

## The Biological Role of Intestine: Overall Inflammation

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Received date: October 24, 2018; Accepted date: November 19, 2018; Published date: November 26, 2018

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**Keywords** Blood glucose; Diabetes; Insulin resistance; Limit in energy intake; Hunger; Meal onset

**List of Abbreviations:** BG: Blood Glucose; an index of energy availability in blood for the whole body; Initial Hunger Meal Pattern (IHMP): Energy intake is adjusted to three arousals of initial hunger per day; Initial Hunger: This consists of gastric pangs or physical weakness associated with low blood glucose that arise after meal suspension for either few minutes or hours; This arousal misses in diabetic patients; MBG: mean blood glucose: mean of BG measurements before three main meals in a week.

During research activity, you may collect insights that you do not publish. E.g., I gave a non-pathogenic *Escherichia coli* to newborn mice. I obtained nothing and I was unable to publish significant results. I was however able to see damages to fuzzy coat, epithelial cells and to see infiltration by lymphocytes and monocytes in the mucosa of some tracts of the small bowel. In other intestinal segments there was however no difference from controls. In experimental mice, the choice of the segment determined the difference amount from control mice. I could not transform these observations into numerical differences between experimental and control mice. I repeated the experiments in vitro [1,2]. Bacteria were capable of destruction on epithelial cells but those that grow in duodenum are soon killed. The small intestine transfers the devitalized bacterial bodies to all body. Before speaking about this invasion, we notice that dividing bacteria into pathogenic and non-pathogenic is too simple. About thousand bacteria species grow in the intestine. About half do not interact with mucosa. The mucosa puts 1017 IgA immunoglobulin into intestine every day. These molecules do not kill bacteria, the IgA molecules only obstacle invasion by bacteria. The responses by immunoglobulin IgM and IgG are low but are capable of inducing inflammation in intestinal mucosa and in all body tissues. The amount of immune cells in the mucosa is impressive. Mowat, Brandtzaeg, van der Waij [3-5] calculated that more than half immune cells in the body were developing in the intestinal mucosa. This impressive defense is active against intestinal microflora [1,2].

No adaptation to a warm environment by lowering energy intake, promotes relapses of functional disorders, e.g. diarrhea in children. Yet the diarrhea relapses in children were associated with high blood glucose (BG) [6]. BG elevation and long persistence of nutrients in the bowel are frequent events during changes in climate or in home heating and during emotions [7-12]. These events (of no metabolic adaptation) prevail more and more in the evolution from insulin resistance to diabetes [13-19]. Fattening is a way to slow and delay the development of diabetes, but has own limits. In the evolution to

diabetes emerges the overall inflammation that is associated to high Mean BG (MBG), insulin resistance, fattening and other functional disorders and deteriorations. Overall inflammation multiplies cellular reproduction in all body tissues. DNA replications increase and produce more and more replication errors in DNA [9-12]. After decades of accelerated replications, malignancy development becomes unavoidable. Although frequent however, high MBG and insulin resistance are not generalized and may be reversed to low MBG and insulin sensitivity by changes in meal pattern [9-12].

All these biological findings and demonstrations suggested that nutrition is conflictual in the subdivision of energy rich nutrients between bacteria proliferation and mucosal absorption. Good nutrition should prevent bacterial growth in the intestine. This was achieved by eating after Initial Hunger in humans. This consists of gastric pangs or physical weakness associated with low blood glucose and arises after meal suspension. Man might eat in order to have a new event of hunger after a desired number of hours. The application of this conflictual view in subjects with relapsing complaints stopped relapsing events better than the application of other clinical suggestions like nutrient deficiency [6,9,12]. To be sure, a subject must check the correspondence of hunger and body sensation with BG measurements [9-12]. After this learning, the subject ought to learn the amount of energy that is necessary to prevent IH arousal in the subsequent hours: e.g., for 4 or 6 hours. In this calculation, the subject must be aware of differences in dependence of climate, physical activity etc. Changes in climate and activity modify expenditure and this needs modification in intake to maintain energy balance. No adaptation to low expenditure by lowering energy intake produces increase in BG, intestinal slowdown, bacteria overgrowth, mucosal inflammation and overall inflammation. Overall inflammation is transient, few days in children with diarrhea, and it shows spontaneous regression. Decades of maintenance are possible [18,19], but DNA may remain damaged (oncogene) in the site of Inflammatory persistence.

### Acknowledgement

The Author acknowledges the indispensable collaboration in writing with Stella Zagaria, David Lowell-Smith (NZ) and Riccardo Bianchi (NY), and the strategic, statistical support by Cutberto Garza (Rector, Boston College), Giuliano Parrini (Professor of Physics, Firenze) and Andrea Giommi (Professor of Statistics, Firenze). The here summarized researches were supported by the Italian Ministry of University, Research, Science and Technology grants for the years 1998-2002 and by ONLUS Nutrizione e Prevenzione, Firenze, for the years 2003-2012.

## Conflict of Financial Interests

No conflicts of interest

## References

1. Ciampolini M, Becciolini PA, Marianelli I (1969) Azione dei germi nel tenue. Azione sulla saccarasi e leucinaminopeptidasi in vitro. *Clinical Pediatrics* 82: 99-104.
2. Ciampolini M, Marianelli L (1969) Azione dei germi nel tenue. Nota II: E.Coli nel ratto lattante. Studio microbiologico, istologico e biochimico. *Clinical Pediatrics* 82: 78-84.
3. Mowat AM (1987) The cellular basis of gastrointestinal immunity. In: Marsh MN (editor) *Immunopathology of the small intestine*. John Wiley & Sons, Chichester 44.
4. Brandtzaeg P, Halstensen TS, Kett K, Krajci P, Kvale D, et al. (1989) Immunobiology and Immunopathology of Human Gut Mucosa: Humoral Immunity and Intraepithelial Lymphocytes. *Gastroenterology* 97:1562-1584.
5. Van der Waaij LA, Limburg PC, Mesander G, van der Waaij D (1996) In vivo IgA coating of anaerobic bacteria in human faeces. *Gut* 38: 348-354.
6. Ciampolini M, Vicarelli D, Seminara S (1990) Normal energy intake range in children with chronic non-specific diarrhea. Association of relapses with the higher level. *Journal of Pediatric Gastroenterology and Nutrition* 11: 342-50.
7. Ciampolini M (1974) Influence of environmental temperature on intestinal absorption xylose in rats in vivo. *IRCS* 2:1545.
8. Ciampolini M (1976) Influence of environmental temperature on xylose absorption in man. *IRCS Medical Science* 4: 208.
9. Ciampolini M (2018) Initial Hunger, a Subjective, Reproducible Limit in Intake Associated with Low Blood Glucose: A Training for Malnourished Infants and Overweight Adults. In: Preedy V, Patel VB (editors). *Handbook of Famine, Starvation, and Nutrient Deprivation*. Springer International Publishing AG, part of Springer Nature.
10. Ciampolini M, Lovell Smith D, Sifone M (2010) Sustained self-regulation of energy intake. Loss of weight in overweight subjects. Maintenance of weight in normal-weight subjects. *Nutrition and Metabolism* 7:1-4.
11. Ciampolini M, Lovell-Smith D, Bianchi R, de Pont B, Sifone M, et al. (2010) Sustained self-regulation of energy intake. Initial hunger improves insulin sensitivity. *Journal of Nutrition and Metabolism* Article ID 286952, 7 pages.
12. Ciampolini M, Sifone M (2011) Differences in maintenance of mean Blood glucose (BG) and their association with response to "Recognizing Hunger". *International Journal of General Medicine* 4:403-412.
13. Abrams GD (1977) Microbial effects on mucosal structure and function. *American Journal of Clinical Nutrition* 30:1880-1886.
14. Ciampolini M, Bini S, Orsi A (1996) Microflora persistence on duodeno-jejunal flat or normal mucosa in time after a meal in children. *Physiology and Behaviour* 60: 1551-1556.
15. Ciampolini M, Borselli L, Giannellini V (2000) Attention to Metabolic Hunger and Its Effects on *Helicobacter pylori* Infection. *Physiology & Behavior* 70:287-296.
16. Cooper IF, Siadaty MS (2014) 'Bacteriums' associated with 'Blood Glucose Level Finding'. *BioMedLibrary Review* 26:1322-1326.
17. McCoy K, Köller Y (2015) New developments providing mechanistic insight into the impact of the microbiota on allergic disease. *Clinical Immunology* 159:170-176.
18. Mauron J, Anantharaman K, Finot PA, Horisberger M, Ingenbleek Y, Wuerzner HP (1982) Nutritional adequacy, nutrient availability and needs. Springer AG, Basel 44.
19. Jayedi A, Emadi A, Shab-Bidar S (2018) Dietary Inflammatory Index and Site-Specific Cancer Risk: A Systematic Review and Dose-Response Meta-Analysis. *Advances in Nutrition* 9: 388-403.