

## The Ghost Aim in Medical Research – A Subjective Limit in Intake to Prevent Fattening/Insulin Resistance/Overall Inflammation

Mario Ciampolini\*, Gaia Cecchi

Department of Paediatrics, University of Florence, 50132 Florence, Italy.

**Corresponding Author:** Mario Ciampolini, Department of Paediatrics, University of Florence, Florence, Italy, **Tel:** 039055215744;

**Email:** mlciampolini@fastwebnet.it

**Received Date:** 01 Mar 2017

**Accepted Date:** 02 Mar 2017

**Published Date:** 08 Mar 2017

**Copyright** © 2017 Ciampolini M

**Citation:** Ciampolini M and Cecchi G. (2017). The Ghost Aim in Medical Research - A Subjective Limit in Intake to Prevent Fattening/Insulin Resistance/Overall Inflammation. *M J Pedi.* 2(1): 009.

### COMMENTARY

The division had a start when I read the Handbook of Physiology of the American Society for Physiology, in 1967. I was charged with the treatment of malnutrition and diarrhoea in the University of Florence. I read the handbook to become aware about mucosal digestion and absorption. At that time, these points had to be diagnosed to treat malnourished children. Before beginning any research, I intended to adapt intake to intestinal physiology. I read that 50% - 60% or more immune cells of the human body reside in the mucosa of small intestine (Mowat, 1987, 44; Brandtzaeg et al., 1989; Abrams, 1977). Bacteria grow in small and large intestine by slow energy production without oxygen use. All fibers and small amounts of sugars, carbohydrates, proteins, fats escape intestinal digestion and provide energy for one bacterial replication per day (Hungate, 1967). Food avoided absorption due either to excessive intake or to incapability to be digested. Fibers like cellulose and pectin escape digestion and promote slow growth of poorly immunogenic bacteria species and prevent harmful microbiome developments. Bacterial growth becomes immunogenic and harmful when energy dense food is largely available. In the mammal intestine, bacterial indiscriminate, harmful growth is proportionate to a positive energy balance in blood and in body. Thus I studied bacteria number on intestinal mucosa in time after last meal. A longer interval from the meal produced a decrease in bacteria number. An increase in mucosal and overall immune stimulation is associated with bacteria growth on intestinal mucosa, with preprandial blood glucose (BG) and with a slowdown of meal absorption (Ciampolini et al., 1996; 2000). Energy balance directly affects these correlated variables either increasing or lowering the conflict between bacteria activity and mucosal immune response. The initial hunger meal pattern (IHMP) was devised to reduce bacterial growth and reduce the mucosal

immune response at nutrient absorption. This conflictual state between bacteria and mucosa has been confirmed (Cooper, Sاداتy, 2014; McCoy, Köller, 2015). The many successful cures of gastrointestinal pathologies suggest that the conflictual theory that was used for recovery was objective, i.e. reproduced the events in small and large intestine. In this view, the question: "what food provokes cancer?" is absurd. Malignancy needs to be surveilled and prevented through an increase in efficiency of immune system (Kristensen, 2017). Lower intestinal stimulation may be the way to achieve higher immune efficiency also in the body (Abrams, 1977; Brandtzaeg et al, 1989; Ciampolini et al, 1996; Kubes, Meahl, 2012; Lynch, Pedersen, 2016). Thus health (general immune efficiency) follows the relation between energy intake and expenditure. Hundreds or thousands of bacterial species live and multiply in the intestine, 5% - 15% of the species elicit IgG production, the intestinal mucosa responds with an immune reaction and, lastly, the immune stimulation by bacterial antigens spreads of to all body tissues (Overall Subclinical Inflammation). Thus, many observations sustain the conflictual view for the absorption of every energy dense food. Unfortunately, a different, confounding opinion may be repeated by a high number of researchers and become the dominant view. Now, hundreds of scientific Journals ask me for submitting articles. I am alone and cannot produce hundred articles that are new and different each other to repeat the statements about the conflictual absorption and the IHMP solution for health maintenance and recovery. Yet, the upsurge of malignant and vascular risks, not to mention malnutrition that affects one billion of malnourished people, impose to spread the awareness on this "ghost aim".

### ACKNOWLEDGEMENTS

This review has been shown in: "Modifying Eating Behavior: Novel Approaches for Reducing Body Weight,

Preventing Weight Regain and Reducing Chronic Disease Risk”  
ASN’s Annual Meeting & Scientific Sessions at

Experimental Biology 2014, April 26-30.

## REFERENCES

1. Abrams GD. (1977). Microbial effects on mucosal structure and function. *Am J Clin Nutr.* 30(11): 1880-1886.
2. Brandtzaeg P, Halstensen TS, Kett K, Krajci P, et al. (1989). Immunobiology and Immunopathology of Human Gut Mucosa: Humoral Immunity and Interaepithelial Lymphocytes. *Gastroenterology.* 97: 1562-1584.
3. Ciampolini M, Bini S and Orsi A. (1996). Microflora persistence on duodeno-jejunal flat or normal mucosa in time after a meal in children. *Physiol Behav.* 60: 1551-1556.
4. Ciampolini M, Borselli L and Giannellini V. (2000). Attention to Metabolic Hunger and Its Effects on Helicobacter pylori Infection. *Physiology & Behavior.* 70: 287-296.
5. Ciampolini M and Bianchi R. (2006). Training to estimate blood glucose and to form associations with initial hunger. *Nutr Metab (Lond).* 3: 42.
6. Ciampolini M and Sifone M. (2011). Differences in maintenance of mean Blood glucose (BG) and their association with response to “Recognizing Hunger”. *Int J Gen Med.* 4: 403-412.
7. Ciampolini M, Love II-Smith D and Sifone M. (2010). Sustained self-regulation of energy intake. Loss of weight in overweight subjects. Maintenance of weight in normal-weight subjects. *Nutr Metab (Lond).* 7: 1-4.
8. Cooper IF and Siadaty MS. (2014). ‘Bacteriums’ associated with ‘Blood Glucose Level Finding’: *Bio Med Lib Review.*
9. Hungate RE. (1967). Ruminal fermentation. In Heidel W, Code CF. *Handbook of physiology, sect 6, Alimentary canal.* Washington DC: Am Physiol Soc. 2725-2746.
10. Kristensen VN. (2017). The antigenicity of the tumor cell. *N Engl J Med.* 376: 491-493.
11. Kubes P and Meahl WZ. (2012). Sterile Inflammation in the Liver. *Gastroenterology.* 143: 1158-1172.
12. Lynch SV and Pedersen O. (2016). The Human Intestinal Microbiome in Health and Disease. *N Engl J Med.* 375: 2369-2379.
13. Mccoy K and Köller Y. (2015). New developments providing mechanistic insight into the impact of the microbiota on allergic disease. *Clin Immunology.* 159(2): 170-176.
14. Mowat A Mcl. The cellular basis of Gastrointestinal Immunity. In: Marsh MN Ed, *Immunopathology of the Small Intestine.* Wiley J and Sons, Chichester UK, Page 44.
15. Van der Waaij LA, Limburg PC, Mesander G and van der Waaij D. (1996). In vivo IgA coating of anaerobic bacteria in human faeces. *Gut.* 38(3): 348-354.