



IJRASET

International Journal For Research in
Applied Science and Engineering Technology



INTERNATIONAL JOURNAL FOR RESEARCH

IN APPLIED SCIENCE & ENGINEERING TECHNOLOGY

Volume: 8 Issue: VI Month of publication: June 2020

DOI: <http://doi.org/10.22214/ijraset.2020.6219>

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Influence of Antibiotics on Gut Microbiota and Stroke - A Mini Review

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Abstract: Commensal gut bacteria can alter the immune system of human beings and can cause disease progression in the nervous system including brain. Nevertheless, it remains ambiguous whether the gut microbiota has an impact on stroke.

Keywords: Metagenome, stroke, 16 S rRNA, DNA, gut

I. INTRODUCTION

Antibiotic-induced changes in the gut microbial flora reduce ischemic brain damage in animal model such as mice [1, 2, 3 and 4]. Severe brain lesions induced dysbacteriosis of the gut microbiota affected neuroinflammatory response after brain injury [5]. However, there are various unanswered questions in the field of gut microbiome and its effect on stroke [6]. One such query is the event that happens after stroke in which the brain maintains a shield and prevents the growth of fermentative bacteria [7]. A recent report also brought the fact in spot light that change in microbiota in C57BL/6 mice after post-stroke condition depends upon the type of breeder [8]. Moreover, stroke also promotes the movement of bacteria from the host gut microbiota to lung [9]. More recently, age-related changes in the gut microbiota and its dependent trimethylamine *N*-oxide has shown the relationship between systemic pro inflammatory cytokines/monocytes and stroke outcome [10, 11, 12, 13 and 14]. In short, stroke alters the gut microbiota which in turn modulates stroke outcome and microbiome plays a significant function in the pathogenesis of stroke [15, 16, 17, 18].

II. POTENTIAL QUESTIONS

Significant questions that arise as a result of relationship between gut microbiota and stroke include: How brain maintains a guard and prevents the growth of bacteria that promotes fermentation? Does the type of breed (in case of laboratory animal) / race (in case of human beings) affects the microbiota which in turn decides the progression of stroke? Does the stroke affect the spread of host microbiota beyond gut? How do the age-related changes in the gut microbiota affect the inflammation and stroke

III. MICROBIOTA STUDY OF THE TYPE OF BREED (IN CASE OF LABORATORY ANIMAL) / RACE (IN CASE OF HUMAN BEINGS).

Metagenomic DNA (Mt-DNA) could be isolated and purified from the gut of the type of breed (in case of laboratory animal) / race (in case of human beings) having stroke. PCR (Mi-seq) and Mt-DNA sequencing could be done. Exclusion of noise data, chimera profile generation, generation of SFF-file and Q-file, Mt-DNA sequence clustering, taxonomic recognition and data assessment could be done.

IV. METAGENOMICS OF ORGANS IN THE VICINITY OF GUT.

Metagenomic DNA (Mt-DNA) could be isolated and purified from the organs in the vicinity of gut of laboratory animal/ human beings having stroke. PCR (Mi-seq) and Mt-DNA sequencing could be done. Exclusion of noise data, chimera profile generation, generation of SFF-file and Q-file, Mt-DNA sequence clustering, taxonomic recognition and data assessment could be done.

V. ANSWERS TO THE POTENTIAL QUESTIONS

Shielding property of brain and reason behind the unfavourable condition for the growth fermentative bacteria. The type of breed (in case of laboratory animal) / race (in case of human beings) that has the significant difference in gut and brain microbiota which decides the progression of stroke. The spread of host gut microbiota to Brain, Liver, Pancreas, Esophagus, Gall bladder. The age-related changes in the microbiota of gut of same individual over a period of 3 years the affect the inflammation and stroke outcome.

VI. CHALLENGES

The following are the challenges : non availability of cadaver / live individuals and non availability of different race of human beings

VII. ALTERNATIVE STRATEGIES

Metaproteomics and metatranscriptomics are considered as alternative strategies.

VIII. ACKNOWLEDGMENT

The author thanks the management of KARE.

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