Hypothesis and Preliminary Results on the Role of MUC1 and MUC2 in Relationship to Autism Aetiology

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Abstract

Background: Aim autism a neurological disorder with either genetic or environmental component Autism generally presents changes of intestinal permeability to produce metabolism’s alteration in the gastrointestinal tract The intestinal macrobiota produces metabolites, opioid like peptides, that show properties experimentally associated with autism The aim of this study is to understand the cause of intestinal permeability’s alteration

Materials and methods: We determine the intestinal MUC2, on stool samples of twelve Patient, and healthy control, with Fecal Mucin Assay and analyzed the results with Mann-Whitney U Test Calculator

Results: The results of the dosage of MUC2 concentration in autistic people decrease vs and healthy control groups: this result is statistically significant: the p-value is 0.012

Discussion: The result shows an increase of MUC2, perhaps of genetic origin We hypnotize also a probable over expression on highly ipoglicosilated MUC1.

Keywords: Autism• Mucins• Intestine• Opioids• Neuropeptide

Description

Previous studies have focused, for the aetiology of autism (ASD), on genetic causes, immune system abnormality, inflammation, exposure to environmental toxins and alterations in the microbiota of the intestine [1]. The percentage of genetic heredity of ASD is about 50% among Swedish children, suggesting that not only genetic factors play an important role to develop the ASD, but also environmental factors [2,3]. Many experimental evidence shows that gastrointestinal symptoms, such as abdominal pain, gastric cancer, diarrhoea, constipation and flatulence, are common in patients with ASD [4]. A recent study [5] identified constipation as the most common symptom (85%) in children with ASD according to parental reports and assessments by pediatric gastroenterologists. The percentage of gastrointestinal symptoms varies from 23 to 70% in children with ASD [4]. In addition, gastrointestinal symptoms show a relationship with ASD severity [4,6]. Although these studies have not shown a cause-effect relationship between gastrointestinal symptoms and ASD, the results suggest that the intestine plays an important role in the etiology of ASD. Recent studies [7,8], have shown that changes in microbiomes can leave one or more microbial *fingerprints*, in their biologic activities. Microbiomes thus become chemical compounds that can characterize people with autism and can serve as markers for diagnostic purposes. More recent studies show the important role of the intestinal barrier and its permeability, in the formation of many neurological diseases, because the alteration Macrobiota’s alteration is probably the consequence of the variation of intestinal permeability. Gastrointestinal symptoms are a common co morbidity in patients with autism spectrum disorder (ASD), but the underlying mechanisms are unknown. It is anyway important to note however that the latest research has shown that autism is also linked to a possible abnormal excitation related to compounds derived from metabolic cycles involved in neuronal transmissions. Recent hypotheses suggest that some phenomena may occur as a result of an altered intestinal permeability, with a consequent incorrect metabolism of some foods. This metabolic process involves the of bioactive peptides’s formation in the gastrointestinal tract. Recent studies [9], have confirmed, such as, that casein forms a compound, beta casomorphin-7, which is an opioid like peptide. Some studies show that the production of these opioids like peptides is experimentally associated with autism these peptides pass through the brain barrier to bind receptors and prevent their smooth function. Our hypothesis is that the main aspect of this phenomenon may result from an alteration of intestinal permeability due to an imbalance of the intra and extra cellular mucin layer. The purpose of this study is to show the possible imbalance of the two mucins amount and with literature data make a reasonable hypothesis for its relationship with the opioids like peptides realise.

Materials and Methods

This study followed a procedure were in accordance with the ethical

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controls of the responsible committee on human experimentation and with the Helsinki Declaration of 1975, as revised in 2000. The independent Ethics Committee of Roma Biomed Research LAB Milan, Italy, obtained informed assent for children aged over 7 years participating in the research. To determine of the intestinal MUC2's concentration we performed, on stool samples of twelve ASD children, and healthy control, both with age between 8 and 15 years. We used Fecal Mucin Assay Kit, to extract and florometrically find the amount of mucin content in feces. Faecal mucin amount can be a measures index of intestinal barrier function.

Step 1: Extraction and partial purification of mucin from faeces.

Step 2: Determination of mucin O-glycosidically linked oligosaccharide chains is β-eliminated by diluted alkali, and reducing end of sugar chain is formed. Reducing carbohydrates are fluorescence-labelled at high temperature to produce intensely fluorescent condensate.

- Working Calibrator: N-acetylgalcosamine 250 µg/mL
- Create a standard curve by serial dilution as indicated in the Figure 1

![Standard Curve](Fig. 1 Standard Curve)

The results of the dosage of MUC 2 in ASD and healthy control (Table 1), show that the amount of MUC 2 is lower in ASD vs healthy control group and the difference is statistically significant the p-value is 0.0124.

<table>
<thead>
<tr>
<th>Table 1. Intestinal MUC2 values in autistic and healthy people.</th>
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<tr>
<td><strong>ASD</strong></td>
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<tr>
<td>Mucins (GalNAc) mg/g feces</td>
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<td>0.30</td>
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Discussion

Many genetic and environmental components can change the normal structure of intestinal mucin, whose normal structure. In any case, whatever the cause, this changes the structure of the mucous layer and its permeability.

Results

The results of the dosage of MUC 2 in ASD and healthy control (Table 1), show that the amount of MUC 2 is lower in ASD vs healthy control group and the difference is statistically significant the p-value is 0.0124.
Conclusion

The intestinal microbiome is a part of human physiology; and changes in the gut microbiota can modulate gastrointestinal physiology, immune function, and even behaviour. Links between specific bacteria from the indigenous gut microbiota and phenotypes relevant to ASD raise the important question of whether microbial dysbiosis plays a role in the development or presentation of ASD symptoms. The genes of this Human Macrobiotic complex are capable to produce opioid like peptides by certain foods that in presence of an abnormal intestinal mucosal layer play an important role in autism disorders. The use of enzymatic products such as plant-based proteases can begin to break down wheat milk proteins while they are still in the stomach. This further boost to protein breakdown leads to deeper digestion of proteins, specifically targeting the peptide bonds present in dietary opiates.

References


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