Research Article

Understanding the Fact of Cancer Diseases and Respiratory Viral Infections Effects on Interstitium Fluid, on G-Actin Filaments, Endothelin_1, and on Capillaries Functions.

Ashraf Marzouk El Tantawi*

***Corresponding Author: Dr. Ashraf Marzouk El Tantawi** Biomedical molecular studies. Cancer and viral infections. Canada, Toronto, Goldwin Av. Egypt, Giza, Abd elmonem R.

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Abstract

The results of blood clotting in filaments and capillaries after long neglect term will lead to mutations and Inhibition in G-actin isoforms functions, in tropomyosin, & in antigen compositions in interstitium fluid between cells & will lead to decreasing & decreasing in AMPK synthesis and activities, & inhibition in MPAK pathways and PPARs functions that can lead to accumulation of peptides in interstitium fluid, lead to isolation to that part of tissue cells with particles trappings, lead to tumor appearances and cancers problems, lead to some symptoms of heart diseases. The major cytoskeletal protein of most cells is actin, which polymerizes to form actin filaments—thin, flexible fibers.

The assembly and disassembly of actin filaments, they are crosslinking into bundles and networks, and their association with other cell structures (such as the plasma membrane) are regulated by a variety of proteins such as tropomyosin, AMPK, and TOR protein, which are critical components of the actin cytoskeleton. Actin filaments are particularly abundant beneath the plasma membrane, where they form a network that provides mechanical support, determines cell shape.

In The G-actin molecular structure the lower cleft between domains 1 and 3 is lined by Tyr143, Ala144, Gly146, Thr148, Gly168, Ile341, Ile345, Leu346, Leu349, Thr351, and Met355, which constitute the major binding sites for most actin-binding proteins (ABPs), (Annu Rev Biophys. 2011 Jun 9; 40: 169–186. PMCID: PMC3130349)

In other words, the most imp binding nucleotides in G_actin are Tyr Gly Gly Ile, ite,& Leu, Which are resemble the same nucleotides arrangement in Leu pentapeptides Tyr, Gl, Gly, Phe, Leu in enkephalin in the brain, which indicates that G-actin activity is controlled by Leu-pentapeptides in enkephalin in the brain and controlled by sestrin Leu carrier activities with its AMPK and TOR proteins activities.

As one or more of those necessary amino acids in G_actin isoforms missed or changed, as inhibition will start in G_actin isoforms will lead to more inhibition in cell metabolic cycles leads to the accumulation of +ve cation& peptides in interstitium fluid, and will block filaments function and will be the result of isolation of those mutated tissue cells, leading to tumor & dysfunction in many other connected tissues lead to health problems and cancers. Gastric cancer (GC) is the result of many of the wrong processes that occurred in gastric cell tissue including inhibition in actin isoforms functions, and inhibition in antigen synthesis.

That GC started by a blockage in capillaries due to many reasons including the increase in +ve molecules in interstitium fluid between gastric cells. The increasing and accumulation of +ve linkages in peptide molecules in interstitium fluid will start to inhibit & break down some of G_actin isoforms and their ATPase enzyme into undefined subunits that will give reductions in transmitting signals and sensations, reduction in oxidations, and will show more +ve precipitations in filaments and capillaries that will stimulate the blood platelets aggregation and TGF-beta activities.

G-actin filaments are controlled by tropomyosin isoforms peptides " that tropomyosin controlling ATPase activities ", and at the same time actin controlled by antigen amino acids compositions activities (including Leu activities in sestrin), that inhibition or mutation in antigen compositions will inhibit G_actin composition and functions, that later can be the main reason for capillaries blockage and isolation of those tissue cells "where blood & supplements will not reach the isolated tissue cells area", lead to decreasing in sestrin Leu carrier activities and its AMPK with TOR protein that'll decrease their MAPK pathway activities that finally lead to tumor and cancers, and also will be helpful for viral infections..

Decreasing in G-actin and their ATPase activities will lead to the precipitation of protein peptides on filaments and capillaries wall, and will precipitate in the interstitium fluid between cells, which will decrease the endothelin_1 and the rest of G_actin activities including decreasing in sensation deliveries, that will inhibit signal transmission across filaments, then mutations will begin to increase in the region of the isolated cell due to Trapping peptides within that isolated tissue cells from other tissues cells.

Tropomyosin is controlling filament activities through switching the increasing or reducing their ATPase loops activities, that as tropomyosin increased by binding with Ca+, as ATPase functions will be reduced, but as tropomyosin blinded to +ve elements reduced as ATPase loops functions will be increased.

The increasing in mTOR with tropomyosin bonding to +ve cation peptides will reduce actin filaments activities and will increase the probability of precipitation "by reducing polarities in molecules due to decreasing in ATPase in G-actin loops " in interstitium fluid and in capillaries that will help to paralysis filaments functions and capillaries, and nerve fibers. Aromatase is the key enzyme "that is depending on its activities on ribosomal ATPase " for estrogen productions, that are controlled by ribosomal ATPase active loops and ATPase in G-actin, were comprised of at least ten partially tissue-selective and alternatively used promoters. Those promoters are regulated by distinct signaling pathways specifically by ATPase and by MAPK pathways.

Aromatase is controlling the activities of Leu function activities with sestrin and with both AMPK & TOR proteins, tropomyosin activities, and ET-1 productions, then consequently will stimulate PPARs pathways through ribosomal and G-actin ATPase enzyme activities for PPARs genes functions via recruitment of various transcriptions processes for estrogen productions. Aromatase is fully dependent on ribosomal ATPase and activities, that the reduction in ATPase activities will reduce aromatase activities, and will affect actin filament functions, which will reduce lipid metabolism and oxidations as it's functions contribute to cytochrome activities.

That Cleaning capillaries from blood clots and from blockage which occurred in filaments and capillaries, followed by rebuilding in actin peptide isoforms & ATPase in the G_actin loops, will be the 1st step & helpful for recoveries from tumor and cancers, and viral infection too.

The reduction in ATPase & MAPK pathways activities will reduce aromatase enzyme, and consequently will reduce cytochrome & ribosomes activity, and cell membrane activities then will reduce G_ actin activities, that will lead to delaying in signal transmission lead to accumulation of peptides and molecules in interstitium fluid than in blood capillaries, then lead to isolation to those cells region in tissues lead to tumor appearances and cancer problems. Also, the inhibition in ATPase and MAPK pathways with capillaries blockage and blood clotting in respiratory filaments will be the main helpful for easy viral infection eg Coronaviruses effects on respiratory tissue.

Alterations & mutations in antigen compositions structures due to Leu deficiency with some more other effective factors as a deficiency in AMPK protein and dysfunction in cytochrome function & dysfunction in ribosomal ATPase will lead to mutations in actin isoforms compositions structures and function that will reflect decreasing in heart muscle efficiency, and blood Coagulation in capillaries and filaments, where amino acids in mutated antigen and G-actin will not be matched together for accepted translation for the favor of living cells that will lead to Precipitation of unreached peptides in interstitium fluid and on filaments and in capillaries.

The main element for helping and accelerate tissue synthesis is sulfur that normally binds to more than three peptides chains for cells & antigen synthesis, where the increase in sulfur than normal will lead to accumulation and precipitation in blood vessels, and missing in proper translations process. Also, increasing in the breaking sulfur imp active bonds in biological mol more than normal may lead to breaking & loosen tissues & the bleeding in that tissue due to increase in blood fluidity, that in briefly sulfur increase thrombin synthesis, but fluorine are so imp for breaking sulfur bonds with the condensed peptide in blood for increasing blood fluidity.

Keywords: *G-actin, endothelin_1, cellular antigen, gastric cells, gastric cancer tissue cells interstitium gastric fluid, alpha and beta subunits,PPARs genes, G-actin with ATPase in their composition, tropomyosin. Rituximab), thrombin inhibitor, beta cells, CTLA-4A protein, neutrophil gelatinase, rapamycin mTOR, Cytochrome P450.*

Introduction

Cancers and many vascular problems stared by a blockage in tissue filaments and blood capillaries, that will lead to isolation to that region of tissues cells then will block the receiving of blood supply and will prevent the getting rid of the isolated peptides in that region of the isolated tissue cells, lead to the appearance of tumor and cancers problems. To modify aromatase activity may lead to the development of novel therapeutic remedies that inhibit estrogen production in a tissue-specific manner **(**33**)**.

Also, estrogen inhibits autophagy and promotes the growth of endometrial cancer by promoting glutamine metabolism **(**37**)**, but the combination of alanine and leucine (Leu) mimics the inhibitory effect on autophagy in near-physiological concentrations **(**36**)**, that can replace glutamine to inhibit its metabolism and restart its activities that activate the main immune cells activities and mimic the inhibitory effect on autophagy through activating Leu pentapeptides activities and neuron activities that are depending on sestrin Leu carrier tool activities.

The inhibition of estrogen in this treatment for breast cancer will lead to other severe problems, where the excess estrogen in normal capillaries status and functions does not increase the risk of breast cancer, but dysfunction in G-actin signal transmission and inhibition in their isoforms functions with inhibition in ribosomal ATPase activities and blockage in capillaries will be the main of increasing the risk of cancers. And the type of cancer will depend on deficiency in some other imp peptides activities as sestrin Leu carrier activities which are connected in their activities results with the availabilities of some enzymes including aromatase enzymes.

Aromatase is the key enzyme for estrogen productions, was comprised of at least ten partially tissue-selective and alternatively used promoters. Those promoters regulated by distinct signaling pathways specifically depending on ribosomal ATPase and on MAPK pathways activities for controlling aromatase expression, estrogen formation, and G-actin peptides isoforms activities, then needed for controlling the activities of productions, then consequently will stimulate PPARs pathways for PPARs genes functions via recruitment of various transcription processes for estrogen production.

There are three major forms of physiological estrogens in women: estrone (E1), estradiol (E2, or 17β-estradiol), and estriol (E3). E1 is synthesized in skin and adipose tissues from circulating androstenedione of adrenal origin indicating that E1 synthesis is related & depending on G-actin filaments isoforms and their ATPase loops activities, consequently, estrogen is fully related to aromatase enzyme availabilities and activities, and related to AMPK protein functions **(**34**)**.

E2 is synthesized by involving E1 in extragonadal processes started by the endothelin_1 activities, and MAPK pathways, and aromatization by ribosome functions in interstitium fluid. E3 is the least potent estrogen and is synthesized in large quantities in the placenta. Estrogen synthesis is depending on Leu function which is protected, and accelerated by sestrin– Leu carrier tool with the availabilities of TOR and AMPK protein, and depending on ATPase activities in G-actin and in ribosomal composition. G_actin filaments contain G_actin isoforms and contain ATPase in loops in its structural composition.

The inhibition to G-actin functions with increasing in +ve peptides molecules will lead to +ve peptides and cations precipitations in interstitium fluid and capillaries, that lead to isolations to that region part of tissue cells and may trap some of the E1 & E2 in the isolated cells tissue (cancer cells), and also will lock up processes in inner cells side inside the isolated tissue with a percentage of actin isoforms with tropomyosin, and PPARs genes, that will lead to tumor & cancer problems.

Gastric cancer "GC" started by a blockage in capillaries due to many reasons including the increase in +ve molecules in interstitium fluid between gastric cells. The increasing in +ve linkages in interstitium fluid will be stimulated to bind to tropomyosin lead to increasing in tropomyosin isoforms that will inhibit or reduce ATPase loops in G-actin activity, lead to a reduction in polarities synthesis in molecules then lead to a reduction in sense transmission and reduction in endothelin-1 production, and break down some of G_actin isoforms and their ATPase enzyme into undefined subunits that will give a reduction in sensation delivers by actin isoforms and will show a +ve precipitations in interstitium fluid that will stimulate the blood platelets aggregation and TGF-beta activities that will help the increasing in capillaries blockage.

G-actin filaments are controlled by tropomyosin and by antigen amino acids compositions, that G_actin isoforms inhibition or mutation can be the main reason for capillaries blockage and isolation of those cells lead to decreasing in mitogen-activated protein kinase (MAPK) pathways activities and decreasing in aromatase activities in the isolated blocked tissue cells, and that finally lead to Gastric cancer. Another cancer is strongly linked to the Blockage in the microcapillaries and filaments which are the main for feeding gastric cells "GC" from neuron cells and other adjacent cells.

G-actin isoforms protein are the main parts of the contractile apparatus system in muscles in heart muscle, in capillaries compositions & in epidermal tissue, that G-actin isoforms which composed of actin globular protein are attached to each cell membrane for helping cells to facilitate their proper metabolic processes and helping cells to get rid of their toxic processes results, and are the main part of deliveries system that transmitted sensations between cells, and neuron cells in muscle and nonmuscle cells.

Each attached actin isoforms protein to cells differs from tissue to another depending on those cells' specificity and functions, and dépending on their functions from the brain responds.

The interstitium fluid compositions are containing the main necessary elements, genes, and peptides for cells metabolism, and contains G-actin isoforms and tropomyosin, which is necessary for controlling muscles flexibilities and filaments functions, that consequently control peptides molecules in metabolic cycles, and controlling signal transmission & and can organize other molecules in all extracellular matrix but with the controlling from their ATPase and the 1st DNA strand regulation to 2nd DNA strand, which include the translated codes for actin isoforms protein filaments, and are performing so imp Contributions for many tissue cells, and connective tool to all living cells.

Method

That G-actin functions are controlled and activated by their ATPase loops functions and by MAPKs pathways activities which are controlled by ribosomal ATPase and by the presence of serine and threonine protein kinases, that are necessary not only involved but main for signal transmission and for nerve and fat G_actin filaments functions, which modulate and accelerate physiological and pathophysiological processes in interstitium fluid and neuron cells.

G_actins are proteins that their functions involved in the contractile and non-contractile system of skeletal muscle and cells including epidermal cells, where are found in thin filaments with different amino acid compositions and has inculcated ATPase in loops in their highly & fine biological structure. In muscle, fibrous actin (F-actin) is a helical polymer of a globular polypeptide chain, G-actin.

Six active primary actin isoforms have been identified in higher vertebrates, being alpha-skeletal (ACTA1), alpha-cardiac (ACTC1), alpha-smooth muscle (ACTA2), gamma-smooth muscle (ACTG2), beta-cytoplasmic (ACTB), and gamma-cytoplasmic isoactin (ACTG1) **(**31**)**.

Those active actin alpha skeletal are having the Thr and Ser amino acids that are necessary for phosphorylations reactions by ATPase and MAPK pathways activities for stimulating actin isoforms peptides, and are also necessary for MAPK pathways activities.

As mutations occurred to G-actin isoforms filamentous composition due to increasing in +ve linkages in interstitium fluid or due to missing some of the main imp amino acids in G-actin isoforms proteins composition, as isolations to those cells region will begin and accumulation of peptides molecules "may due to platelet aggregation" will begin then blockage in filaments and blood capillaries will begin then cancer will start whether in gastric tissue or any other tissues.

Actin is characterized by its high-quality, compositions with high function in speeding - ve charges across tissues.

Tropomyosin is considered to be able to control & regulate actin filament functions through binding to +ve elements that lead to a decrease in ATPase activities. Tropomyosin isoforms are differentially able to alter actin filaments organizations “by binding to +ve elements ”, cell size, and shape, and help to recruit myosin II into stress fibers, which resulted in decreasing in cellular migration.

That tropomyosin control G-actin filaments through binding to +ve elements for producing a selective dimerization due to inhibition or decreasing the actin ATPase activities, and can decreasing MAPK pathways that can reduce cellular migrations including sense and signals transmission, that is why tropomyosin can be involved in isolated tumor tissue bonded to +ve molecules, with aromatase which has been inhibited due to inhibition in actin or ribosomal ATPase.

G-actin and tropomyosin isoforms are constructed to be matched to each other in the nucleotides arrangement for controlling cellular signal deliveries and tissue contracts and heart muscle contractions too. Actin isoforms nucleotides quality and quantity arrangement differ according to tissues functions and according to the received amino acids and tissue biological atmosphere in vivo.

That, the increase in tropomyosin isoforms activities with decreasing in sestrin Leu carrier activities will delay actin filament functions, lead to an increase in platelet aggregations and activities.

The most important reason for capillaries -blockage is the dysfunction of Leu pentapeptides and decreasing in sestrin_Leu carrier activities "which contain two more sites in its structure with Leu for TOR and AMPK protein binding functions ",Also the decreasing in blood fluidity with decreasing in G_actin and ribosomal ATPase will lead to a reduction in actin isoforms activities and accumulation of peptides in the interstitium fluid between cells, and thus will reduce all Metabolic processes where actin contribute main imp root for performing metabolic cycles through their ATPase enzyme activities, also decreasing in actin functions and activities will lead to reductions in the MAPK pathways, and thus lead to a reduction in the blood fluidity through increasing binding with +ve elements or +ve molecules & will reduce muscle contractions flexibilities, or reduces peptides signal migrations.

Also, decreasing in G-actin and ATPase activities will lead to precipitations of protein peptides on the vessel wall, and will precipitate in the interstitium fluid too, which will decrease the endothelin-1 activities.

The increase in protein accumulations in interstitium fluid and capillaries will paralysis filaments, capillaries, lymph, and nerve fibers...

In studying the rule of Leu amino acids in endothelin A (ETA)-receptor functions and its importance in arterial smooth muscle **(**38**)**, that Leu7ET-1 is a contractile agonist with lower

potency and similar maximal effect compared to ET-1 and greater sensitivity to BQ123 than ET-2 **(**38**)**.

The availabilities of Leu amino acids in endothelin-1 and actin isoforms proper receptor will modify & accelerate their functions through increasing their flexibility in reacting with sestrin Leu carrier activities and accelerating endothelin-1 and actin isoforms activities, that lead to simulating aromatase proper activities in the availabilities of ribosomal ATPase functions.

Thus, the binding of the myosin heads to the muscle actin is a highly regulated process controlled by tropomyosin isoforms activities, by endothelin-1, and by sestrin- Leu carrier activities and their AMPK & TOR peptides functions.

The fin filaments are made of G-actin with specific fine arrangements of amino acids in the brain to its structure compositions contains Tyr, Gly, Gly, Phe, & Leu for facilitating brain receive and response messages to tissues and to accelerate Leu activities through sestrin Leu carrier activities tools, for controlling muscles flexibilities.

G-actin filament structure consists of two lobes separated by a cleft. This structure represents the “ATPase folds” where their functions control muscle flexibilities.

Tropomyosin isoforms are synthesized to be joined to G-actin isoforms for controlling their activities through controlling their ATPase activities, which are the main tool for G_actin isoforms functions in biological processes.

Cytokines are one of the modulator's keys of inflammations, participating in acute and chronic inflammations via a complex and sometimes contradictory network of interactions.

The availabilities of antigen beta subunits synthesis are linked to their alpha subunits synthesis and activities, and linked to antigen compositions, which have approximately free 45 to 15 amino acids tail or free chain connected to their main long sequence which covering their living cells.

The antigen composition is controlling their actin isoforms compositions and functions, but some other cells are controlling tropomyosin productivity which controls G_actin functions, through decreasing ATPase activities. The deficiency of some amino acids in the antigen composition will affect the synthesis of their actin isoforms and will affect their functions including oxidations, deliveries, and leads to blood platelets aggregation too.

The stimulation of actin isoforms functions will stimulate MAPK pathways and their protein, they will be the stimulator for PPARs genes functions, that any inhibition or delay in one of the previous steps can be the reasons of reducing the sensation of cellular deliveries, and accumulation of +ve peptides in interstitium fluid between cells, and in capillaries, then will be the results of tumor synthesis.

As capillaries and filaments "which are responsible for feeding gastric cells and composed of G_actin which controlled by tropomyosin " have blocked or contained blood clots, as will lead to isolations to those cells with their interstitium fluid and compositions & will prevent the feeding to those gastric cells area with their imp peptides and with necessary active amino acids for their biological functions.

Sestrins is the active Leu carrier tool (CLCT) for the activation, regulation, and conservator for meta anabolic energy for doing the stimulating work or exercise for the favor of immune efficiency, that help meta_anabolic processes which is the anabolic processes with generating energy for the favor of doing that exercise or works and in the same time for the favor of anabolic processes for whole neuron and hepatic cells.

sestrins that have three functional sites for each of its identified activities: mTOR regulation, AMPK suppression, and leucine binding & carrier functions are so important for recoveries from diabetes, cancer, and many of viral infections effects.

Deficiency in Leu cycles activities due to the deficiency in sestrin-Leu carrier activities, or due to deficiency in Leu pentapeptides functions from enkephalin in the brain, in cells, and Thrombin, will lead to a deficiency in AMPK protein activities and decreasing in TOR protein activities, that lead to decreasing in the molecules polarities, and will lead to activating the blood platelets aggregations, and activities, and will lead to Precipitation of some peptides in interstitium fluid between cells, and in capillaries and then will lead to isolation to those sick cells tissue, that'll be the results of tumor appearances or results of dysfunction in those isolated cells tissue.

Only when TGF-B is lacking Leu functions activities or lacking the sestrin_Leu carriers activities and their active linkages, will stimulate endothelin-1 productions throughout the expression of the NF-κB" from the sick TGF-B.

What is the fibrosis?

Fibrosis is the death of main old cells due to a severe decrease in the main of imp genes or hormones functions and due to decreasing in interstitium fluidity compositions and functions, and due to preventing deliveries genes & peptides supply to those Fibrosis cells and tissues across filaments and blood capillaries.

Fibrosis starts by decreasing in Leu_pentapeptides activities in the brain with increasing in methionine pentapeptides functions due to decreasing in Leu activities with its carrier sestrin, and increasing in methionine that its functions related to the presence of sulfur.

Together with cysteine, methionine is one of two sulfur-containing proteinogenic amino acids, that the sulfur is the element that important for blood synthesis and tissue biosynthesis. Overconsumption of methionine, the methyl group donor in DNA methylation, with an acute deficiency in Leu activities is related to many health problems including cancer.

Both methionine and leucine are so necessary for blood synthesis and cell proliferation under the control of availabilities of necessity cycles and AMPK protein with MAPK pathways activities.

The lack of sestrin Leu carrier activities with increasing in Meth activities lead to decreasing in proper fine fluidity and constraints in interstitium fluid between cells, and that lead to blockage in the actin filaments and blood capillaries that can be are the main reasons for tissue fibrosis, that considered to be a definition of some cancer problems.

Gastric glands considered that are containing foveolar cells, chief cells, parietal cells, G cells, and enterochromaffin-like cells (ECLs).

There are three types of gastric glands, distinguished from one another by location and type of secretion in gastric glands, that all must have serine, aspartic, and glutamic acids for pepsin productions.

The (G cell) gastrin cell, is a type of cell that secretes gastric fluid, that actin filamentous and capillaries are penetrating the gastrin cells layer. It works in conjunction with gastric chief cells and parietal cells. G cells are found deep within the pyloric glands of the stomach antrum, and occasionally in the pancreas and duodenum, all must contain.

The amino acid composition of G_actin isoforms in normal gastric muscle layer contains a specific arrangements number of Tyr, Leu aspartic acid, isoleucine, and serine amino acids, while gastricsin contains a significantly higher number of glutamic acid for resynthesis pepsin which secreted by gastric garlands, where those actin isoforms functions act as a filter through translations & transcriptions to genes and peptides which are reached by blood vessels for pepsin resynthesis by gastric gland cells.

Why gastric cells contain a significantly higher number of glutamic acid not Tyr amino acids for resynthesis pepsin?

Tyr amino acids under specific internal diseases atmosphere can convert to tyrosine hydroxylase which able to cause blood platelets aggregation and lysis to Gastric cells, but glutamic acid can form layers to protect gastric cells to resist Tyr hydroxylase effects if found.

The arrived genes with the structure of a proper peptide from blood vessels "capillaries and actin filaments" will distribute across tissue cells for feeding their interstitium fluid which contains actin isoforms with their ATPase in G-actin loops, that will stimulate ET-1 for starting its functions for removing and functioning toxicity including any inflammation molecules, and then will stimulate MAPK pathways functions then will stimulate PPARs genes functions.

During activating MAPK pathways and PPARs activities in the interstitium fluid between cells, the translations & transcriptions will occur properly for pepsin production, which secreted by gastric glands, but Leu active function has to be clear in proper availability.

In the case of cancer, as gastric blocked capillaries increased or as +ve linkages increased in interstitium fluid "that will lead to Blockage in capillaries", as the isolations to those cells metabolic processes regions will start, that indicates there is a problem with interstitium fluid and with G-actin compositions and functions, that will lead to decreasing in the getting rid of the unwanted toxic results from the isolated interstitium fluid and their cells.

Cancers can be considered to be as results from mutations in actin isoforms compositions, blockage in tissue filaments & capillaries, and decreasing in the proper functions of alpha & beta subunits which are related strongly to sestrin-Leu carrier activities in interstitium fluid or their main endothelial tissue process, and decreasing in the ET-1 functions in some cases in gastric muscle interstitium fluid, and in mucosal cells, that will be the main reason for tumor formation in those tissue cells, that those isolated cells will lack the receiving any support from any other adjacent gastric cells and other adjacent tissues.

The increasing in +ve linkages in interstitium fluid between cells will help in capillaries blockage and will inhibit G-actin, and lead increase the availabilities of bonding tropomyosin isoforms to +ve elements or +ve molecules, that will inhibit & hold the sensation delivers & signals transmission and also will inhibit or decrease the ET-1 functions "not availabilities", and will lead to decrease in the activation the MPAK pathways which consequently needed for PPARs activations.

Also, inhibition in G-actin isoforms functions with a deficiency in AMPK proteins will decrease both PPARs activities specifically beta subunits functions and its productions in interstitium fluid between gastric cells, that will lead to blood clotting in capillaries then will hold most of the metabolic processes in the region of the isolated cell or the isolated tissue, then will be the main reason for tumor formation in that isolated tissue cells region.

The isolations of some gastric cells due to increase in blood platelets aggregation and due to Blockage in capillaries will lead to preventing the supplies from immune to that isolated cells, and if cells need any necessary amino acid will not be able to receive it eg Tyr, aspartic acid, isoleucine, Gly, Leu, glutamic acid, and serine in actin isoforms, in ET-1, and PPARs genes in filaments and the interstitium fluid between cells, that will not be able to reform the proper pepsin, and also cancer can start.

Actin contains these residues Tyr143, Ala144, Gly146, Thr148, Gly168, Ile341, Ile345, Leu346, Leu349, Thr351, and Met355, which are predominantly hydrophobic. This cleft constitutes the major binding site for most ABPs and also mediates important longitudinal contacts between actin subunits in the filament **(**39**)**. Notice, the availabilities and arrangement of amino acids in actin is almost the same as enkephalin Leu pentapeptides which are: Tyr, Gly, Gly, Phe, Leu.

That indicates the strong relations between enkephalin and actin in gene functions including Leu functions in neuron and tissue cells, also indicate to me the necessity of sestrin-Leu carrier activities for actin functions and muscle flexibilities.

Aromatase, which is the key enzyme for estrogen productions, is comprised of at least ten partially tissue-selective and alternatively used promoters. These promoters regulated by distinct signaling pathways to control aromatase expression and estrogen formation and G-actin peptides isoforms functions then stimulations the ET-1 production that consequently will stimulate PPARs pathways for PPARs genes functions via recruitment of various transcription processes for estrogen production. A shift in aromatase promoter that responsible for the excess estrogen production seen in fibroblasts surrounding malignant epithelial cells in some breast cancers (

that the increase in estrogen in cancer tissue regions is not the meaning that its the reason for cancer diseases but it is a sign of the isolation of those cancer cells with their interstitium fluid from other tissue and natural cells).

Also, the Targeting of those distinct previous pathways steps for stimulating and modulating aromatase enzyme will help only to understand the origin of tumor in cancer, but targeting the recoveries from blood clotting which occurred in filaments and capillaries then rebuild the peptide isoforms & ATPase in the G_actin loops will be so helpful for recoveries those tissue cells from cancer problems and viral infection problems. After rebuilding proper blood fluidity in filaments and capillaries we will be able to support sick tissue cells with the missing amino acids needed for reactivating G-actin peptide isoforms again for stimulating ET-1 production than for activating MAPK pathways than for the stimulation of the PPARs genes functions for their full pathways steps functions. In some tissue which are containing a unique G-actin peptides isoforms compositions that are responsible for estrogen and aromatase expressions.

The coding region of aromatase contains nine exons (II–X) with the ATG (meth amino acid), that as the Meth amino acid in the coding region has been missed or mutated, as the expression of aromatase and estrogen will be inhibited, But as the coding region is in a proper composition but there is a blood clot in that tissue filaments and capillaries, as the isolation of that tissue cells will begin with continuing the expression of aromatase and estrogen inside the isolated tissue cells.

The beta subunits don't have only the antigenic protection & translations function to living cells, but also have Offensive imp functions even if are not having mobility function & specifications as gastric cells.

When B subunits "that is the main for an antigen which covered gastric cells", in interstitium fluid" are lacking some their necessary amino acids as Leu, Tyr, Gly, and isoleucine & lacking the necessary active -ve bonding energy linkages with specific active elements (eg fluorine or and phosphorus), due to deficiency in AMPK protein functions and due to decreasing in ATPase activities, will lead to decreasing in antigen synthesis and functions and decreasing in actin isoforms and will be the results of precipitation peptides in interstitium and capillaries.

We know that, RITUXAN targets and attaches to the CD20 protein & found on the surface of blood cells with cancer and healthy blood living cells. That I consider that RITUXAN is imp for alpha and beta subunits resynthesis, that are containing helpful amino acids for antigen

synthesis, and for recoveries the proper compositions of interstitium fluid for rebuilding any damaged of cellular metabolic cycles.

RITUXAN can be defined as a type of antibody that can be used for antigen recoveries, and recoveries any missed amino acids in interstitium for actin peptides isoforms recoveries, & beta & alpha subunits recoveries, and ET-1 reactivities.

The immune system normally works to protect the body from infections by reactivating beta subunits production causing inflammation through increasing beta subunits synthesis, which will lead to increasing the antigenic protection & increasing in their free connected amino acids functions.

Beta cells (β cells) are a type of cell found in pancreatic islets that synthesize and secrete insulin and amylin. When beta-cell mass and function are diminished will lead to insufficient insulin secretion and hyperglycemia,

Where the most imp functions of beta subunits are recovering the missing or the damage alpha subunits.

But in some cases, a group of B-cells makes proteins which attack the body's tissues cells not by mistake but it is only when interstitium and antigen are having much lower binding energy lower than B_cells with a deficiency in the – ve long linkage " eg phosphorus and fluorine linkages" for the previous mechanism for fast-acting on toxic viral effects & any infections, and also for antigen resynthesis for more cells protection.

The life of a T cell begins in the thymus, where immature cells proliferate and create a wide repertoire of TCRs through recombination of the TCR gene segments. A selection process then begins, and T cells with strong reactivity to self-peptides are deleted in the thymus to prevent autoreactivity (as they have unlikely amino acids to original antigen) in a process called central tolerance.

1T cells with insufficient MHC binding undergo apoptosis, but those that can weakly respond to MHC molecules and self-peptides are not deleted and are released as naive cells to circulate through the blood, spleen, and lymphatic organs and specific amino acids are playing so imp roles in every cell antigen activities & adding more or altering those free amino acids in antigen will be résults of different functions or extra functions.

Results

Sestrin Leu carrier activities with the proper AMPK and TOR proteins is the main tool for functioning and reactivate fibrosis tissue again. But Leu pentapeptides functions and activities in enkephalin in the brain are the main regulators for leu_ pentapeptides which is related to polarities and phosphorylations functions by G-actin filaments and by ribosomal ATPase activities. Also, the disappearance of Gly amino from leu_pentapeptides will be main for fibrosis in tissues, and also, the disappearance of Tyr from Leu pentapeptides will lead to inhibition in antigen resynthesis and activities and lead to many diseases including fibrosis, heart disease and severe diabetes.

The dysfunction or decreasing in G-actin isoforms activities, decreasing in signal transmission, decreasing in ATPase and MAPK pathways activities, then decreasing in endothelin_1 activities, and increasing in tropomyosin isoforms functions, that'll lead to accumulation of undelivered peptide molecules in interstitium fluid in that tissue cells region and will be the main reason for capillaries blockage and Atherosclerosis and their obstructions.

RITUXAN is imp for alpha and beta subunits resynthesis which are containing helpful amino acids that helpful for antigen synthesis and for recoveries the proper composition of interstitium fluid for rebuilding any damaged cellular metabolic cycles.

RITUXAN can be defined as a type of antibody that can be used for antigen recoveries, and recoveries any missed amino acids in interstitium for actin peptides isoforms recoveries, & beta & alpha subunits recoveries, and ET-1 reactivities.

secreted glycoproteins (called lipocalins) are important in the CTLA 4 synthesis, functions, and in the maintenance of health and in combating diseases effectively. A prototype of this family is a small, secreted glycoprotein called Neutrophil gelatinase-associated lipocalin or NGAL are important for phosphorylations for antigen synthesis means are mainly important for beta subunits synthesis and functions for several antigen structures in different tissues specificities.

Changing & mutations in antigen composition structures will lead to changing & mutations in actin composition structure that will reflect blood Coagulation in capillaries and filaments, where amino acids in mutated antigen and G-actin will not match together for accepted translation for the favor of living cells that will lead to Precipitation of unreached peptides in interstitium fluid and on filaments and in capillaries. The main element for helping and accelerate tissue synthesis is sulfur that normally binds to more than three peptides Chains for cells & antigen synthesis,

where the increase in sulfur than normal will lead to accumulation and precipitation in blood vessels, and missing in proper translations process by CTLA-4 and by other beta subunits. Also, increasing in the breaking sulfur imp active bonds in biological mol more than normal may lead to breaking & loosen tissues & the bleeding in that tissue due to increase in blood fluidity, that in briefly sulfur increase thrombin synthesis, but fluorine are so imp for breaking sulfur bonds with the condensed peptide in blood for increasing blood fluidity.

The type & sequences of antigens with a free amino acids tail are reflecting their functions and tissue, As the main amino acids in hormone molecular composition are :Tyr, Île, Gln,, Cys,Gly, Gly, Phe, & Leu, The presence of Tyr inside & at the head of the free amino acid chain in antigen chain means is for controlling all gene chain functions (that the tRNAs formed from antigen can act as anti-inflammatory tools and as antiviral nucleic acid, and the main amino acid in its compositions is Tyr).

In the case of Arteriosclerosis & clogged arteries, treatment can be throughout using peptide molecule contain "fluorine" and phosphate groups that will help clean capillaries from blood clotting and remove precipitations in the interstitium fluid, and for breaking precipitated molecules in capillaries and interstitium fluid.

In case of increasing efficiency of macrophages with increasing brain activities & immune cells effectively through availabilities & increasing this amino acids sequence :Cys+Tyr +Meth+Leu+Cys (TGC) + Cys+Gly +Gly +Leu+Gly+ Gly(GGC)+ Asp+ Ser+ Arg+ Ala+ Thr+ Ser+Ala+ Asp+Thr+ Ser+Tyr+ Ser+ Gly+ Gly + Leu + Tyr.

In case of increasing macrophages, and increasing in analyzing inflammations maybe by using RNA fragments containing this sequence : Leu+ Cys+Gly+Ser +Tyr + Gly +Gly +Leu +Asp+Gly+Asp+Gln+Cys+Thr+Asp+Ser +Tyr+Thr+leu+Gly +Tyr.

Deficiency in thymines will lead to the deficiency in hormones synthesis and dysfunction in controlling the main gene functions & controlling translations processes.

But decreasing in the % of Cytosine will lead to precipitation of molecules in arteries and other tissues, plus a deficiency in hormones and CTLA-4 activities and will lead to a deficiency in tRNAs productions & inhibition in translations functions.

Cysteine (TGC) and Tyr (TAC) are so imp amino acids for tRNAs synthesis, but the function of tRNAs will depend on the type and arrangement of conjugated amino acids in the chain arrangement sequence in tRNAs.

The decreasing of Cytosine with guanine in the active sites will reflect dysfunction in signal transmission, and dysfunction in the tRNAs and lead to tumor appearances in cancer.

Deficiency of Cytosine and Leu will reflect dysfunction in sestrin-Leu carrier activities, and deficiency in CTLA4 and brain activities also will reflect decreasing in tRNAs productions, and decreasing in the migration of molecules with inhibition in signal transmissions, that will be the main reason for tumor appearance.

Breast cancer can be due to Blockage in filaments and blood capillaries without any mutations in the isolated genes inside the isolated tissue cells region. But the missing in Tyr amino acids in G-actin isoforms and inner cells genes components will reflect a deficiency in endothelin-1 that reflect a deficiency in catalyzing inflammations and infection effects, and reflect a deficiency in the most of antigen functions for cells, also, reflects reduction in signal transmission with reductions in tRNAs productions, and reduction in immune effectiveness.

If there is disappearance in Gly (GGA), or Arg (GAG) lys (AAG), and Leu, will reflect decreasing in brain pentapeptides activities, and in neuron activities, and dysfunction in Leu activities and sestrin_Leu carrier activities & processes, that lead to many diseases problems including diabetes, heart disease, and cancers. Also, Sestrins is the active Leu carrier tool (CLCT) for the activation and regulation of many necessary metabolic processes, and sestrin considered as conservator for meta anabolic energy for doing the stimulating work or exercise for the favor of immune efficiency, that help meta_anabolic processes which is the anabolic processes with generating energy for the favor of doing that exercise or works and in the same time for the favor of anabolic processes for whole neuron and hepatic cells.

Sestrin Leu carrier tool is necessary for running necessary metabolic processes and control endothelin-1 and AMPK protein activities, and regulate blood fluidity and lipid metabolism under the control ribosomal ATPase and MAPK active pathways.

Cytosine is controlling most of the cell functions, and tissues cycles, including macrophages, but at the same time Cytosine is controlled by Thymine nucleotides in specific active sites.

The appearance of tumor in cancer indicates the disappearance of Cytosine that reflect inhibition in migration of molecules with a blockage in filaments and capillaries in that tumored tissue region, and decreasing in immune effectiveness, also will reflect inhibition in sestrin Leu carrier activities and inhibition in ATPase activities, that lead to Blockage in the capillaries and isolation to tissue cells in the tumor tissue area.

Monocytes can block macrophages, and at the same time macrophages can kill & break monocyte.!!!!

That monocytes are the main for macrophages synthesis due to sestrin_Leu carrier activities, but if monocytes will loose & miss the necessary Leu amino acid and Tyr amino acid for its protection and functions, then macrophages will Devour those monocytes easily.

Deficiency in sestrin_Leu carrier activities will lead to NFκB productions and platelet aggregations, that can lead to, capillaries blockage, diabetic problems, and arteriosclerosis, and stimulations to endothelin-1, then the tumor formation problems.

Cytochrome P450 (CYP) 3A4 is responsible for catalyzing six-member molecules. Aromatase is one of the cytochrome P450 family that oxidize & catalyzing many vitamins and six members cpds for cells anabolic and catabolic processes.

The aromatase enzyme is fully dependent on ribosomal ATPase functions and MAPK pathways activities, that decreasing in ATPase activities will decrease aromatase activities and increasing in ATPase and MAPK pathways functions will increase aromatase activities. But decreasing or inhibition in ATPase, in MAPK pathways activities, and aromatase will lead to inhibition in signal transmission and inhibition in G_actin activities accumulation of peptides molecules in interstitium fluid and capillaries, leading to Blockage in filaments and capillaries, will lead to isolation to that part of tissue cells, then will lead to the formation of tumor and cancer.

Cancer sometimes occurs in tissues of secretions of glands or hormones where there are clusters of capillaries wrapped around each other that each of them having a connection with other specific tissue for sending & receiving messages, that at the time of blockage in that specific capillary will prevent the sending and receiving feedback between the two tissues cells, leads to dysfunction in that capillary and its connected tissues. I want to say that each of those filaments (the main of capillaries) is connected to & between specific tissues for performing only specific functions with a specific cycle in the favor of specific function for its connected tissues cells and from immune cells,. for example, there are some capillaries connected to the brain to liver &

pancreas separately each connected to other specific tissue, each of those filaments is for a specific purpose and functions, if a blockage occurs in one of those filaments, will block & inhibit its connection functions, and will affect on its connected tissues function (that who are doing surgery to remove cancer tissue, their tissue filaments that had been controlled to other tissues had been removed from Connection that later will be the result of other connected tissue. .And I have to report that the bases of filament synthesis are the G_actin which represents specific arrangement part of nucleotides in 1st DNA strand only, but tropomyosin is formed from translations processes from G-actin and represent specific arrangement part of nucleotides in 2nd DNA strand and controlled in their synthesis by G_actin that at the time of exited or stimulated muscle is the ATPase will be activated for the beginning of translation for tropomyosin "Tr" isoforms synthesis than those Tr. Isoforms will be bind to Ca+ that will reduce the ATPase functions and G-actin isoforms functions.

The G_actin isoforms and their functions with tropomyosin are the main root of capillaries compositions, that start first its formation of filaments, then will construct capillaries, and later arteries and vain, that if filament will be blocked, will affect on capillaries and arteries that will be blocked too and their +ve molecules will be precipitated in their wall too. Thus I started to study to treat cancer from G_actin and their tropomyosin and endothelin-1 to be reduced or to be activated in the favor of activating MAPK activities and their AMPK protein in the presence of sestrin-Leu carrier activities with the proper activation of ribosomal ATPase and cytochrome for PPARs activities and blood functions in the availabilities of proper blood fluidity for all patients body. "

Most of the severe health problems including cancers and viral infections are mainly due to Blockages in blood filaments, in capillaries, then arthritis and or veins blockages, and to start treating first have to reactivate blood vessels blockages, even it can be started by treating by carefully using – ve long-distance rays to clean fine blood filaments from blockages followed by biological treatment to insure removing the blockages from filaments and capillaries and then increasing proper blood fluidity, then can reactivate blood circulations in proper fluidity to feed & recover sick tissue cells system again with all their necessary cycles & enzymes and peptides supplies from other cells in proper pathways.

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