

Metabolism of Carbohydrates and Lipids' Role in MS Development

Mikael Cohen*

Department of Neurology, Schulthess Clinic, Zurich, Switzerland.

*Corresponding Author: Mikael Cohen, Department of Neurology, Schulthess Clinic, Zurich, Switzerland, E-mail: cohenm@gmail.com

Received date: August 29, 2022, Manuscript No. IPMCRS-22-14860; **Editor assigned date:** August 31, 2022, PreQC No. IPMCRS-22-14860 (PQ);

Reviewed date: September 12, 2022, QC No. IPMCRS-22-14860; **Revised date:** September 22, 2022, Manuscript No. IPMCRS-22-14860 (R);

Published date: September 29, 2022, DOI: 10.36648/2471-8041.8.9.242

Citation: Cohen M (2022) Metabolism of Carbohydrates and Lipids' Role in MS Development. Med Case Rep Vol.8 No.9:242.

Description

In Multiple Sclerosis (MS), a complicated autoimmune condition, inflammatory and demyelinating events affect the central nervous system. MS's precise causes and pathogenesis are still unknown. On the other hand, a number of metabolic changes, as well as how they affect immune cells and how the brain works, have been explained. This review highlights the role that metabolism of carbohydrates and lipids plays in the etiology and pathogenesis of MS. After that, we hypothesized that MS patients' immune systems and these metabolic changes were connected. The potential clinical effects of these metabolic changes on diagnosis, prognosis, and therapeutic target discovery have also been discussed, but this is not the last point. It is concluded that one approach for advancing MS treatment might be to investigate the pathophysiological changes in lipid and carbohydrate metabolism. In Traditional Chinese Medicine (TCM), diagnosis of pathogenesis, also known as Bing Ji, is one of the most important tasks. It serves the same purpose as the diagnosis of diseases in Western medicine. In keeping with the nature of Systems Science (SS), TCM theory defines pathogenesis as a multifaceted system of interconnected factors. In this paper, we present a heuristic definition we refer to as Pathogenesis Network (PN) to represent pathogenesis as a directed graph. A computational pathogenesis diagnosis technique known as Network Differentiation (ND) is therefore proposed by incorporating the holism principle into SS. In ND, there are three stages. Utilizing a Cartesian product that generates all possible diagnoses based on specific prior knowledge and input symptoms is the first step. In the second stage, the validated diagnoses are screened using the holism principle.

Steroid-Induced Osteonecrosis

Utilizing physician-computer interaction to select the clinical diagnosis is the third step. 100 clinical cases were the focus of our simulation experiments, and some theorems for further optimizing ND are stated and demonstrated in this paper. The experiments' results show that the proposed method is a good fit for bringing holistic thinking into the physician inference process. Steroid-induced osteonecrosis of the femoral head is frequently observed in clinical practice, but the cause is still poorly understood. Why some cases do not progress while others do not is puzzling. We conducted a comprehensive and

up-to-date literature review to ascertain its prevalence, associated risk factors, and pathogenesis. A higher dose and prolonged use of steroids, in addition to the underlying disease for which steroids were prescribed, were identified as risk factors. We discovered that the incidence was higher when associated with risk factors ranging from 3% to 40%. The pathogenesis of this disease is complex and poorly understood. If they are aware of this condition and the risk factors that are associated with it, doctors should be able to identify patients who are more likely to develop osteonecrosis while they are taking steroids. Due to significant immunosuppressive viruses like the Chicken Infectious Anemia Virus (CIAV) and subgroup J Avian Leukosis Virus (ALV-J), the global poultry industry has lost a lot of money.

In recent times, domestic chicken flocks in China frequently co-infected with CIAV and ALV-J. However, the combined pathogenesis of CIAV and ALV-J has not been thoroughly investigated. In order to gain a deeper understanding of the potential synergistic pathogenesis of CIAV and ALV-J, a co-infection study was conducted. An in vitro study revealed that ALV-J could not increase CIAV replication, but CIAV could promote ALV-J replication in HD11 cells. According to a chicken infection study, the infected chickens lost a lot of weight due to the synergistic effects of CIAV and ALV-J. In the co-infection group, CIAV significantly increased ALV-J viremia, viral shedding, and tissue load, whereas ALV-J had no effect on CIAV viral shedding or tissue load. All of these data demonstrate that the co-infection of CIAV and ALV-J has a synergistic pathogenesis and that CIAV has a positive effect on the pathogenesis of ALV-J. Alzheimer's disease is the most common form of dementia in humans and is a complex neurodegenerative disease that affects the elderly. CIAV and ALV-J may also significantly inhibit the humoral immunity to the H9N2 influenza virus and the serotype 4 fowl Neurofibrillary tangles, composed of hyperphosphorylated tau protein, and amyloid plaques, which are composed of beta-amyloid peptides, are two examples of abnormal protein aggregates that are characteristic of AD. Synaptic plasticity, neuroinflammation, calcium signaling, etc. also demonstrate the dysfunction of AD patients. Autophagy is a lysosome-dependent cellular process that has existed for a long time in eukaryotes. Through modulation of protein metabolism, damaged organelles and misfolded proteins are degraded and recycled to keep protein homeostasis intact. There is increasing evidence that impaired autophagy contributes to AD pathogenesis.

Chemokines in the Process of Inflammation

In this review, we focus on how autophagy, both bulk and selective autophagy, regulates metabolic circuits during AD pathogenesis. Chemokines Involved in the Inflammatory Process Strategies for treating AD that stimulate autophagy are also a topic of discussion. Giant cell arteritis is a primary granulomatous vasculitis that only affects people over the age of 50. It is characterized by a strict tissue tropism for vessels of a large and medium size. Our comprehension of some of the pathophysiological mechanisms involved in the pathogenesis of giant cell arteritis has advanced significantly over the past ten years. However, specific disease triggers and chronic damage mechanisms have not yet been identified. The definition of a particular pro-inflammatory hierarchy among the various cell types and inflammatory-related cytokines or chemokines remains an unexplored field of inquiry. The ultimate goal of precision medicine is to find the best treatment option for a particular person or group of people with a disease. The fundamental prerequisite for this strategy is the discovery of

molecular biomarkers that enable an accurate patient stratification and adequate baseline prediction of the therapeutic response. As a result, taking temporal artery biopsies for diagnosis opens up a whole new world of possibilities for defining various disease pathotypes that may respond to various therapeutic interventions. In this Series paper, we will try to define a new pathogenetic-centered approach to patients with giant cell arteritis by describing the most recent evidence regarding the disease's pathogenesis. Inflammation's role in the pathogenesis of depression is becoming increasingly apparent, despite the fact that its role in perinatal depression is less well-studied. Because pregnancy and the postpartum period are marked by distinct and shifting inflammatory profiles, the study of depression-related changes is complicated. This review demonstrates that both antenatal and postnatal depression are influenced by the immune system. Before the discussion concludes with suggestions for future research in this rapidly expanding field, the literature on the impact of the mother's immune system on the composition of her breast milk and the immunological and behavioral outcomes of her offspring is also discussed.