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Mini Review

Factors that Have a Major Role

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Abstract

A largely demonstrated causal chain crosses human life. Conditioned eating/fattening & insulin resistance/slowdown of absorption and progression of nutrients/pathologic modification of microbiome (increase in expansion, change in proportion among species, prevalence of immunogenic species) immune stimulation. The state of increased (either acute or chronic) immune stimulation has the name of Overall Subclinical Inflammation. We have trained people to take food after perceiving signals of hunger (Initial Hunger Meal Pattern, IHMP): after training, subjects have 30% lower energy intake for months and years. This lower intake changes their life: prevents fattening/insulin resistance, slows down absorption and progression of nutrients, prevents alteration of microbiome, overall inflammation, functional disorders, vascular risks and malignancy development. This state promotes malignancy by greatly increasing cell and DNA replications and errors DNA replications in correlation to the overall inflammation and the lifelong increase in energy intake. To understand the origins of malignancies, research must focus the increase in DNA replications that is associated with Overall Inflammation and conditioned eating. The alternate conditions of functional disorders are paralleled by microbiological events.

Keywords: Blood Glucose; Energy

Abbreviations

BG: Blood Glucose, an index of energy availability in blood for the whole body; IH: Initial Hunger consists of gastric pangs or mind or physical weakness: India is the Italian word for this weakness. In sedentary adults and in children, IH corresponds to 76.6 ± 3.7 mg/dL BG. In infancy corresponds to demand before sight of food. IHMP: Initial Hunger Meal Pattern: Energy intake is adjusted to three arousals of IH per day; OGTT: Oral Glucose Tolerance Test; AUC: Area Under Curve of GTT; MB: The mean of 21 BG measurements before the three main daily meals reported by a week diary. MBG measures the compliance with IHMP, MBG shows changes after training and it is negatively correlated to insulin sensitivity. Below 81.8 mg/dL (Low MBG) MBG indicates a healthy meal pattern in sedentary people. Over 81.8 mg/dL, High MBG is associated with fattening/insulin resistance; NSV: Non-starchy Vegetables, food with lower content than 30 kcal/100 grams.

Introduction

About thousand bacteria species grow in the intestine. Among 1000 total species in large intestine, 10 to 100 species do not provoke a known illness like salmonella or cholera but interact with the immune system. E. coli includes strains that are pathogenic by multiple mechanisms (by invasion, by enterotoxin, etc.). In proportion to the growth e.g., the E. coli stimulate production of IgG and IgM and inflammations. About half do not interact with mucosa [1,2]. I gave a not pathogenic Escherichia coli to newborn mice [3]. I was unable to publish significant results. I was however able to see spot damages to fuzzy coat, epithelial cells and to see infiltration by lymphocytes and monocytes [3]. 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 significant numerical differences between experimental and control mice. I repeated the experiments *in vitro* [4]. Bacteria were capable of destruction on epithelial cells. The small intestine transfers the invasion from local to all body tissues. The mucosa puts 1017 IgA molecules into intestine every day. These molecules do not kill bacteria, the IgA molecules only obstacle invasion by bacteria. There are low responses by IgM and IgG, these are capable of inducing inflammation. 50% - 60%

or more immune cells of the human body reside in the mucosa of small intestine [5-9]. Bacteria grow in the small and large intestine in dependence on nutrients, mainly those nutrients that produce energy availability (sugars, carbohydrates, amino-acids, fats [1]. These events were concluded after I studied bacteria number on the intestinal mucosa in time after last meal.

The number of immune cells in the mucosa is impressive. Mowat, Brandtzaeg [5-9] calculated that more than half immune cells in the body were developing in the intestinal mucosa. This defense acted against intestinal microflora [1-9]. The diarrhea relapses in children were associated with high blood glucose (BG) [10-14]. BG elevation and long persistence of nutrients in the bowel are frequent events during changes in climate or in home heating [7-9]. No adaptation by lower energy intake to warm increase promotes relapses of functional disorders, diarrhea in children. These events (of no metabolic adaptation) prevail more and more in the evolution from insulin resistance to diabetes. Fattening is a way to slow and delay (transiently!) the development of diabetes. In the evolution to diabetes emerges the overall inflammation that is associated to high BG, insulin resistance, other functional disorders and deteriorations.

All these biological findings and demonstrations suggested that nutrient absorption is conflictual or at least competitive. In the conflict, good nutrition should prevent bacterial growth in the intestine. This was achieved by eating after Initial Hunger in humans. 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 interpretation like nutrient deficiency [10-13]. A subject must learn the correspondence of hunger and body sensation with BG measurements. 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, activity etc. A longer interval from the meal produced a greater decrease in bacteria number. This association was exploited in chronic diarrhea [12-15], repeated vomiting and Malnutrition [16], Thus I concluded that meal absorption develops in a competition between mucosa cells and bacteria [8,9]. The conflictual nature of mucosal absorption has been confirmed.

In past studies, energy availability and Mean Blood Glucose (MBG) are maintained at different levels within the (sedentary) normal variability between 65 mg/dL and 110 mg/dL before meals and before any training on energy intake [11,12]. After bearable meal suspension, IH and MBG correspond to 76.6±3.7 mg/dL BG. The MBG before any training may be decreased from 91.6 ± 7.7 mg/dL to 76.6 ± 3.7 mg/dL after an ad hoc training [11,12]. This MBG decrease is associated with decrease of insulin resistance but no perception of subjective hunger [11,12]. The associated decreases (BG and insulin resistance) imply an abatement of Overall Subclinical Inflammation. The inflammation is associated with increased turnover in immune and parenchymal cells, and lifelong, increased DNA replications. After years and decades, errors in DNA replication cumulate and promote malignancies [17-20]. This view corresponds to the fact that only a consistent minority (about 35%) of population maintains insulin resistance, high BG, overall inflammation on one hand and cancer development on the other. Prevention thus is uneasy as eating control up to now. Training recognition of Initial Hunger and prediction of current BG is easy in young people [10,11]. After this learning, people decrease energy intake by a mean 30% (of recruitment value). In animal studies, this decrease was associated with significant decrease of cancer development [17].

Pollution has been often considered as a cause of malignancies or of their increase. A more scientific objective in research, the increase in DNA replication over years and decades may be more fruitful.

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Conflict of Interests

No conflicts of interest

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