

The Mechanisms Behind the Paradigm of Dysbiosis-Autoimmunity

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Abstract

This Dysbiosis –autoimmunity paradigm may be thoughts as friend and Foe partners . The objective of the present , minireview was to elucidate the mechanisms behind the dysbiosis-autoimmunity paradigm. Dysbiosis can be initiated by prolonged induction with either one or more of the followings; Boron , mRNA vaccine ,infection ,inflammation ,antibiotics and aging. It is associated with a characteristic molecular and immune micro-environment encompassing; presence of defective allele encoding immune functions, intestinal barrier dysfunction ,tight junction impairment ,and translocant passage. On passing through the impaired intestinal barrier, the translocated pathobiont behaves in either of three ways ,as reach blood stream through intestinal vascular barrier ,reach lymphatic circulation through intestinal epithelial barrier or reach Peyres patches through M cell ,then, the interaction with immune mediator molecules and, immune cells such as ; brook of immune tolerance events, inflammatory-anti-inflammatory cytokine imbalance, T cell subsets imbalance as T reg./TH1/TH17. Memo-tope, epitope spreading , antigen bystander and stimulating production of autoantibodies or autoreactive cells possible pointing to autoimmune disease induction . Effector auto-reacting B cell may migrate to lamina propria to produce autoantibody there .Effector auto-reacting T cells and T reg. cells may migrate to remote tissue compartments or stay there in. This theme has well been documented in laboratory animal models but to what extent can be extrapolated to human is still a matter of debate.

Keywords: Autoimmunity, boron, cytokines, dysbiosis, epitope ,gut, memtope pathobiont

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1. Introduction

The oldest new entity of human body, the microflora residing in or on body compartments. The microbiome/microbiota has been advocated as a life long co-partner to the immune system[1]. The sharing parts of this life long correlation are the microbiome and the immune system in rather unifying recognition concept. Insults affecting this sharing co-partners has been found in a parallel cues of occurrence events as; microbiome symbiosis “Resilience Microbiome” came in line with immune homeostasis ,microbiome dysbiosis is being in line with infectious immune dysregulation .Though,microbiome dysbiosis are found in association with aging ,infection ,infestation, and autoimmunity .Infectious Immune dysregulation is being thought as it is consistent criteria for these disease states.The diversity, composition and function of the normal human microbiome in immune homeostatic state are different as individual variation holds in operation ,and different from that of human disease conditions .Such differences reside in alpha and beta diversities that forms the basis of microbiome signatures[2,3] .The mechanistic steps for identification of gut microbiome driven autoimmunity have been delineated by Miyachi et al.[4] as ; dysbiosis, microbial translocation, molecular mimicry, autoantigen overproduction and cytokine overproduction. The current information on the correlation between gut dysbiotic microbiome and autoimmunity are in an increasing frequency. Though there is some debate still exist .The objective of the present mini-review was to elucidate the mechanistic aspects of this correlation.

2. Review Plane

The nature of the inducer for dysbiosis is considered as infection. The chose microbiome was that of gut and the mechanistic analyzed autoimmune diseases were multiple sclerosis, rheumatoid arthritis and systemic lupus erythematosus. As well as the chose literature were published mainly from current reputed periodicals . The deduced mechanisms were encompassing genetic ,molecular and molecular immune nature.

3. Gene-Immune Functions

When we trace the scientific holdings of the paradigm of dysbiosis-autoimmunity it seemed suitable to brief gene-immune functions, since there are intimacies in between them. Five eligible terms came in the way that should be expressed. Namely, Gene Trait, Immune Trait, first genome, second genome and immune resilience. The structural profiles of the encoding genes for human being is known as genomes[5]. The sequences of the structural and functional genes are known as genomics. While, the amino acids sequences of the proteins resulting from gene expression known as proteomics. Collectively, the overall genotypic characters of human beings is denoted as gene trait and the overall immune characteristics of the human being is known as immune trait, "Immunome"[6]. In infection state the human genome termed as the first genome, while the microbiome genome known as the second genome during pathogenesis of the infection there could be some sort of first-second genome interactions[7]. The gene trait of the human twins as well as familial traits have shown similarities in their microbiomes, particularly for the phyla; Firmicutes and Verrucomicrobia [8,9]. Genetic variation directly can lead to disease phenotypes which in turn can trigger changes in the microbiome balance[8,9]. At the human immune homeostatic state, the individual "Immune Trait" can be defined as the overall proteomic profiles of its immune functions. The coincidence of gene trait and immune trait of an individual immune system, can be elucidated in its immune resilience ability to resolve infection via vaccination or to restore the immune homeostasis from the autoimmune condition[5].

4. Features

A Shift from the resilience balanced symbiotic gut microbiome signature to the an imbalanced microbiome signature, the dysbiosis is accompanied by an array of biologic features as in the followings;
i-Shift from normal immune homeostasis to infectious immune dysregulation, ii-Change in cross-talk mechanisms with mucosal epithelial and luminal immune cells, iii-Change in quorum sensing signaling and production of short chain fatty acids and amino acids, iv-Change in the microbial entities forming the microbiome in term of; composition, diversity, richness and function, v- Appearance of the dominant pathobiont[10], vi-Tend to translocate the pathobiont [10,11,12], iix-The possible causals are: mRNA vaccine[13], antibiotics[14,15], infection[16], inflammation [117] and aging[18].

5. Mucosal Barrier Impairment

Microbial adhesion to the mucosal epithelium may initiate a sequence of inflammatory responses by the prolonged activation of signal transduction through TLRs and zornulin signaling with net results of loss of integrity of tight junction TJ. Such chronic barrier dysfunction can cause passage of bacteria through translocation events in continuum with the inflammatory responses that take part in further damage of TJ barrier and the epithelial cell apoptosis via the action of the co-existing inflammatory cytokine production [19]. The intestinal patho-bionts at times were overexpressed within the dysbiotic microbiome in the patients with the inflammatory diseases where they accelerate systemic inflammation via the translocation across-epithelial barrier to reach the remote extra-intestinal tissues as in the case of *Enterococcus gallinarum* which frequently detected in the livers of autoimmune hepatitis[20]. In the post-translocation events of the patho-bionts in the body of the dysbiotic host disturbs the immune homeostasis through the induction of systemic inflammation. While, the other symbiotic entities of the gut microbiome are important so far confined to the intestinal lumen. These symbionts produce metabolic products that promote the proliferation and differentiation of intestinal epithelia and multiple immune cells like; Treg and TH17 cells[21].

6. Barrier Passage Patterns BPP

Gut mucosal barrier encompasses three physical barriers as; mucus layer MB, intestinal epithelial barrier IEB and gut vascular barrier GVB. Mucosal barrier MB contains mucus that play essential immune regulatory role. Mucin 2 induces toleragenic dendritic cells and host commensals that are key player in modulating surface immunity such butyric acid producing bacteria. Intestinal IEB is a single layer of epithelial cells integrated together with tight junction TJ, and adhesion junctions AJ that regulate trafficking of bacterial antigens, toxins, antigens food antigens across IEM reaching lymphatic circulation and initiate immune reactions GVB the structure that determine which microbial components can migrate from the gut into the systemic circulation[22]. Commensal bacteria as whole cell bodies may pass through local lymphatic circulation via IEB[23]. Gut commensals Rikenellaceae, Bacteroides, Streptococcus and Balutia pass through systemic blood

circulation via GVB [24]. Mucosal pathobionts may adhere to IEB M cells transported to macrophage with in the Peyer's Patches activated and activate T helper cells which in turn activate either B or T cells to develop into effector or memory lymphocyte subsets taking part in immune and autoimmune reactions local or distal body parts. Hence, there are three BPPs as; lymphatic circulation, blood circulation via M cell transport and mucosal mechanisms[25].

7. Molecular Genetic Mechanisms

Autoimmunity is outstandingly considered to be a result of interplay between genetic predisposition and environmental factors. Such interplay has been considered as heterogeneous and complex. The heritability of these diseases has been well documented and quantified. And exhibits three important features as; i- all autoimmune diseases have strong genetic component, ii- relatively large numbers of risk alleles are shared among multiple autoimmune diseases, iii- the products of most of the autoimmune-associated genes are parts of immunological pathways, in particular T cell signaling, TNF signaling or innate immunity[26]. The analysis of autoimmune diseases reveals a stepwise sequence of four rules that starts with; a- foundation of genetic architecture representing autoimmunity, b- chronic repeated skewed and biased responses over years yield a pathological system, c- the pathological system induces loss of immune tolerance and d- have noxious potentials. Based upon these four rules of conceptual landscape, there are four endeavours that will improve the understanding of the autoimmune diseases. These endeavours[27] were as follows; 1- How does the immune system behave before and after the onset of autoimmunity?, 2- How does the immune adaptation facilitate the development and perpetuation of autoimmune diseases?, 3- Which genetic element triggers pathological immune behavior?, and 4- How do local tissue factors modulate the function and survival of infiltrating immune cells[28].

8. Immunopathogenesis:

The gut microbiome take part in the development of the immune system through the maintenance of intestinal barrier maturation and regulation of immune cells via production of short chain fatty acids SCFA. SCFA producing bacteria have the ability to regulate immune cell differentiation and development of regulatory lymphocytes which are the cells that function for maintaining immune homeostasis and controlling immune responses[29]. The symbiotic microbiota produce a considerable amounts of fatty acids, hydroxyl fatty acids and indoles which have substantial impacts on host immunological functions via metabolic reprogramming[30] and epigenetic mechanisms[31]. The mucosal barrier encompasses; mucin, antimicrobial peptides, dimeric IgA, Paneth and plasma cells. Cellular and effector molecules of the mucosal barrier constitute a barrier between lumen bacteria and the intestinal mucosal epithelium to prevent bacterial adhesion to epithelium. Though, the impairment of mucosal barrier through the disruption of tight junctions often leads to enhanced permeability[32,33].

Dysbiosis impairs the epithelial barrier function and elicits proinflammatory cytokine responses. Like-wise, microbial adhesion to epithelial cells can induce pro-inflammatory cytokine responses which further damage the tight junctions integrity, leading to leaky gut syndrome LGS. LGS enhances the translocation of bacterial pathobiont to systemic circulation[34]. Pathobiont translocation stimulate local and systemic inflammatory responses that renders the animal host vulnerable to immune mediated and metabolic diseases[35]. Dysbiosis can induce loss of immune tolerance, overactivation of T cells and production of pro-inflammatory cytokines. These immune events can activate autoimmune responses. Current studies have shown an association of specific bacteria and/or their metabolites, such as butyrate producing bacteria with the development of autoimmune diseases. Dysbiosis may activate several different immune pathways resulting in the upregulation of pro-inflammatory cytokines; IL1B, IL6, TNFalpha, IL17, IL12 and IFN gamma and down regulation of anti-inflammatory cytokines; IL10, IL4, TGFB and IL1ra. It constitute a state of cytokine imbalance. Besides the triggering an imbalance of regulatory T cell subsets in continuum with other T cell subsets TH17/TH1. As well as the dysregulation of TH17 cell and Th17 cytokines production. Thus the net results of dysbiosis effect is cytokine imbalance and effector lymphocyte subsets imbalance[29]. At the mucosal surface barrier pro-inflammatory cytokine impaired the tight junction integrity while anti-inflammatory cytokines restore the tight functions. Hence cytokine milieu is critical for influencing epithelial barrier functions[34].

9. Inflamo-Immune Mechanisms

On reaching local and systemic lymphatic and / or blood circulation microbe or their molecular metabolic products induced excessive pro-inflammatory and inflammatory cytokine production "hyper-cytokemia, the cytokine storm" that is one influence, the other influence of microbial antigens may stimulate; humoral, cellular immune and autoimmune responses which may finalized by production of antibodies, autoantibodies, activated

immune cells and autoreactive cells. The final fate of effector cells either remain in the vicinity of immune reaction or trafficked and homed in other body sites for promotion of pathogenesis. Memory lymphocyte subsets are either remained at the site of immune induction or migrate to other distal lymphoid tissues to be of help on recall. Local gut Treg. phenotypes may migrate to affected body parts with autoimmune reactions to restore normal immune homeostasis through tolerance mechanism[36,37].

10-Autoimmune Mechanisms

The possible auto-immuno-gens in case gut dysbiosis-autoimmune reactions are; Brock of immune tolerance initiated by; clonal deletion, anergy and or Treg activity, molecular mimicry, epitope spreading, antigen bystander, B cell polyclonal activation aberrant cytokine signaling, infectious immune dysregulation and micro-RNA[38,39]. Both autoantibodies and auto-reactive B and/or T cells. The scoring parameters were including; the levels of autoantibodies, immune complexes formed and autoreactive B and T cells should be higher than that matched in physiological autoimmune reactions with an apparent pathognomic immune tissue reactions[40].

11- Association Versus Causation

The correlation between gut microbiome dysbiosis and regional or systemic autoimmune diseases seems to lack directionality. Though its being an association rather than causation state. Production of autoimmune disease in laboratory animal have shown at most causation state but on extrapolation to human disease it is difficult to elucidate direct relationship. However, at few cases there could be proved in human being. There is being still a debate and un-certainty in scientific conception of dysbiosis associated with autoimmunity or autoimmunity associated with dysbiosis.[30, 33,41]. Neagu et al.[42] have been reviewing the recent advances in autoimmune diseases. They came to conclusion naming it as "Autoimmunity Unified Theme" which was defined as; autoimmunity is a multidimensional process shaped by immune dysregulation, genetic predisposition, proteomic signature, microbial interactions and systemic consequences.

12- Sample Analytic Approach

The immune-biologic analytic features for a sample autoimmune diseases were depicted in Table -1. These features were encompassing; possible cause, dysbiotic characteristics, rise of pathobiont, barrier impairment, barrier passage patterns, molecular immune mechanisms, clinical therapeutics and preclinical therapeutics.

Table -1 :Analytic view to gut dysbiosis -Autoimmune Diseases.[40].

Features/Diseases	Multiple sclerosis	Rheumatoid arthritis	Systemic lupus erythematosus
Possible Etiology	Genetics-Environment	Genetics-Environment	Genetics-Environment
Gut Microbiome Dysbiosis	Decrease of ;F.prausitzii Prevotella, Bacteriodes Increase of Akkermansia	Enrichment with; Prevotella, lactobacillus sabotage, Segmented filamentous bacteria	Enrichment with ; Campylobacter, Streptococcus Vellonella Depletion of Bifidobacterium Lactobacillus
Molecular Mechanisms within dysbiotic micro-environment	Change in cross-talk with mucosal epithelium and with immune cells, change in quorum sensing signals between microbial entities, induction of cytokine production by host cells	Same	same
Intestinal mucosal barrier	Tight junction impairment	Tight junction impairment	Tight junction impairment
Pathobiont translocation	Unspecified	Prevotella corpi	E.gallinarum
Barrier passage	1-Through blood stream	1-Through intestinal IEB	1-Through intestinal

pattern	and M cells 2-systemic inflammatory responses	lymphatics 2-systemic inflammatory responses	intravascular barrier IVB 2-systemic inflammatory responses
Molecular immune –genetic mechanisms	A-Mucosal immune reactions ;defective allele encoding immune functions 2-modulation of TH17/Treg.,increase of anti –Akk,P/histicola increase Treg and decrease of pro- inflammatory bacteria ,autoreactive T cell in Peyres Patches**	A-Mucosal immune reactions; 1- defective allele encoding immune functions 2-increase of inflammatory cytokines,increase TH1/Th17,trafficking of lymphocyte to systemic circulation.P corpi induces mucosal inflammation via TLR stimulation toTH17 leading to pro-inflammatory cytokine production	Mucosal immune reactions 1-defective allele encoding immune functions 2-TH17/Treg imbalance leading to cell proliferation and production of inflammatory cytokines that derives sysytemic inflammation and with lack of complement
	B-Systemic immune reactions; 3-miRNA molecular mimicry	Systemic immune reactions; 3-Molecular mimicry,anti- double strand DNA antibody titre and complement activity***	Systemic immune reactions; 3-Break self tolerance to self RNA ,molecular mimicry ,B cell hyperactivity, IFNI activation associated with immunogenic pathology***
1-Clinical therapeutic	Anti-CD20 monoclonal antibody directly damage B cell, Probiotics	P. histicola act as probiotics ,through IL10 and T reg. pathway	Gut microbiome composition,metabolite regulate antibody production by B cell ,Th17/T reg.balance
2-Preclinical Therapeutics		RA patients showed*** reduced butyrate. Butyrate supplementation alleviate RA severity in murine model RA patient MAIT cells transfer to MHC1knocout mice worsen disease	Probiotics and prebiotics control ***inflammation, reduce antibody production, reduction of severity

*Mousa et al.2022 [40] ;**Somasundaram et al.2026[23] ***Zeng et al.2026[41]

13-Conclusions

Microbiome dysbiosis in experimental animals induces autoimmune diseases in gut and/or in distant remote organs. While , in human being gut microbiome dysbiosis mostly associated with an autoimmune responses. When the immune tissue microenvironment of dysbiosis favors the appearance of pathobiont through the impaired intestinal barrier ,autoimmune responses may be initiated. In other word the relation seemed to association rather than causation. Molecular immune mechanisms encompassing; rise of pathobiont, barrier impairment, pathobiont translocation through lymphatic ,blood circulation or M cells, brock of immune tolerance via clonal deletion, anergy suppression of T reg. function; lymphocyte subsets imbalance, and cytokine imbalance. Such transfer stimulate systemic inflamma-immune responses leading to production of

autoantibodies, autoreactive T cells through the interaction with memotope, epitope spreading , antigen bystander with or with out the presence of complement .Direct causation of dysbiosis to autoimmune diseases still a matter needs to be assured in human beings.

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