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SERVICES FOR SCIENCE AND EDUCATION

An at Glance Insight to Gut Microbiome Driven Autoimmunity

Ibrahim M S Shnawa
Department of Medical Biotechnology, College of Biotechnology,
ALQasim Green University, Qasim, Babylon, IRAQ and
College of Nursing, University of Hilla, Ranringia, Babylon/IRAQ

ABSTRACT

Autoimmunity is attributed to genetic predisposition [Genome] and environmental factors[exposome] and microbiome[microbiota]. The objective of the present opinion paper was to present an at glance insight to gut microbiome driven autoimmune diseases GMDADs based upon current published literature. The exposome components are; infection ,vaccination ,allergy and stress .Human balanced microbiome is noted in immune homeostasis. While, in its dysbiotic state in which specific microbe linked to induction of auto immune reactions, the infectome. On the onset of autoimmune diseases, leaky gut initiated, the antigens of the infectious agent translocated, molecular mimicry, autoantigen overproduction and cytokine overproduction, all of these were moved through blood to other extra-intestinal compartments into which brook of tolerance and dysregulate homeostasis. Then, changes happened in transcription and translation of the gene(s) encoding the autoimmune reaction pathway along with the coevolved changes immune reaction pathways. To this end autoimmune reactions are either goes on or blocked by the dysbiotic microbiome. In case of rise up of autoimmune reactions producing clinical concentration levels of either auto –antibodies or auto-reactive T or B lymphocytes indicating autoimmune diseases. Auto-immune T cells produced in the gut lymphoid tissues migrate from gut to an eye auva leading to auto-immune auvitis. Gut lymphoid tissue produced T reg., migrate to reach the autoimmune inflamed central nervous system leading to inhibition of auto immune reactions. A suggestion for an animal model for infection induced auto immune disease in lapin animal was made. Not all dysbiotic microbiomes driven autoimmune diseases showing clear immunopathological mechanisms. A call suggestion for study the molecular mechanisms of the presently unknown microbiome driven autoimmune diseases.

Keywords: Allergy, autoantibody, auto-reactive, dysbiosis, exposome, extra-intestinal, infection, infectome, gut.

INTRODUCTION

The collective description for the normal human microflora harbouring various body organ compartments is known as microbiota. It constitutes the compositional and functional attributes. While the genetic detected normal human microflora known as microbiome,though both of the terms were used interchangeably .Microbiome impacts human immune system evolution and function both in normal and dysbiotic states. The compositional spectrum of the microbiome is termed as microbiome signature MS. Dysbiotic MS were different in different autoimmune diseases. objective of the present opinion paper was to present an at glance insight on gut microbiome driven autoimmune diseases GMDADs based upon the current published

literature [1-7]. Such GMDADs insight tempts to; i – build up GMDADs timeline , ii – Suggest unified mechanism for GMDADs , iii – Propose a laboratory animal model for verifying GMDADs paradigm and iv – Putforward a comprehensive table form view to the attributes of this paradigm.

CONCEPTUATION

Normal individuals may showed low concentration of some auto antibodies below the baseline limits.Such a case is known as physiological autoimmunity .Other pepoles may have autoantibody concentrations below the clinical threshold for an autoimmune disease this case is denoted as subclinical autoimmunity .While those pepoles whome have autoantibody concentration within the clinical limits is known as pathologic autoimmunity,and the same for autoreactive cells.The state of the gut microbiome in normal healthy persons known as biosis in which the microbiome contained balanced composition and function for its formed microflora.While when the microbiome undergoes infection ,vaccination ,inflammation ,allergy and/or stress.The composition and function becomes distorted and termed as dysbiosis.Hence normal microbiome parallels with normal homeostasis and dysbiotic microbiome along with disease states.The compositional and functional spectrum of microbiome is designated as microbiome signature .The translocation of the specific antigen of the invading pathogen passing through gut leak may be involved in the induction of immune mediated disease such as autoimmunity via a number of mechanisms as; molecular mimicry,autoantigen over production,antigen bystander inducing gene(s) expression that may leave human body in acase of recognition of self as nonself antigen with the consiquences of auto immune diseases[1-7].

TIMELINE

Digging through past sparks light to the present and draw basic lines to the future. On passing from the emergence point of gut microbiome driven autoimmunity, one may found that apparently it emerges in 2010 and continue up to date 2024 [1-28], Table – 1.

Table 1: Gut Microbiome driven Autoimmunity Timeline.

Acheivment	Reference
Germ free animal with filamentous bacteria develop experimental autoimmune encephalitits	Lee et al 2010 [9].
Ulteration in gut microbiota cause immune dysregulation leading to autoimmunity	Wu and Wu 2012[10].
The terms exposome, infectome and autoinfectome were coiend	Bogdanos etal. 2013 [11].
Gene expression of the genes encoding autoimmune reactions cause signaling pathway finalized by autoimmunity Book Publication on Gens and autoimmunity	Stanilova 2013 [12].
Inheritance of familial microbiome produce familial autoimmune disease	Poral et al. 2013 [13].
Development of autoimmunity requires genetic predisposition and environmental factors that triggers immune pathway	Wang et al.2015[14]
Sex hormones influences autoimmunity via dysbiosis of gut microbiome composition	Gomez et al., [15].
Gut microbiome holds the position of major player in intestinal and extra-intestinal autoimmunity Book puiblicationon Infection and Autoimmunity	Bogdanose et al.2015 [16].

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Gut microbiota provide a source of cross-reactive antigens that activates gut autoreactive lymphocytes. Gut microbiota regulate autoimmunity	Zarate-Blades et al.2016[17]
Clonal B cell expansion and affinity maturation induce formation of high affinty antibody response that acts as central driver of autoimmune disease	Degn et al.2017[18].
Gut regulatory T cell modulate gut microbiome leading to reduction in autoimmune nervous inflammation	Colpritts and Kasper 2017[19].
There was dynamic interplay between gut microbiota and autoimmune disease where Aryl hydrocarbon receptor, a ligand activated transcription factor is a master moderator of host-microbiota interaction	Xu et al.2019[20]
Gut normal microbiota take part in both immune homeostasis and autoimmune reactions in its dysbiotic state	Luca and Shoenfeld 2019[21].
Host immunity and the immune system are regulated by microbiota through barrier maintanance and immune exclusion	Brown et al.2019[22].
Microbiome dysbiosis take part in evolution of arthritis	Rath 2021[23].
Gut microbiome dysbiosis impacts gut barrier integrity	Mangalam et al.2021[24].
Bifidobacterium genus has causal relatioships with Diabetes militis type I and coeliac disease	Xu et al 2022[25].
There is a possible cause-effect relationship between microbiome and autoimmune disease	Christovich and Luo 2022[26].
There were different dysbiotic microbiome signature in different autoimmune diseases	Shaheen et al., [27]
There found to be changes in compositional and functional aspects in different autoimmune diseases	Rodrigo 2022[28].
There found a triportite axis between intestinal regulation, multisystem autoimmune diseases and gut microbiome	Zhang et al.2023[7].
Coining the term preclinical autoimmunity	Pisetsky 2023[6].
Dysregulation of oral microbiome triggers and promotes autoimmune diseases through microbial translocation, molecular mimicry, autoantigen overproduction and amplification of cytokine production	Huang et al.2023[5].
Bacteroides ovatus IgA coating index constitute individual autoimmune disease risk predicator in a mouse model	Steimle et al.2024[4].
As an environmental factor, dysbiosis of gut microbiome can modulate immune responses and contribute to the development of autoimmune disease	Haravi 2024 [2].
A mucin siplitting bacterium Akkermansia muciniphila involved in IgA nephropathy in a mouse model	Eauropean Biotechnology 2024 [1].

HOMEOSTASIS

Homeostasis,in physiological sense means the human body tempts to mantaine stable internal environment though acheiving some sort of balance.Homeostsis is based upon three basic mechanisms to monitor and mantaine adynmamic state of equilibirum within narrow range.These mechanisms are integrated to make homeostatic balance as; i-receptors , ii- control centers and iii - effectors.The receptors can sense both external and internal environmental changes and provide information on the changes to the contral centers.The control centers determine what a particular value of blood pressure for instance should be and sends message

to the effectors.Effectors once they have recieved informaton from the control centers ,the effector cause a response to take place within the bodys internal environment. Such response produces the changes that will return the internal environment to its normal values [29]. Immune homeostasis is the optimal immune response that eliminates the invading pathogen and restoring the immune equilibrium without affecting harm to the host. Infection, vaccination and /or allergies are mostly triggering immune system a causing dysregulation to the immune homeostasis. Cytokine balance between pro- and anti-inflammatory cytokines derived from both of innate and adaptive immune responses, thus reaching the balance. Such balance can also be attained in cells originated from lymphoid organs [8].

EXPOSOME

The exposome holds the position of both exogenous and endogenous factors which we are exposed to in a life time. It form the umbrella for infectome and auto-infectome [11,17,30]. Exposome represent an important trend in the study of autoimmunity linking classical immunological tools and recent genome wide associations .Exposomes are mainly subdivided into infectious and no-infectious aetiology .Infectome which is a part of exposome referring to the collection of individual exposures to the infectious agent in a life time. Infectome is a plateform to trace infectious trigger of autoimmunity [11]. The oral cavity,gut and urinary tract microbiomes are informative but can only be useful if further explored from the infectome issue .In other word the apparent association between pathogenic bacteria with the autoimmune disease,it should be tested experimentally and the underlying mechanisms shoud be identified[30]. There were an instances where a specific bacterial pathogen within the composition of the microbiome that are in association with autoimmunity.Dysbiotic urinary microbiome enriched with Proteus mirabilis have longly been linked with rheumatoid arithritis[32] .Bacteroides ovatus was a reliable disease risk predicator before disease onset,due to its ability to reflect autoimmune mediating properties of a given gut microbial network within a specific host such as the setting that predicate autoimmunie neuroinflammation[4].In mice and cell culture Akkermansia muciniphila modified IgA1 and converted it into a degylcosylated form associate with autoimmune IgA nephropathy. This process created new antigens that crossed the intestines, inter the blood stream then eventually setting in the kidney. Such mice model expressing human IgA ends up developing IgA nephropathy after being colonised the mucine splitting bacteria Akkermansia mucininphila [1]. Auto-infectome is concerned with microbiome variability that could contribute to polymorphism of autoimmunity across the genaral public[33].

MECHANISMS

Microbiome may affect a control of the immune system functions as innate,cross-road and adaptive immune responses.Dysbiotic microbiome can initiate, adavance and /or block autoimmunity[31].In human autoimmune diseases marked failure obsereved in metabolic processes resulting from common underlying pathogens as the successive accumilation of such pathogens within the continuum of the microbiome overtime and the ability of such pathogens to dysregulate;gene transcription and traslation which may influence failure of metabolic processes leading to autoimmune reactions[13].Microbiota may provide a source of cross-reacting antigens that activate auto-reactive lymphocytes within the gut micro-environment in continuum with a spontaneous model of auto-immune auvitis,in which specific retinal autontigen T lymphocyte are activated through the specific antigen receptor in the gut aquire the ability to activate inflammatory autoimmunity of the eye[34].In central nervous system

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autoimmune disorders,autoreactive T cells dystroy nervous tissue resulting in severe disabilities.There were a gainwine evidence suggests that modulation of the gut microbiome will affect reduction in nervous autoimmune inflammation[19].The exposure of human being to exposome,such exposure lead to genetic defects mediating broke of tolerance and and change in homeostatic mechanisms[22].Aryl hydrocarbon receptor,a ligand activated transcription factor holds the master modertor position for the host- microbiome interactions because of its ability to shape the immune system[25].Gut microbiome dysbiosis can compromize gut barrier integrity ,resulting in translocation of bacterial contents across the epithelial barrier. Bacterial contents like lipopolysaccharide, other antigens can induce inflammation environment through the activation and induction of the immune cells [24]. The passage of leaking microbial antigens into the internal environment may break up the self tolerance generating either autoantibodies or autoreactive lymphocytes [28]. Both human and mice autoimmunity can be attribute to leaky gut and microbiome dysbiosis and stand as frequent phenomena[26].The microbiome signature in healthy human is different in different persons. Likewise, the dysbiotic microbiome signatures were different in different auto-immune diseases. Thus gut microbiome are contex dependent and can not be generalized in the same exact way in other autoimmune diseases[27].There were a causal relationship between Bifidobacterium genus and T1D and coeliac diseases [25].Colonization of mice kidney with the mucin siplitting bacteria leads to IgA nephropathy[1].The mechanism of microbiome driven autoimmune diseases as, microbiome dysbiosis, microbial translocation, molecular mimicry,autoantigen overproduction, and cytokine overproduction[5].It seed worthy to unify mechanisms in a sum up as in the followings;

1. Human individual genome may bear genetic predisposing autoimmune encoding allels
2. Exposure to an exposome to rather long time [infection, vaccination, allergy stress]
3. Leaky gut
4. Translocation of the specific enriched bacterium microbiome[dysbiotic]
5. Molecular mimcry, autoantigen overprodution, cytokine overproduction
6. Broke of self tolerance and homeostatic dysregulation
7. Change in the transcription and translation and gene expression of the pathogenic auto immune encoding allele along with the change of immune reaction pathways
8. Dysbiotic microbiome either potentiate or block the resulting autoimmune reactions
9. Microbiome signature is different in different autoimmune diseases and different in different normal human subjects.
10. Autoreactive T lymphocyte from gut lymphoid tissue migrate from gut to an eye uva causing autoimmune uvitis
11. T reg. evolved in intestinal lymphoid tissue migrate to autoimmune central nervous system causing reduction in the autoimmune inflammation.

INVESTIGATIVE APPROACHES

The laboratory immunology of microbiome driven autoimmune diseases, Table -2 can be categorized into organ specific and multiorgan autoimmune diseases.

Table 2: Microbiome driven autoimmune diseases

Organ Specific	Multiorgan
Diabetes militis I	Rheumatoid arthritis
Thyroditis	SLE
Coeliac disease	Spondylo- arthritis

	Multiple Sclerosis
	Neuroinflammation

The essential criteria for the laboratory immune diagnosis of the microbiome driven autoimmune diseases are depicted in Table - 3.

Table 3: Microbiome driven Autoimmune disease diagnostic criteria.

Steps	Criteria
1	Clinical limits for the autoantibody
2	Clinical limits of autoreactive lymphocyte
3	Identification of microbiome signature
4	Identification of the possible infectome
5	Identification of the possible chemical mediator operable in the mechanism leading to autoimmune disease

ANIMAL MODEL SUGGESSION

The animal model will be the rabbit and mice. The infectious agent be Campylobacter fetus. Specific immune serum for Campylobacter fetus will be prepared in rabbit. Mice anti-brain tissue homogenate antigen will also be prepared. A group of rabbits will be dosaged orally with sub LD50 live C.fetus for seven consecutive weeks, then followed by three weeks leave. Immunized rabbits with C.fetus will be eviscerated and intestinal tissue blocks for histology be done. Mucosal scarp saved and homogenates made for intestinal antigens and for mucosal antibody separation C.fetus antigen tested with mouse antirabbit brain. Brain homogenate antigen tested with ati-C.fetus immune serum. To this end bidirectional molecular mimicry tested. Whether it is of reciprocal or nonreciprocal nature. Microbiome signature test for normal control and for the C.fetus orally immunized rabbits[34,35].

THE PARADIGM

The title phrase ensembles an at glance insight. To elucidate the issue, one may put-forward a comprehensive view to the paradigm of microbiome driven autoimmune diseases [1-39] as that presented in, Table- 4.

Table 4: Comprehensive View on Microbiome Driven Autoimmune Diseases MDADs in Human Beings.

Exposome	**Genome**	**Microbiome**	**Host – microbiome interaction [36-39]**	**Host physiological state**	**Host immunological state**	**Outcome**
Nile	Host genome	Balanced Host microbiome	Organ-organ axis cell-cell crosstalk	homeostatic	homeostatic	Healthy normal subject
Infectome	Microbiome genome-host genome interaction	Dysbiotic, compositional and functional changes	Organ-organ axis Cell-cell cross talk Microbe-host cell cross talk Gut leak Autoantigen overproduction, molecular	dysregulation	Dysregulation as broke of tolerance Recognition of self as non-self antigens	Autoimmune disease goes on or blocked

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			mimicry, cytokine overproduction, Changes in expression of autoimmune encoding genes			

GMDADs VERSUS ADs

Abbas et al., [40], rise up two fundamental questions related to the issue of ADs mechanisms. First question was how the self tolerance fails and the second question was how self reactive lymphocytes are activated. To answer the questions need to understand aetiology and pathogenesis of Ads. For the seek of elucidation for the link between MDADs and Ads in the mechanistic sense, a comparative table form view was depicted in Table-5

Table 5: MDADs and Ads comparative mechanismic view

MDADs [1-39]	**Ads [40,41]**
Genomics reveals Gene polymorphism of the autoimmune encoding all(s)	Genomics reveals gene polymorphism of the autoimmune encoding allel(s)
Exposome: infectome	Exposome: toxins, food, stress
Immunonome reveals; 1. Failure of tolerance 2. abnormal display of self antigens 3. inflammation	Immunome reveals; 1. Faiure of tolerance 2. abnormal display of self antigens 3. inflammation
Possible mechanisms i. Microbiome dysbiosis ii. gut leak iii. microbial antigen translocation iv. Autoantigen overproduction* v. Antigen bystander* vi. Molecular mimicry* vii. cytokine imbalance* viii. broke of tolerance ix. dysregulation of immune homeostasis x. gene expression of autoimmunity encoding allele(s) xi. change in immune raction pathways xii. goes on or blocked	Possible mechanisms* Release of sequestrated antigens Modification of antigens Cross reacting B cell epitope Polyclonal activation molecular mimicry Cytokine imbalance Stochastic events

*Either one or more of these mechanisms.

CONCLUSIONS

Compositional and functional Balanced microbiomes are flowing with immune homeostasis. While dysbiotic microbiome are found in association with the immunopathogenesis of immune mediated diseases like autoimmune diseases. The consequences of the reaction between the dysbiotic microbiome and with cellular events of the immune responses is either going on to initiate autoimmunity or to subside the reaction pathway. A timeline was built up. A unified mechanism of microbiome driven autoimmune diseases was suggested. Besides a suggestion of laboratory animal model that mimic microbiome driven autoimmune diseases.

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