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Microbiology its Bearing on Immune Health and Cancer Genetics

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Abstract

Our immune system whether innate or adaptive, requires the activity of a number of cells, whether thru the phagocytic mechanisms, or the release of special factors of various kinds (cytokines, lymphokines, or chemokines).

The immune factors thru immune cells may either stimulate, delay, hinder an immune reaction, or serve by effector cells, to monitor an immune response. Genetic Mutations, either at the genetic or cellular level, can result in either an altered protein, overproduction, or proteins that are diminished in function. Other cases result in unwanted proteins which can lead to overzealous cellular activity, resulting in cells that have lost their contact inhibition, and become cancerous. By beginning to understand the operation of genes, as well as how cells function, cellular metabolism, a metabolic pathways, and the microbiome, these new understandings will lead to better treatments for immune disorders, neurological disease, and cancer therapies. The article is in a effort to make available new information on immune function, and metabolic pathways which may lead to immune dysfunction, mutation, neurological disease, genetics and ultimately cancer.

Keywords: *Microbiology; Immune Health; Cancer Genetics*

Engineering Cells

Today engineering has developed new types of solar cells, and the ability to create photosynthetic cells, that are able to function in the same manner photosynthetic pigments function, in various plants [1-3]. Scientists are also developing new ways to follow the activities of cells at the molecular level [4]. They have revolutionized not only the way we look at the world, but also the very way we look at each other, and a better perspective on our body, and particularly the cells that scan our body, and protect us from harm [5,6]. These important cells that originate in the bone marrow are called stem cells, that give rise to all types of cells that make up the tissues, and bones of the body [7].

Innate and adaptive immunity

Our main protection relies on both innate and adaptive immunity. Innate immunity is similar to a general reaction to the presence of foreign antigens, that stimulate an immune reaction. Innate immunity involves a number of generalized protection mechanisms, protections such as the barrier of the skin, chemical barriers (saliva, tears, gastric juices, and body fluids), and another type of immunity called cellular immunity. The presences, and activity of phagocytic cells (WBC's), which impart either inflammation or fever, play their part, in the immune response [8,9]. Other significant ways of protection include the presence of interferon and complement. All these interactions are important in the first line of defense, with innate immunity [9].

Microscopy and noncoding RNA

Recent research on innate immunity by Cryo-electron microscopy, has unveiled a unique important pathway used by innate immunity, and called STING (Simulator of Interferon Genes), the structure of the smallest membrane protein, and an algorithm that boosts microscope imaging [4,8,10,48]. Other fascinating news on innate immunity is the discovery of noncoding RNA called nc886. This RNA is important in that it can respond to the presence of viral microorganisms, by activating a "signaling pathway," by a protein called OAS. OAS can thus lead to a second activator, and to viral destruction. The unique aspect is that nc886 can exist in two forms, one active, and another inactive. Its ability to be in the active state, depends on it three dimensional shape. Although nc886 is present in all human cells, its unknown which form its 3-D molecular shape predominates, in human cells [10,11]. RNA (DINO) also is important, in that it binds to a suppressor protein p53, and helps repair a damaged DNA. However, p53 can be a serious problem when mutated in related cancers [12].

Cellular differentiation, stem cells and environmental influence

What about our cells in general. We know that all cells originate from the bone marrow, and undergo a series of transformations, and differentiation to become mature and functional cells. These cells arise from stem cells, and their progeny can now be followed in their journey, towards identity and maturation. In a study of 40,000 embryonic zebra fish cells, Alex Schier used a new software called (URD). His research provided for a method (high-throughput single-cell RNA sequencing method), for following active genes during "cell development." This method has provided a way to identify all the active genes that occur during a fertilized embryo, and with its map of origin. This study therefore suggests that signals from the environment can have a strong influence on the path that cells can take. Cells can thus have some flexibility to change their path and identify, its path is therefore not as restricted as once thought [5,13].

Mesenchymal stem cells and cell differentiation

Other aspects of stem cells that are very important in the function of cellular repair, are the mesenchymal stem cells. Important because they function primarily in the repair of "damaged organs." They also function in the repair of complex tissues like "cartilage and bone formation." The cell-membrane proteins called caveolin-1 and N-Cadherin, are significant in a process called condensation, a step that "chondrogenic differentiation" (cartilage repair) and bone formation [14,50].

Some current research on cell differentiation has focused on the hunchback (hb) gene. Researchers have been able to follow how a gene can be turned on or off. They discovered that a gene called hb could be turned off, when its promoter switch is turned off. In this case the hb promoter can be turned on by a protein called Bicoid, when it binds to an enhancer (different DNA sequence). When the bicoid protein makes contact with the hb promoter, this contact can turn the promoter on, and then the gene will produce an RNA copy. Thus, this research provides insight into the process how a first "cell-specific gene" has turned on a particular region, during of early embryonic development" [15].

Stem cells and neurodegenerative disease

Stem cells are also on the forefront for the treatment of neurodegenerative diseases, such as dementia and Alzheimer's disease. Researchers in the past have used stem cells to produce astrocytes, as a treatment, but this can be a cumbersome, and a complicated process in the laboratory. In a recent research article by Lund University in Sweden, their investigation has shown that astrocytes can be generated by the gene editing tool CRIISP-Cas9. In this approach, a virus is used to insert genetic information into embryonic stem cells, in order to generate functional astrocytes [16]. Human blood cells can now be used as neural stems and resembling "neural stem cells (during embryonic development of the nervous system). Therefore, even human blood cells can be used to convert human blood cells, into different nerve cells, such as "peripheral sensitive nerve cells" and or cartilage, and bones of the skull [17].

Cell protection strategy

Some other aspects that are important to cells, depends on whether they are somatic, or stem cells during cell division. Our cells during mitosis need to protect their chromosomes during cell division. They do this thru the protective help of the protein PLK1. Not only is PLK1 important in stabilizing chromosomes during mitosis, but it also is important in maintaining the "rigidity of the centromere," which is critical during cell division. PLK1 thus, plays an important function of proper partitioning of chromosomes during cell division, and by helping to maintain the functional health of centromeres, during mitosis [18].

Now that we're aware of some of the protection methods that cells perform during mitosis, and the nervous system. Much like normal cells, which protect against free radicals, some microorganisms must also have mechanisms in place, to solve similar problems during metabolism. One similar mechanism that yeasts undergo, occurs when they accumulate a high level of lysine, resulting in a "reconfiguration" and production of excessive amounts of glutathione. With the accumulation of glutathione, this molecule has a ravaging attack on free radicals. This allows for the elimination of free radicals and could result in cell death otherwise [19].

Superantigens

In another interesting mechanism, pathogenic bacteria like *Staphylococcus aureus* and *Streptococcus pyogenes*, are able to breach the mucosal surfaces by the cell surface protein molecule of CD40 (also on antigen presenting cells), when it reacts with superantigens, and can result in the release of chemokines. The release of chemokines can disrupt the integrity of the mucosal surface, and allowing for bacterial mucosal penetration [20,21].

Microbial involvement in innate immunity

The immune system, how does it react during an infection? The immune system defends itself by producing neural chemicals in the brain, particularly acetylcholine, allowing T-cells to be able to enter the blood circulation, and begin to act on tissues, and the infection [22]. Complement is also an important player in the fight against infections, because it is a part of the innate immunity. However, as a major player in immunity, as with many systems of the body, its activity must be monitored, in such a way as to prevent cellular damage. This can be accomplished through a "complement control protein" called Factor H. Factor H occurs freely in the blood circulation and protects cells from attack from the activity of complement. Factor H occurs on the surface of some pathogenic microorganisms, particularly Group A streptococci. This Factor H allows bacteria to avoid detection, and removal by the immune system. Recent research at Lund University has found that Factor H "fusion protein" can serve as a (treatment from gram-negative infected mice), to remove Factor H from the surface of bacteria. Fusion protein has the capability of serving as a broad spectrum antibacterial agent, as an asset that can work against both gram and gram positive infections. In this way, this protein could help in the functionality of the immune system and reduce the severity of an acute sepsis infection [23]. Along with the ability to regulate the activity of complement by Factor H, scientists at the University of Turku have discovered a way to regulate T cells of immune system. Proteins such as SATB1 are important in that they function by "regulating the transcription of numerous genes." This research could provide new clues into the treatments of immune-mediated diseases such as multiple sclerosis or rheumatoid arthritis [23,24,51].

Macrophages in a dual function

Other forms of immunity that are commonly seen as a result the presence of infection, namely occur under the operation, and function of macrophages as a part of the immune system. Macrophages have a dual function, namely to incite inflammation, and or damping inflammation (anti-inflammatory). However, in some instances, inflammation can sometimes get out of control in inflammatory diseases, as has been shown by recent research using mouse models, during acute lung injury. This research has found that when macrophages become depleted, an inflammatory condition may not be resolved, due to the loss of the anti-inflammatory protein Gas6. However, when Gas6 levels can be "artificially boosted," the associated inflammation was much quickly resolved [25]. Other lesser known types of T-cells are found in association with esophageal cancer. These T-cells are called MAIT (mucosal-associated invariant cell), and are decreased in these cancer patients, as compared to controls. MAIT cells can kill cancer cells *in vitro* but are reduced in their activity from "tumor biopsies."

It seems possible; by using MAIT T-cells it could be possible by this mechanism to "reverse" the inhibition of esophageal tumor cells, by providing new and better treatments, and outcomes for esophageal cancer [26].

Magic bullet for Cardiovascular disease?

There seems not to be a magic bullet, that can promote better overall health, but scientists seem to believe that a metabolic protein called AMPK might do the trick, for helping in the problem of diabetes, cardiovascular disease, mitochondrial disease, as well as to extend life expectancy. AMPK may not be a cure for all, but it does seem to change the activity in the tissues of the body, particularly of how fats are metabolized. This could be a plus, for those suffering from diabetes, obesity and or heart disease [5,27].

DNA mutations

Recent developments in the research of cancer, scientists at Baylor College of Medicine, have been studying the importance of DNA damage, due to genetic mutation. Their research has focused on using *Escherichia coli* as a method to search for changes in genes, as a result of mutations. This researcher has described cancer as a disease of mutations [28]. In this experimental therapy, engineered nonpathogenic bacteria were used to combat tumors. This therapy was engineered for targeting a protein called CD47, which is comely associated with tumor cells. Tumors use this protein in order to be overlooked by the immune system. Although not a new approach to cancer therapy, it does have some advantages. For one fact, the bacteria are specific for "colonizing tumor tissues. This type of therapy also helps to reduce side effects that can occur after treatment targeting cancerous tissue by CD47, particularly during clinical trials. This use of bacteria to target tumorous tissue with CD47 can stimulate phagocytosis of cancerous tissue, as well as an increase in the number, and proliferation of T cells, within the tumor tissue itself [28,29].

Bacterial DNA as a template for unique genes

The search for better antimicrobials, researchers are seeking to reawake silent genes. A new approach has been to use the technique of CRISPY technology, to un-mute "silent gene clusters," by injecting DNA fragments that are of interest. In this way, by "pulling away repressors which can prevent gene expression. In the hope of discovering new silenced genes, that could be useful as antibiotics, or anti-cancer candidates [30]. Bacteria (like *Pseudomonas*) offer many alternate biochemical pathways, that can make their "abundant genetic information" available, for the formulation of pharmaceutical drugs, peptides, and important molecules thru a genetic pathway-like fashion [31]. Harvard Medical School's research on unique genes that make up the microbiome, can serve as a blue print, on how a person's microbiome can provide insight, as to previous exposure, from various pathogenic bacteria, as well as an environmental influence [32].

DNA methylation

Other ways of studying gene regulation, have revolved around the mystery of DNA methylation. Researchers have known in the past that DNA methylation is important in regulating cell functions. Their new experimental model is a method that can characterize methylation patterns by "artificial methylomes. In the hope, to determine the methylation pattern of a particular strain of bacteria, that will be introduced as bacterial DNA. This can be experimentally done by using DNA rings or plasmids, containing one type of methyltransferases, with multiple copies of "certain DNA patterns (motifs)." By using both the plasmid with methyltransferases, and copies of particular DNA, the methyltransferase can be transferred by the plasmid, and reveal the "enzyme's methylation pattern. This experimental method in essence can provide a way, to identify methyltransferase methylation patterns, and the ability to understand the "regulation of gene expression, and cell differentiation [33].

Gene modulators

The use of ARID1A (protein cancer suppressor) could make it possible for treatments for a variety of cancer. This is because without ARID1A, not all cells are able to recover after radiation. Treatments with radiation and PARP inhibitor were also not found not to be effec-

tive. However, at John Hopkins Medicine, they discovered when tumor-bearing mice are exposed both to irradiation, and anticancer poly ADP ribose polymerase (PARP inhibitor, ARID1A), ARID1A-deficient tumors were dramatically reduced. By this research, they also found why radiation treatment alone is not always effective. They concluded that with these types of resistant cancers, cancer relies on homologous recombination, and not nonhomologous end joining [NHEJ). This is because the NHEJ pathway is affected by radiation, whereas after radiation homologous recombination is not affected. Thus could explain why radiation alone is not always sufficient for treatment [34].

New drug delivery system

Researchers at the University of California-San Diego have developed a delivery system, utilizing tumor-associated macrophages (TAMs). By using avβ3antibody LM609, this antibody can seek out tumor associated macrophages (macrophage associated tumor tissues), that display the avβ3 receptor, in a way to recognize, and kill avβ3 expressing tumor cells. This approach should help to reduce in the treatment of more aggressive, and drug-resistant tumor cells [35].

New drug in combination with SOD1 and Side affects

In relationship to neurological diseases, scientists have found that the production of synaptotagmin 17 (syt-17), can help accelerate axon growth. This provides a new avenue for the possible treatments of neurological diseases, and spinal injury [36]. In a recent developmental research on Lou Gehrig's disease (ALS), a new drug may have potential therapeutic treatment, for this severe neurological disease. The drug is called telbivudine, seems to reduce the "toxic properties of SOD1 protein, which is a protein that can cause misfolding with ALS patients [37].

Leukemia and neurological treatment new ideas

In the case of Leukemia, new research has found that although scientific investigation has primarily centered on the protein called β-catenin. β-catenin is responsible in driving tumor growth and is stored in the nucleus of cells. It helps to activate genes that are important for leukemia development. The significance of this research has shown that β-catenin needs the help of another protein called LEF-1, in order to actively control the level of β-catenin in the nucleus. More research needs to occur, but this research could lead to therapeutic treatments for this disease [38].

New possible treatments for neurodegenerative disease has centered on the identification of coiled-coil structure of polyglutamine toxic protein, which causes an entanglement of neurons, and a "rapid deformation of neurons as well." This damage that occurs within neurons, an early "neuropathy" occurs in conjunction with Foxo protein, which is an early identification of symptoms of neurological diseases. Just the identification of the toxic protein, and identifying the Foxo protein mechanism, should help in the development of new avenues, for the treatment of these, and other neurological diseases [39]. Research by the Netherlands Institute for Neuroscience - KNAW, their research has shown that gene therapy, in conjunction to surgery, can lead to faster recovery from nerve damage, when in conjunction with gene therapy [40].

In the case of Parkinson's disease, there is an accumulation of the toxic protein α-synuclein, which can result in the appearance of "defective astrocytes. The protein α-synuclein causes axons and dendrites to "shorten and disintegrate, even with healthy individuals" [41]. With acute myeloid and acute lymphocytic leukemia, a key protein called MLL can slow cancer progression, once when "stabilized" and possibly offer a new approach for treating breast, and prostate cancer [42]. Other encouraging research finding was discovered by Oregon State University in the regards to tumor growth. They discovered how the oxidant called peroxynitrite, alters the amino acid tyrosine, and changes the metabolism of tumor cells, enabling them to proliferate. This could provide a new strategy for treating tumors of the nervous system [43]. Another approach is to look how cells undergo cell death or apoptosis. Current work on the apoptosis of nerve cells may provide new clues in understanding neurological disease. The protein SARM1 has been found necessary for the breakdown of nerve cells. Its 3-dimensional shape should help to provide an understanding why this process occurs, and development of new drugs [44]. One

encouraging development is the break-through in the ability to "interface with brain signals. This will result in a better understanding of the brain, possible therapies for neurological diseases, more efficient ways of learning, and particularly for those with disabilities, and newer diagnostic machines [45].

T-cell relationship with neurological disease

A recent article in The Scientist, the author points out, that commensal microorganisms do play an important part in various autoimmune diseases. In the case of multiple sclerosis, T cells from MS patients were found with both versions of human and the bacterial protein guanosine diphosphate (GDP)-L-fucose synthase. Researchers believe that gut bacteria are responsible for activating T cells, that can attack the central nervous system. In the example of commensal gut bacteria and rheumatoid arthritis, some bacteria have been found to share "sequence homology" with two proteins that have been isolated from the blood of rheumatoid arthritis patients, and joint fluid. Antibodies and T cells have been found that react to both human, and bacterial peptides. In the case of Lupus, commensal bacteria from the mouth, skin and gut, produce an "ortholog" of the human protein Ro60. In essence, it is found that Ro60-specific antibodies from lupus patients, also bind to bacterial Ro60. This implies that commensal microorganisms are capable to a certain degree, in activating antibody producing B cells, and responsible in autoimmune disease. This has been described by the author, as "commensal mimicro" in autoimmune disease [45-47].

Conclusion

In Summary, the relationship between our normal flora can be a delicate balance, between protecting us from the incidence of disease causing microorganisms, and the cellular controls that prevent an overreacting immune system. This can result in out of control inflammation due to superantigens, autoimmune disease and or cancer. Our immune system thus, protects us from invading microorganisms, and fighting infections. Our immune system whether innate, which acts by either mechanical barriers, the presence of phagocytic cells, by the process of inflammation, fever, and complement, and or whereas adaptive immunity is carried out by our lymphatic B and T-lymphocyte cells [8,9].

Therefore, our immune system is a coordinated system that involves the action of a variety of cells, that must function together, and under some capacity under the influence of our microbiome. This can occur thru the action directly at the genetic level [5,15,46], such as thru the action of noncoding RNA (nc886), that signals DNA repair, or the destruction of invading viruses [10,11]. Our cells also need to protect themselves during cell division, and they do this in part, by protecting the integrity of the centromeres during mitosis, through the efforts of a protein called PLK1. Microbes particularly *Streptococcus pyogenes* and *Staphylococcus aureus* produce what are termed superantigens, which can breach mucosal surfaces, due to the presence of the cell surface protein molecule CD40, when they react with superantigens. This reaction leads to a release of chemokines and allows for pathogens like the *S. pyogenes* and or *S. aureus* to enter the gut mucosa [20,21]. SATB1 is also important in the regulation of T-cells, since it functions in the regulation and transcription of numerous genes [24]. CRISPY technology has made possible workings of what has been described as "silent gene clusters" that shed light on new alternate metabolic pathways that could lead to the formulation for new pharmaceutical drugs, peptides, and significant biological molecules [30]. There have also been new developments in the treatments of cancers, by developing new ways to arm the immune system, characterizing model microorganisms that can construct methylation patterns, thru artificial methylomes. In an effort to understand the "regulation of gene expression, and cell differentiation [33,49]. The immune system can now seek out tumors (tumor associated macrophages), that display the avβ3 receptor, by utilizing the avβ3 antibody [35]. Neurological diseases such AL (LouGehrig's disease), synaptotagmin 17 (syt-17) has been found to accelerate axon growth, and the drug telbivudine, seems to be helpful in the toxicity of SOD1. SOD1 is a protein that currently is believed to be responsible for the misfolding that ALS patients experience [37]. In the case of Leukemia's, β-catenin under the influence of the protein LEF-1, is able to activate genes that drive tumor growth [38]. With Parkinson's disease, there is an accumulation of the toxic protein α -synuclein, which is believed to "shorten and disintegrate axons and dendrites". However, a key protein called MLL, may slow the progress of acute myeloid, and acute lymphocytic leukemia [41,42]. Cancers have also been found

to alter the amino acid tyrosine, by the oxidant peroxynitrite, enabling tumor cells to proliferate. Understanding these and other genetic and molecular mechanisms, could offer new methods for treating neurological diseases [43]. One encouraging development is the breakthrough, in the ability to "interface with brain signals. This should result in a better understanding of the brain, possible therapies for neurological diseases, more efficient ways of learning, and particularly for those with disabilities, and newer diagnostic machines [43-46].

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