Review Article Open Acces

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Introduction

According to rst law of thermodynamics [$Q = U + W_{int} + W_{ext}$], Stationary State of open non-equilibrium non-linear thermodynamic system of an able-bodied organism and also Quasi-stationary State of open non-equilibrium non-linear thermodynamic system of a sick organism are characterized by stability of Internal Energy (U) (stable temperature 36.0-36.9C by which all enzymes operate etc.). e mechanisms stability of Internal Energy (U) are maintained by Internal Works (W_{int}) and External Works (W_{ext}), which generate the Total Heat Energy (Q) dissipating into Environment for maintenance of stability Internal Energy (U). Just stability of Internal Energy promotes stability of Internal Medium (constant concentration substances in blood and in neurolymph) (Figure 1). Mechanisms maintenance stability of Internal Energy (U) and Internal Medium an organism are based on mechanisms regulation of biergn-n-n4a(t)6te 4mno12ue tn-n-n4ve procesTm [(), w)-7(hdef o)11(2)12((t

Structure Regulatory Processes for Maintenance Stability as Stationary State in Norm As Well As Quasi-stationary States in Pathology

Common mechanism of maintenance stability as Stationary State in norm as well as Quasi-stationary States in pathology is divided into three levels of regulative mechanism: highest level regulation, high level regulation and low level regulation (Figure 1).

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organism (Figure 1). All above mentioned Equilibrium Constants are contributed to mechanisms stability as Stationary State in norm, and as well as Quasi-stationary States in pathology. Central nervous system is highest level regulation, which in uences as on high level regulation, as well as on low level regulation through high level regulation [3] (Figure 1).

Biochemical and Biophysical Processes in Cells Promoting as Stability Stationary State in Norm As Well As Stability Quasi-stationary States in Pathologic States of an Organism

Considering mechanism maintenance stability Internal Energy and Internal Medium of an organism as in Stationary State as well as in some Quasi-stationary States, it is necessary to estimate mechanisms occurring in cells which maintain stability of basophilic chemical potential of cellular cytoplasm (μ) [1,2,4]. Interactions between cellular mechanisms and organism's mechanism for maintenance stability Internal Energy and Internal Medium of an organism operate via moderate oscillating shi s into catabolic pathway, and into anabolic pathways in norm. Excessive shi s into catabolic pathway and into anabolic pathways cause di erent Quasi-stationary States of an organism. Besides they can promote either excessive in ow substances into an organism with excessive endocytosis or out ow substances from an organism with excessive exocytosis.

Participation pro-factors and anti-factors in mechanisms maintenance stability as Stationary State in norm as well as Quasi-stationary States in pathologic States of an organism

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processes of glycolysis produce energy, which are divided in “Node” point of bifurcation of anabolic and catabolic processes in Acetyl-CoA [NPBac] into anabolic endoergonic pathway and catabolic exoergonic pathway [2-4]. erefore, glycolysis is the primer all above mentioned as pro-factors as well as anti-factors.

Mechanisms maintenance stability Stationary State during a life as each cell of an organism as well as an organism

e open thermodynamic systems of each cell and of an organism develop in advance of age terms, moving their metabolisms to the di erent stationary states: from young ages up to elderly age and up to old age [1] (Figure 2). At young ages, they show positive uctuations entropy (+ $_x$), according to Glansdor –Prigogine stability criterion. Progressive interactions of balanced catabolic and anabolic processes contribute to generating energy catabolic processes and consumption energy into anabolic processes with moderate shi s into anabolic processes, that create positive uctuation entropy (+ $_x$) and ascending ow of linear graph of Stationary State at young age as cell, as well as an organism [1,2] (Figure 2). Transition into Quasi-stationary pathologic States provokes mainly acute in ammatory processes due to excessive expression catabolic processes at young age. At middle ages, balanced interactions catabolic and anabolic processes are xed by equilibriums between generating energy with dissipation energy into environment and preserving energy for processes biosynthesis that maintains stability of balance catabolic and anabolic processes. Stability of balance catabolic and anabolic processes contribute to horizontal ow of linear graph of Stationary State at middle ages because equilibrium positive uctuations entropy (+ $_x$) is drawn side by side with negative uctuation entropy (- $_x$) suppressing negative uctuation entropy (- $_x$) [1] (Figure 2). Transition into Quasi-stationary pathologic States provokes mainly chronic in ammatory processes due to excessive expression both catabolic in ammatory processes and anabolic defensive immune processes at middle age. At elderly age and old age, it takes place regressive interactions of balance catabolic and anabolic processes, in which occurs moderate prevalence of catabolic processes with moderate dissipation energy. Such moderate dissipation energy contributes to moderate negative uctuation entropy (- $_x$), leading to descending shi s of linear graphs of Stationary State according to Glansdor –Prigogine stability criterion (Figure 2). Such descending shi s of linear graphs of Stationary State can promote either transiting into excessive negative uctuation entropy ($_x$), leading to Quasi-stationary pathologic States due to decrease of defensive system an organism (immune system, hormonal system etc.), or to natural death due to total exhaustion of energy, i.e. increase gain of entropy according to Prigogine theorem [1] (Figure 2 and 3]. At elderly age such transition into Quasi-stationary pathologic States can lead to chronic infectious diseases. Also, transition into Quasi-stationary pathologic States can o en provoke malignant proliferative processes due to excessive expression anabolic processes at elderly age. e life of each cell advances from youth age till old age. At youth age, it occurs as increase production and consumption energy leading to moderate oscillating balance anabolic and catabolic processes, as into anabolic processes as well as into catabolic processes. At old age, it occurs that decrease production and consumption energy leading to decrease as anabolic processes, as well as catabolic processes and nishing in cell death, in which arise gain of entropy due to total exhaustion of energy according to Prigogine eorem [1].

Role of Autophagy in mechanism maintenance stability Stationary State as each cell of an organism as well as an organism

Autophagy takes part in defensive mechanism of an organism, which develops according to changes of uctuation entropy advancing as an organism life as well as a cell life from infancy till old age. Normal distinctions between Stationary States in various ages of an organism, connecting with the moderate uctuation entropy, contribute to normal development of an organism according to Glansdor –Prigogine stability criterion [1,2]. Also, each cell has the speci ed term of its life which is ended with cell death [1,2] (Figure 2). In the beginning of termination cell life, there are occurred apoptotic processes, which exhibit degradations of nucleus and mitochondria leading to damage of

Maintenance stability Internal Energy and Internal Medium by defensive mechanisms in in ammatory processes and infection diseases of an organism

Stability of Internal Medium and Internal Energy both an organism and cells of an organism is maintained by balance catabolic and

via cellular mitochondrial cytochrome and system of haemoglobin in an organism. Catabolic aerobic pathway occurs in mitochondria via system cytochrome, which accept oxygen and promote oxidation of the products metabolism down to CO_2 and H_2O with dissipation energy in environment for maintenance stable temperature 36.6 37.2°C by which all enzymes operates. us, even partial suppression anaerobic catabolic processes leads to forming of excessive quantity Reactive Oxygen Species [ROS] in mitochondria of cancer cells, unlike moderate quantity Reactive Oxygen Species [ROS] in able-bodied cells. Generated by Reactive Oxygen Species [ROS], the excessive quantity of superoxide [O_2^-] produce free radicals $^{\bullet}OH$, which can ruin mitochondria of cancer cells. However, cancer cells metabolism demonstrates property of Apoptosis Resistance. It is meant that free radicals generated by ROS are neutralized in some metabolic processes. It can be realized either by the redox transformation of glutathione disul de for free radical's elimination [32], or by the use of free radicals for advance cellular cycle, causing irrepressible proliferative processes of cancer cells [33-36]. us, the mechanism of neutralization free radicals occurs due to their participation in mechanism of nuclear DNA replication. Free radicals $^{\bullet}OH$ promote separation nuclear DNA from new DNA in process of nuclear DNA replication, and were neutralized in process of nuclear DNA replication. Excessive quantity ROS in cancer cells' mitochondria, as opposed to normal cells' mitochondria, cause irrepressible proliferative processes in cancer cells metabolism [4,33-36]. ROS in small quantity ful ls the same role in able-bodied cells of G_2 phase cellular cycle exerting normal process of nuclear DNA replication. Just accelerating cellular cycles in cancer tissue consume a lot of substances and energy for excessive anabolic processes in G1/S phases cellular cycles, and then it occurs, distribution this energy due to nuclear DNA replication in G_2 phase cellular cycle transiting in M phase of cells division [4].

Bene ts of using prolonged medical Starvation for the new approach to cancer therapy

Taking into account mechanism of suppression catabolic anaerobic processes in cancer tissue, the new method cancer disease treatment, which use Prolonged medical Starvation 42 -45 days, gives chance to suppress anabolic processes due to increase of catabolic processes, both in an organism metabolism and in cancer metabolism. is method cancer treatment causes suppression cancer metabolism targeting Warburg e ect leading to depression of cancer development that gives possibility to use e cient cancer therapy, with considerably decreased dosage of cytotoxic drugs for preserving immune and hormonal systems of an organism and prevention of recurrences cancer disease a er long treatment with cytotoxic therapy [5,6].

Reviews From e Point of View of the O ered Concepts of Results of Some Researchers Studying Mechanisms Pathologic Processes

Reviews of results researches studying in ammatory processes and Infectious diseases

Studying mechanisms development in ammatory processes in 5(s)-8(es6)13(o.099)1(o)1243(e)-6(a)k

ese acute in ammatory processes have di erent mechanisms of contact biochemical reactions. e mechanism of excessive catabolic processes stimulates expression of anabolic processes that contribute to some decrease of temperature an organism, and to expression of immune defensive system, maintaining stability Internal Energy and Internal Medium of an organism. Expressions of anabolic processes stimulate action cellular Fcy receptor's capacitors of B-leucocytes and monocytes, which induce syntheses IgG antibodies exerting immune process in humoral liquids of an organism (blood, lymph, neurolymph, tissue liquid) [2,29]. Gram-negative bacteria can cause as acute in ammatory processes, and as well as chronic in ammatory processes. e mechanisms of acute in ammatory processes induced by Gram-negative bacteria are the same as induced by Gram-positive microbes. Also, immune cells are rearranged corresponding to antigenic properties of Gram-negative bacteria due to interaction between cellular capacitors of immune cells and Ligand-dependent transcription factor, creating by the nuclear receptor glucocorticoid receptor (GR), mediated by the activator protein 1 (AP1) [38]. However, these processes progress less vigorously because Gram-negative bacteria have antigens which exhibit similar biochemical properties to the active pro-/anti-in ammatory factors of an organism [37]. Moreover, Gram-negative bacteria can also cause violation of both "Equilibrium Constants ions exchanges" and "Equilibrium Constants oxidative-reduction exchanges", shi ing into excessive reduction processes that leads to shi of balance catabolic and anabolic processes into excessive anabolic processes, unlike Gram-positive microbes (Figure 1). en, the excessive anabolic processes, induced by Gram-negative bacteria, exert the proliferative pathway, and Gram-negative bacteria are located within new maturated cells forming chronic in ammatory processes. Besides, Gram-negative bacteria inducing excessive anabolic processes stimulate biosynthesis of antibodies against antigens of Gram-negative bacteria. us, the mechanisms of in ammatory processes, induced by Gram-negative bacteria, have the several pathways: acute infectious disease, chronic infectious disease, bacteria carrying and immunity against these Gram-negative microbes. us, immunoglobulin-like receptor (LILRA2), inducing pro-in ammatory cytokines, Toll-like receptor (TLR) 4 and Fc RI receptor can operate via divergent pathways of chronic in ammatory processes [39,40].

Reviews of results researches studying oncological diseases

Studying mechanisms of proliferation in malignant cells, Elstrom et al. [41] has surprised that proliferation of malignant cells did not increase in culture with the activated serine/threonine AKT kinase, though there was stimulation of glucose consumption in the transformed cells, without a ecting the rate of oxidative phosphorylation. Indeed, the activated serine/threonine AKT kinase stimulates glycolysis, which is the primer for both anabolic processes and catabolic processes. e stimulation of glucose consumption in the transformed cells indicates expression anabolic processes with partial suppression of catabolic oxidative phosphorylation. However, anabolic processes occur also in the stationary state of normal tissue in cellular quiescence G_0-phase cellular cycle, which does not require a lot of energy for the moderate biosynthesis of simple substances, which can be excreted via oxidizing and exocytosis processes [2,4]. Unlike cellular quiescence G_0-phase cellular cycle, the malignant proliferative processes require considerably more energy accumulated in lactic acids for biosynthesis of compound high-molecular substances in G1/S phases cellular cycle, which cannot be excreted from the cell via exocytosis [2,4]. e exocytosis of the high-molecular substances "Alternative excretion" occurs in malignant proliferative processes, owing to division cell through G2/M phases cellular cycle and distribution these nigh-molecular substances among the new cells [2,4]. Driving mechanisms exerting anabolic processes for advance of cellular cycle in proliferative processes are located in nuclei of cells. ere are two mechanisms of the driving mechanisms in nuclei: a) e result of normal changes nucleus metabolism due to natural development cellular cycles with moderate shi metabolism into moderate anabolic processes, which in uence on development normal cellular cycle, i.e. each cell is limited with only 50 mine divisions. b) e result of incorporation into nucleus the strange gene, i.e. oncogene, which changes nucleus proliferative program of cellular cycle into viral accelerative proliferative program, and viral accelerative proliferative program causes shi metabolism into excessive anabolic processes in cancer tissue. Resistance between the driving mechanisms de nes pathways of proliferative processes: either v-oncogene is acclimatized into nucleus changing nucleus proliferative program into excessive anabolic processes with partial suppression catabolic processes, or v-oncogene is not acclimatized into nucleus because insu ciency of respiratory mechanism in cells culture leads to preservation certain level of catabolic processes inhibiting excessive increase of anabolic processes. erefore, expression anabolic processes of malignant cells in cellular culture occur only in moderate level without overload "nodal point bifurcation anabolic and catabolic processes [NPBac]–in Acetyl-CoA" due to su ciently of Acetyl-CoA. Hence, anabolic processes of malignant cells induce only biosynthesis of some simple substances in cells culture [2,4]. Bellacosa et al. [42] have exclaimed surprise that tumor cells rarely display increase size in comparison to their normal counterpart, in spite of the mTOR/cIF4E pathway that is o en activated in human tumours. However, additional increases of quantity Acetyl-CoA are not formed in these mechanisms, and therefore, it fails strengthening of anabolic endoergonic processes and increase of proliferation [2,4]. Bonnet et al. [43] studied aerobic glycolysis of Warburg e ect, and have found high mitochondrial membrane potential and low expression of the K_v channel, contributing to apoptosis resistance. Really, the excessive anabolic processes cancer tissue promote some increase mitochondrial membrane potential (μ) for cancer cells proliferation, causing their survival in condition of suppression catabolic anaerobic processes. Suppression o

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