text places particular emphasis on the role of quantum events which are not trivial, including coherence, entanglement, tunneling and superposition, in explaining mutations related to cancer and its metabolism. Despite the speculative nature of the investigations presented and the absence of experimental evidence to support these claims, the theoretical predictions merit further research.

Acknowledgment

The authors would like to express their gratitude to all the authors whose contributions have been included in this systematic review.

Authors' Contribution

Study concept and design: A.A, M.Y.N
Acquisition of data: Z.S, M.R.N
Analysis and interpretation of data: A.A, M.Y.N, Z.S
Drafting of the manuscript: A.A, M.Y.N
Critical revision of the manuscript for important intellectual content: Z.S, M.R.N
Administrative, technical, and material support: A.A, M.Y.N, Z.S, M.R.N.

Ethics

We hereby declare all ethical standards have been respected in preparation of the submitted article.

Conflict of Interest

The authors have declared no conflicts of interest.

Funding

It is important to note that this research did not receive any external funding.

Data Availability

The data that underpins the findings of this study are available upon request from the corresponding author.

References

1. Uthamacumaran A. A biophysical approach to cancer dynamics: Quantum chaos and energy turbulence. Biosystems. 2017; 156:1-22.
2. Hanahan D, Weinberg RA. Hallmarks of cancer: the next generation. Cell. 2011;144(5):646-674.
3. Tomasetti C, Li L, Vogelstein B. Stem cell divisions, somatic mutations, cancer etiology, and cancer prevention. Science. 2017;355(6331):1330-1334.
4. Knudson Jr AG. Mutation and cancer: statistical study of retinoblastoma. Proc Natl Acad Sci U S A. 1971;68(4):820-823.
5. Ashley D. The two"hit" and multiple"hit" theories of carcinogenesis. Br J Cancer. 1969;23(2):313.
6. Wu S, Tao T, Zhang L, Zhu X, Zhou X. Extrachromosomal DNA (ecDNA): Unveiling its role in cancer progression and implications for early detection. Heliyon. 2023.
7. Bielas JH, Loeb KR, Rubin BP, True LD, Loeb LA. Human cancers express a mutator phenotype. Proc Natl Acad Sci U S A. 2006;103(48):18238-18242.
8. Loeb LA, Loeb KR, Anderson JP. Multiple mutations and cancer. Proc Natl Acad Sci U S A. 2003;100(3):776-781.
9. Rahman MS, Islam MF, Al Mamun M, Abdul-Awal SM, Sobhani ME. Evolution of cancer: a quantum mechanical approach. Eur J Biophys. 2014;2(4):38-48.
10. Goodwin B. How the Leopard changed its Spots-The Evolution of. 2015.
11. Bordonaro M. Quantum biology and human carcinogenesis. Biosystems. 2019; 178:16-24.
12. Jd W, Fh C. Genetical implications of the structure of deoxyribonucleic acid. Nature. 1953;171(4361):964-967.
13. Löwdin PO. Proton tunneling in DNA and its biological implications. Rev Mod Phys. 1963;35(3):724.
14. Chan JY. On genetic information uncertainty and the mutator phenotype in cancer. Biosystems. 2012;108(1-3):28-33.
15. Zhao ZM, Zhang QR, Gao CY, Zhuo YZ. Motion of the hydrogen bond proton in cytosine and the transition between its normal and imino states. Phys Lett A. 2006;359(1):10-13.
16. Godbeer AD, Al-Khalili JS, Stevenson PD. Quantum measurement and thermally assisted proton tunnelling. arXiv preprint arXiv:1310.2737. 2013.
17. Ogryzko VV. A quantum-theoretical approach to the phenomenon of directed mutations in bacteria (hypothesis). Biosystems. 1997;43(2):83-95.
18. McFadden J, Al-Khalili J. A quantum mechanical model of adaptive mutation. Biosystems. 1999;50(3):203-211.
19. Patel A. Why genetic information processing could have a quantum basis. J Biosci. 2001; 26:145-151.
20. Jacobson JI. A quantum theory of disease, including cancer and aging. Integr Mol Med. 2016;3(1):524-541.
21. Shashaani H, Faramarzpour M, Hassanpour M, Namdar N, Alikhani A, Abdolahad M. Silicon nanowire based biosensing platform for electrochemical sensing of Mebendazole drug activity on breast cancer cells. Biosens Bioelectron. 2016; 85:363-370.
22. Parker AL, Kavallaris M, McCarroll JA. Microtubules and their role in cellular stress in cancer. Front Oncol. 2014; 4:153.
23. Pokorný J, Pokorný J, Foletti A, Kobilková J, Vrba J, Vrba Jr J. Mitochondrial dysfunction and disturbed coherence: gate to cancer. Pharmaceuticals. 2015;8(4):675-695.
24. Timofte D, Eva L, Vasincu D, Buzea CG, Agop M, Popa RF. Implications of the "Subquantum Level" in Carcinogenesis and Tumor Progression via Scale Relativity Theory. Selected Topics in Applications of Quantum Mechanics. 2015:399-458.
25. Zink D, Fischer AH, Nickerson JA. Nuclear structure in cancer cells. Nat Rev Cancer. 2004;4(9):677-687.
26. Hiley BJ. Quantum reality unveiled through process and the implicate order. Birkbeck: University of London. 2008.