OPINION

Immunity and metabolism interaction dictates on health and illness

Kristofina Eliot

Eliot K. Immunity and metabolism interaction dictates on health and illness. J Genet Disord Genet Med. 2022; 6(3)33-35.

ABSTRACT

The immune system has lately been linked to managing systemic metabolic homeostasis, in addition to its conventional role in anti-infectious and anti-neoplastic defence. Similarly, various metabolic mechanisms were involved in immunological homeostasis maintenance. Given the importance of immune-metabolic alignment in promoting "metabolic health" and its fundamental role in endorsing appropriate adaptations to a host's ever-changing environmental setup, we highlight current understanding of this immune-metabolic cross-talk and illustrate the role of the gut microbiota, diet, and host genetic and epigenetic factors in this immune-endocrine communication, paving the way for future research and promising therapeutics.

Key Words: *Immunity; Metabolism; Gut microbiota; Diet; Genetics; epigenetics; Heath; Disease*

INTRODUCTION

The intertwined relationship between immunity and metabolism is thought to be very old, dating back to a few billion years ago, when general practitioners thought infection was a source of metabolic disorders. Several investigations have since confirmed this immune-metabolic alliance. Acute inflammatory disorders like meningitis, for example, were strongly linked to diabetes in humans in the 1800s. Additional studies in the 1900s linked obesity to hyper insulitis-insulin resistant-diabetic patients, highlighted the role of gram-negative bacterial Lipopolysaccharides (LPS) in inhibiting insulin's ability to induce glucose uptake in muscles, promoting insulin resistance in dogs, and demonstrated that acute infections in humans are accompanied by a decrease in insulin's ability to bind to its receptor on blood cells. These findings highlighted the presence of metabolic problems in the context of infection and opened up new avenues for research linking obesity to insulin resistance and diabetes [1].

It was discovered that various metabolic processes are involved in fostering immunological homeostasis, which is consistent with the immune system's involvement in maintaining metabolic balance. The fact that the immune system cannot function in malnutrition and that activation of many immune pathways necessitates intensive metabolic reprogramming of immune cells to meet their sufficient energy demands supports the existence of such a close relationship between the host's nutritional status and its immune system. For example, the transcription factor FOXO, a critical regulator of metabolism, promotes the generation of Antimicrobial Peptides (AMPs) independently of any immune regulatory pathway. In human peritoneal mesothelial cells, the relative transcript expression of certain Toll-like Receptors (TLRs) has been identified in a high glucose environment and linked to fibrosis and inflammatory diseases. In this regard, a few studies have suggested that TLR function is linked to metabolism. Some macronutrient metabolites, such as saturated fatty acid, directly influence the activation of some TLRs and the production of target gene products. In mice, one kind of endogenous lipid, branched palmitic acid esters of hydroxyl stearic acids, modulates intestinal innate and adaptive immunological responses. Indeed, the importance of commensal flora in this process is shown by the involvement of certain metabolites in orchestrating this immune-metabolic alignment. For example, an overabundance of commensal bacteria in the gut microbiota affects host nutrition, causing the host's intestinal immune system to activate and

Editorial Office, Journal of Genetic Disorder and Genetic Medicine, London, United Kingdom

Correspondence: Kristofina Eliot, Editorial Office, Journal of Genetic Disorder and Genetic Medicine, London, United Kingdom, E-mail geneticmedres@esciencejournal.org

Received: 2-Mar-2022, Manuscript No. puljgdgm-4525; Editor assigned: 4-Mar-2022, PreQC No. puljgdgm-4525(PQ); Reviewed: 18-Mar-2022, QC No. puljgdgm--4525(Q); Revised: 20-Mar-2022, Manuscript No. puljgdgm-4525(R); Published: 27-Mar-2022, DOI:10.37532/Puljgdgm.2022.6(3).33-35.

diminish the population of commensal bacteria. This anticipated alignment does not appear to be directed solely against gut flora, but could also be used by the host to combat invading pathogens, given that ingested nutrients frequently contain large numbers of bacteria, necessitating the presence of such a protective gastrointestinal-immune defensive strategy. Interestingly, multiple studies have found that the existence of such an interaction between the gut microbiota and various immunological receptors, including TLRs, is required for immune system homeostasis to be maintained [2,3].

For many years, scientists have been baffled by the close contact between these two seemingly distinct systems. The idea of competition over energy resources, as well as the existing trade-off between metabolically conserving energy on the one hand and draining energy by immune defensive mechanisms on the other, is one consideration overseeing this immune metabolic arrangement. Taken together, all of these observations and lines of evidence inspired ongoing studies to understand the immunological nature of a metabolic disease and the metabolic foundation of an immune poise. In normal and disturbed conditions, our knowledge of the molecular and cellular mechanisms involved in coordinating immune microbe-mediated metabolic equilibria and metabolic immune-medicated microbial equilibria is still in its infancy. We present an overview of the concepts of immune-metabolic communication in health and illness, as well as the role of the gut microbiota, food, and host genetics and epigenetics. In normal and disturbed conditions, our knowledge of the molecular and cellular components involved in coordinating immune microbe-mediated metabolic equilibria and metabolic immune-medicated microbial equilibria is still in its infancy. The role of the gut microbiota, food, and host genetics and epigenetics in health and illness is depicted in this article, which provides an outline of the concepts of immune-metabolic communication [4].

Gut Microbiome in Immune-Metabolic Interactions: Bacteria generally found in the gastrointestinal system make up the gut microbial population. This bacterial "factory" is in charge of a wide variety of processes. Metabolic and physiological processes that it necessitates host. The significance of the gut has been discovered in several investigations. The role of microbes in nutrition absorption and weight loss via nutrition acquisition, energy expenditure, and obesity harvesting, and the control of a variety of hosts routes of metabolism Nutritional burdens that have changed For example, either a low or high calorie intake causes dramatic changes in the gut's bacterial makeup as a result of the microbiota, resulting in relative variances in the Lean people have more energy in their stools. The gut flora's key metabolic roles include vitamin production, carcinogen and dietary toxin catabolism, mineral and electrolyte absorption aid, and fermentation of complex-indigestible food elements. The gut microbiome's involvement in sustaining a healthy gastrointestinal system, on the other hand, is assumed to be immune-related. The inflammatory response seen in high-fat diet-induced metabolic syndrome is thought to be triggered by the LPS of gram-negative bacteria in the gut flora. Recent research has looked at the function of the gut microbiome in regulating TLR-mediated insulin signaling. In one of these investigations, mice lacking the microbial pattern recognition receptor TLR5 demonstrate hyperplasia and obesity, as well as other metabolic syndrome symptoms such as hypercholesterolemia, hypertension, dysregulated interleukin-1 signaling, and insulin resistance. TLR2-deficient animals have a different gut microbiota, with more Firmicutes and fewer Actinobacteria of the family Bifidobacterium, and develop glucose intolerance, insulin resistance, and obesity, according to another research. Intestinal bacterial compounds that function as TLR 4 and 9 agonists have also been shown to produce severe hepatic steatosis, inflammation, and obesity. Surprisingly, new research has revealed that the gut microbiota plays a significant role in the regulation of type 1 diabetes. Whole genome metagenomics and metabolomics in metabolic diseases, accompanied by bioinformatics resources and large databases similar to those derived from the Human Microbiome Project, to pave the way for a better understanding of the gut microbiota's role in a wide range of metabolic disorders such as diabetes, metabolic syndrome, and obesity [5,6].

Diet in Immune-Metabolic Interactions: Nutrition influences immune system function either directly by interacting with immune cell receptors or indirectly via modifying gut bacteria metabolites. Vitamin A, Vitamin D, and Indole 3-carbinol, for example, stimulate local hematopoietic cells while still maintaining the integrity of the gut mucosal barrier. Saturated and polyunsaturated fatty acids also interact with adipose tissue immune cells, regulating the immune system and exerting metabolic consequences. Nutrition affects the mucosal architect of the gut, its digestive function, immune tolerance, and various metabolic pathways, primarily those associated with conjugated bile acids and short chain fatty acids, as well as the mucosal architect of the gut, its digestive function, immune tolerance, and various metabolic pathways, including those associated with conjugated bile acids and short chain fatty acids. . It is assumed that the pathogenesis and accelerated ageing of illnesses linked with low-grade systemic infections begin in the gut and move outward, causing pathogenesis and accelerated ageing in other organs such as the liver, brain, and adipose tissues [7].

Host Genetics and Epigenetics in Immune Metabolic Interactions: Aside from the gut microbiota and food, another intrinsic element that contributes to the induction and maintenance of an immune-metabolic balance is the host genome. Several genetic players implicated in metabolic illnesses have been found using large-scale genomic techniques, some of which are immune-related genes. CD44, an immune cell receptor, has been linked to type 2 diabetes mellitus, for example. Other studies have found a relationship between Single Nucleotide Polymorphisms (SNPs) and metabolic readouts, with the IL6 variation rs7801406 being linked to reduced fasting insulin levels. And the TNFA variation rs3039662, which is associated with high fasting insulin levels. Furthermore, SNPs in the TNFA and CRP genes were strongly linked to changes in blood HDL-C levels. These findings not only show how crucial genetics is in networking immunity and metabolism, but they also provide an intriguing environment for identifying new genetic biomarkers for metabolic illness early detection. Epigenetics, in addition to genetics, is an essential mechanistic connection between environmental signals and host gene expression, and hence plays a significant role in regulating the cross-talk between immunity and metabolism. Recent research on epigenetic alterations in obese people's peripheral blood leukocytes discovered changes in the methylation of two genes involved in macrophage and T cell modulation .Similarly, methylation in the TLR2 and TLR4 genes has been linked to the microbiome and obesity . Methylation markers in genes associated to inflammatory pathways, such as TNFRSF4, MAP3K2, and IL5RA, were discovered in a recent epigenome-wide-association research in obese adults. Another

study found that high blood sugar can change the histone methylation landscape, resulting in epigenetic activation of inflammatory genes such NF-B-p65. Increased global methylation levels have also been seen in the natural killer cells of type 2 diabetes patients and in the B cells of obese and type 2 diabetic patients on a larger scale.

This increased methylation in the epigenetic plot of particular immune cells coincides with insulin resistance, and functional changes in immune cells are strongly linked to metabolic diseases. Obesity has been shown in recent research to shift adipose tissue macrophages from an anti-inflammatory M2 stage to a pro-inflammatory M1-like stage, where DNA methyltransferase 3a and 3b perform de novo methylation. Surprisingly, saturated fatty acids, a common trait of obesity, were shown to activate DNA methyltransferase 3b, leading in M1 polarization, a hallmark of obesity and inflammation. Surprisingly, the gut microbiota has been found to trigger epigenetic changes, influencing both immunity and metabolism, mostly through short chain fatty acids. Short-chain fatty acids suppress inflammation by inhibiting histone deacetylase and thereby regulating the expression of immune-related genes [8,9].

The significance of microbe-derived chemicals, such as short chain fatty acids, stems from their capacity to easily cross the placenta and play a role in epigenetic immunological reprogramming and long-term metabolic dysregulation in the offspring of sensitive women. In diabetic and obese people, additional epigenetic processes linked to certain microbiome compositions, such as methylation in the promoter region of genes involved in immunity and metabolism, have been discovered [10].

CONCLUSION

Understanding the function of the gut microbiota, nutrition, and host genetic and epigenetic variables in maintaining a mechanistic concordance between immunology and metabolism is paving the way for promising treatment approaches to a variety of metabolic and inflammatory illnesses.

REFERENCES

1. Hotamisligil GS. Foundations of immuno metabolism and implications for metabolic health and disease. Immunity. 2017; 47(3):406-420.

2. Fox MJ, Kuzma JF, Washam T. Transitory diabetic syndrome associated with meningococcic meningitis. Arch Intern Med.1947;79(6):614-621.

3. Rabinowitz D, Zierler KL. Metabolism in obesity and its response to intra-arterial insulin. Characterization of insulin resistance and evidence of adaptive hyperinsulinism. J Clin Invest.1962; 41:2173-2181.

4. Raymond RM, Harkema JM, Emerson. In *vivo* skeletal muscle insulin resistance during *E coli* endotoxin shock in the dog. Circ Shock. 1981;8(4): 425-433.

5. Drobny EC, Abramson EC, Baumann G. Insulin receptors in acute infection a study of factors conferring insulin resistance. J Clin Endocrinol Metab. 1984;58(4):710-716.

6. Scrimshaw NS, San Giovanni JP. Synergism of nutrition, infection, and immunity an overview. Ame J Cli Nut. 1997;66(2): 464S4-477S.

7. Becker T, Loch G, Beyer M, et al. FOXO-dependent regulation of innate immune homeostasis. Nature. 2010;463:369-373.

8. Choi SY, Ryu HM, Choi JY, et al. The role of Toll-like receptor 4 in high-glucoseinduced inflammatory and fibrosis markers in human mesothelial cells. Int Urol Nephrol. 2016;49(1):171-181.

9. Zu L, He J, Jiang H, et al. Bacterial endotoxin stimulates adipose lipolysis *via* toll-like receptor 4 and extracellular signal-regulated kinase pathway. J Biol Chem. 2009;284(9):5915-5926.

10. Lee JY, Sohn KH, Rhee SH, et al. Saturated fatty acids, but not unsaturated fatty acids, induce the expression of cyclooxygenase-2 mediated through Toll-like receptor. J Biol Chem. 2001;276(20):16683-16689.