



## The Study of Intestinal Permeability and its Relationship with Autism

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### Keywords

**Aim:** To verify the development of some disorders of Autism by a metabolic alteration in the digestion of some foods for the change of the permeability of the intestinal mucus layer.

**Methods:** A critical review of the process of the formation of opioid-like peptides produced for an alteration in intestinal permeability able to overcome the encephalic lumen barrier.

**Results:** Recent studies show that some opioid peptides as the beta-casomorphin-7 pass through the encephalic barrier binding to receptors and inhibiting the normal neuronal transmitter's reuptake.

**Conclusion:** The phenomenon of the alteration of the intestinal permeability is always associated with the change in the intestinal mucin expression and with the increasing of the concentration of MUC2 (gel forming). The same phenomenon occurs in the oral mucosa when is present the diabetes; there is an increasing of the excreted mucins MUC7 and MUC5B with a modification of the Spinnbarkeit. The parametric value of mucosal adhesion, with changing its normal continuous layer in a succession of bubbles, separated by channels. So for the similar properties of MUC2 with MUC5B and MUC7, the alteration of intestinal mucosal layer may facilitating the cycle of the opioid peptides and the free radicals formation

### Keywords

Autism; Opioids like peptides; Intestinal permeability; Mucins

### Introduction

In recent times many studies have been carried out that emphasize the important role of the intestinal barrier and intestinal permeability for health and disease because an intact intestinal barrier protects the human organism against invasion of microorganisms and toxins. On the other hand, this barrier must be open to absorb essential fluids and nutrients. Such opposing goals are achieved by a complex anatomical and functional structure the intestinal barrier consists of the functional status of which is described by 'intestinal permeability'. The regulation of intestinal permeability by diet and bacteria is depicted. In particular, potential barrier disruptors such as hypo perfusion of the gut, infections and toxins also selected over-dosed nutrients, drugs, and other lifestyle factors have to be considered. The barrier assessments are further hindered by the natural variability of this functional entity depending on species and genes as well as

on diet and other environmental factors that may be the first step in the promotion or at least of the development of symptoms typical of some important diseases comprising those that affect the cerebral functions, as the autism. In fact all these diseases are characterized by inflammation that might be triggered by the translocation of luminal components into the host. In summary, intestinal permeability which is a feature of intestinal barrier function is increasingly recognized as being of relevance for health and disease and therefore this topic warrants more attention. The most and recent researches are focused on the relationship of intestinal permeability and the human gut microbiome in the brain diseases. Many studies are showed as structural bacterial components such as lipopolysaccharides provide low-grade tonic stimulation of the innate immune system or excessive stimulation due to bacterial dysbiosis produces a small intestinal bacterial overgrowth or increased intestinal permeability with a systemic and/or central nervous system inflammation. Bacterial enzymes may produce neurotoxic metabolites such as D-lactic acid and ammonia, even beneficial metabolites such as short-chain fatty acids may exert neurotoxicity. Gut microbes can produce hormones and neurotransmitters that are identical to those produced by humans. Bacterial receptors for these hormones influence microbial growth and virulence. Gut bacteria directly stimulate afferent neurons of the enteric nervous system to send signals to the brain via the vagus nerve.

The aim of my studies is to study the possible alterations of the mucins that play a fundamental role in the formation and proper functioning of the intestinal barrier. We are studying this process in relation to the presence of symptoms present in autism and which may be related to proper alteration of intestinal permeability. I refer to the phenomenon of so-called OPIOIDS LIKE-PEPTIDES [1,2].

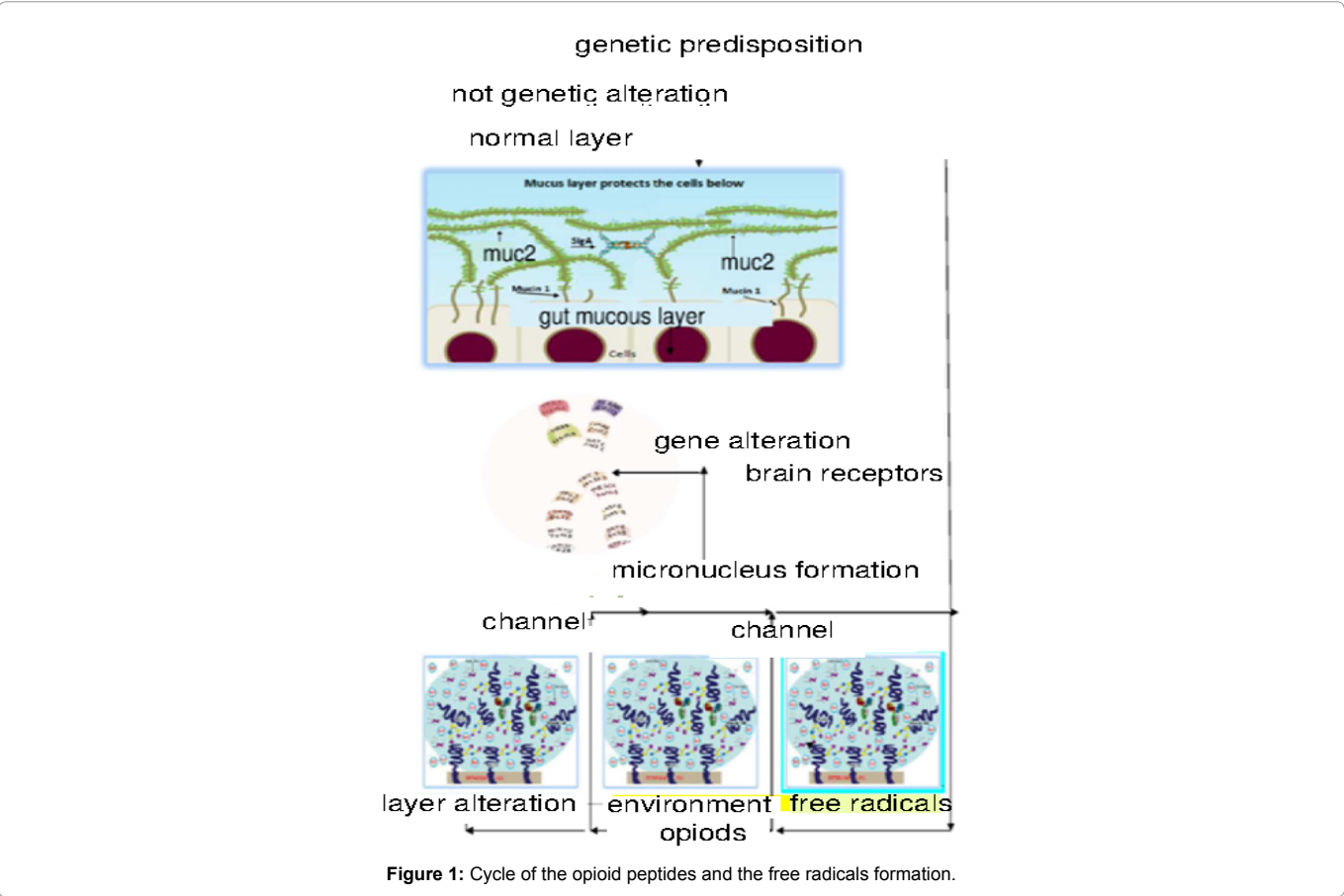
Recent studies show that some opioid peptides as the beta-casomorphin-7 [3,4] pass through the encephalic barrier, binding to receptors and inhibiting the normal neuronal transmitters reuptake.

The phenomenon of the alteration of the intestinal permeability is always associated with the change in the intestinal mucin expression and with the increasing of the concentration of MUC2 (gel forming). The same phenomenon occurs in the oral mucosa when is the diabetes there is an increasing of the excreted Mucins MUC7 and MUC5B with a modification of the Spinnbarkeit the parametric value of mucosal adhesion with changing its normal continuous layer in a succession of bubbles separated by channels. So for the similar properties of MUC 2 with MUC5B and MUC7, the alteration of intestinal mucosal layer may facilitating the cycle of the opioid peptides and the free radicals formation below see flow sheet (Figure 1).

In autism the presence of a modification of normal intestinal mucous layer in the cause valves with a complex feed-back mechanism, a stream of biochemical compounds [5-7] that further alter central nervous structures. It is possible restoring the conditions that are underlying the intestinal permeability and inhibit the reuptake of opioids.

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Received: October 12, 2017 Accepted: October 13, 2017 Published: October 17, 2017



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