



International Journal of Clinical Endocrinology

Opinion

Feeding either Conditioned or by Demand -

Mario Ciampolini*

Preventive Gastroenterology Unit, Department of Pediatrics, Università di Firenze 50132 Florence, Italy

***Address for Correspondence:** Mario Ciampolini, Preventive Gastroenterology Unit, Department of Pediatrics, Università di Firenze 50132 Florence, Italy, E-mail: mlciampolini@fastwebnet.it

Submitted: 06 November 2017; **Approved:** 25 November 2017; **Published:** 27 November 2017

Cite this article: Ciampolini M. Feeding either Conditioned or by Demand. *Int J Clin Endocrinol.* 2017;1(2): 056-058.

Copyright: © 2017 Ciampolini M. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

There is evidence that conditioned hunger promotes fattening/diabetes that cannot be actually contrasted if conditioned intake is scientifically accepted as normal. The adoption of a meal pattern based on demand is easy in the first days of life and effective in suppressing or reducing conditioned intake. The National Child Study might compare the prevalence of diseases development between rearing children by conditioned intake versus rearing by demand. In the case of a failure, the Study shall show the role of conditioned eating on fattening diabetes.

Keywords: Blood Glucose; Diabetes; Insulin Resistance; Overweight; Fattening; Energy Balance; Energy Intake; Limit in Energy Intake; Hunger; Meal Onset; Energy Availability; Bowel disorders; Malnutrition.

BACKGROUND

There is evidence that conditioned hunger promotes fattening/diabetes [1-6] that cannot be actually contrasted if conditioned intake is scientifically accepted as normal and equivalent to intake after meal suspension. Conditioned hunger is usually modest as a sensation, but may be reported as intense as or even more intense than the hunger sensation after exhaustion of previous intake or meal suspension [1,2]. Conditioned hunger is even associated with peristaltic movements in small intestine. The distinction is uneasy and requires long focusing on subsequent meal onsets for about a week [1-3]. The main difference resides in the onset: either before or after noticing meal cues (e.g., sight of food, ready table or prepared commensals before a meal, etc.) [1-3]. The BG measurement by portable device is useful to confirm recognition of the same metabolic state of low energy availability [1,2]. Adults were trained by measuring BG at hunger perception and after training, were able to predict BG [1-3]. The American J Clinical Nutrition and other Authorities did not accept portable measurements, defending the use of conditioned eating (status quo) as signal for eating. High preprandial BG might be compensated by lower meal intake, but this revealed to be not true [3-6]. In our investigations measuring BG by a portable device has the unique chance of biochemical checking at the moment of arousal of hunger sensation [4]. Eighties-and-five adult subjects measured BG in a blood sample that was drawn for autoanalyzer measurement. The absolute difference was about 5 mg/dL and this difference does not hamper statistical conclusions in comparison with mean differences between meal patterns of about 20-30 mg/dL. Measurements by glucometer at hunger arousal have about 5 mg/dL mean absolute error, while by autoanalyzer show 1% error in daily confrontation among 50 labs in Tuscany. We attribute the inconsistency of fasting BG to variations in BG from a minute to another. Every twelve minutes glucose influxes into blood from liver and BG increases by about 10% [7-16]. Fasting BG includes different dinner digestive situations. Sometimes, dinner absorption from small intestine has been not completed in the morning. Inconsistency in fasting BG is consequent to this uncertain absorption time and not to portable measurements. Instead the moment before meals is definite and reproducible [17-22]. Low BG was 76.6 ± 3.7 mg/dL at the definite moment of IH perception (Initial Hunger). The confidence interval for this measurement is 3.2 mg/dL [18]. The Low BG sharply distinguishes IH from conditioned intake.

CURRENT MEDICAL ASSISTANCE

Reasonable outlooks sometimes suggest no information of “lay” readers about consequences of a usual behavior like conditioned eating [23]. This partial information is sometimes chosen by Medical Journals with high impact factor. In these circumstances, authorities that detain big communication canals decide what is right or wrong for all [23]. This way, Science is substituted by an arbitrary opinion

and a large part of population will go on by eating a mean 20% energy surplus at every meal [17-23]. Events like vascular and malignant diseases will develop unexplained. Medicine cannot be rigid as Science, but if authorities chose what reports are true or false Science ends. We came across the National Child Study (NCS) [24].

The NCS was conceived in the late 1990s and authorized through the Children’s Health Act of 2000. It was intended to be a prospective, epidemiologic, birth-cohort study that would follow a nationally representative cohort of 100,000 U.S. children from shortly after conception to 21 years of age and possibly possibly beyond. - A “children’s Framingham study”. The study was catalyzed by rising rates of chronic diseases in children. Increases in asthma, autism, birth defects, dyslexia, attention deficit–hyperactivity disorder, schizophrenia, obesity, and diabetes that were too rapid to be of genetic origin-and by growing concern over children’s exposure during vulnerable stages of early development to hundreds of new and untested chemicals [2]. The goal of the NCS, like that of the Framingham study, was to identify preventable risk factors for disease.

CONCLUSION

The CNS list of diseases includes at least two components, obesity and diabetes that depend on energy intake, unequivocally [24]. The choice between scheduled and requested meals may be a historical or fashion issue. In the first days of life, the two choices are equivalent and are dictated by familial and physician customs, local current fashion, convenience. A null effect hypothesis between scheduled and demanded meals has been rejected by our studies in infants [18-20]. Given the facts that,

- a. By free choice 30% of the population maintains preprandial low blood glucose like during IHMP [18-22].
- b. Demanded meals (IHMP) have been shown to be maintained up to 12 years of age [18-28].
- c. The equivalence of early instructions for novel mothers [20].
- d. The habitual, persistent nature of mean BG due to associated organic changes, and emphasizing the better health in children and adults who maintain IHMP [18-28], we suggest that a change in instructions on rearing is obvious and mandatory from the neonatal days. The CNS might take into consideration energy intake, a real issue in USA [25]. The Study might compare the prevalence of diseases development between rearing children by conditioned intake versus rearing by demand. At least, prevention of fattening/diabetes will become feasible.

REFERENCES

1. Mayer J. Glucostatic mechanism of regulation of food intake. *N Engl J Med.* 1953; 249: 13-16. <https://goo.gl/5qhdD>

2. Steffens AB: The influence of insulin injections and infusions on eating and blood glucose level in the rat. *Physiol Behav.* 1969; 4: 823-828. <https://goo.gl/in1JoZ>
3. Melanson KJ, Westerterp-Plantenga MS, Campfield LA, Saris WHM. Blood glucose and meal patterns in time-blinded males, after aspartame, carbohydrate, and fat consumption, in relation to sweetness perception. *Br J Nutr.* 1999; 82: 437-446. <https://goo.gl/EbMb3B>
4. Campfield LA, Smith FJ. Functional coupling between transient declines in blood glucose and feeding behavior: temporal relationships. *Brain Res Bull.* 1986; 17: 427-433. <https://goo.gl/A328Cg>
5. Campfield LA, Smith FJ, Rosenbaum M, Hirsch J. Human eating: evidence for a physiological basis using a modified paradigm. *Neurosci Biobehav Rev.* 1996; 20: 133-137. <https://goo.gl/qfAEut>
6. Campfield LA, Smith FJ. Blood glucose dynamics and control of meal initiation: a pattern detection and recognition theory. *Physiol Rev.* 2003; 83: 25-58. <https://goo.gl/V7n6yp>
7. Woods SC, Stein LJ, McKay LD, Porte D Jr. Suppression of food intake by intravenous nutrients and insulin in the baboon. *Am J Physiol.* 1984; 247: R393-R401. <https://goo.gl/B2TbgR>
8. Gavin JR. Pathophysiologic mechanisms of postprandial hyperglycemia. *Am J Cardiol.* 2001; 88: S4-S8. <https://goo.gl/Skvwww>
9. de Graaf C, Blom WAM, Smeets PAM, Stafleu A, Hendriks HFJ: Biomarkers of satiation and satiety. *Am J Clin Nutr.* 2004; 79: 946-961. <https://goo.gl/MxX6Qm>
10. Elliott SS, Keim NL, Stern JS, Teff K, Havel PJ. Fructose, weight gain, and the insulin resistance syndrome. *Am J Clin Nutr.* 2002; 76: 911-922. <https://goo.gl/sgP9mA>
11. Vea H, Jorde R, Sager G, Vaaler S, Sundsfjord J. Glycemic thresholds for hypoglycemic responses in obese subjects. *Int J Obes Relat Metab Disord.* 1994; 18: 111-116. <https://goo.gl/ZvyWfr>
12. Chapelot D, Marmonier C, Aubert R, Gausseres N, Louis-Sylvestre. A role for glucose and insulin preprandial profiles to differentiate meals and snacks. *Physiol Behav.* 2004; 80: 721-731. <https://goo.gl/hcEWZg>
13. Nicolaidis S, Even P. Spontaneous and 2DG Induced metabolic changes and feeding: the ischymetric hypothesis. *Brain Res Bull.* 1985; 15: 429-435. <https://goo.gl/qd6mmb>
14. Jenkins DJA, Wolever TMS, Jenkins AL, Josse RG, Wong GS. The glycemic response to carbohydrate foods. *Lancet.* 1984; 2: 388-391. <https://goo.gl/vCJFg3>
15. Cryer PE. Glucose counterregulation: prevention and correction of hypoglycemia in humans. *Am J Physiol.* 1993; 264: E149-E155. <https://goo.gl/yiLixh>
16. Barnett JL, Owyang C. Serum glucose concentration as a modulator of interdigestive gastric motility. *Gastroenterology.* 1988; 94: 739-744. <https://goo.gl/GEgR4J>
17. Ciampolini M, Bianchi R. Training to estimate blood glucose and to form associations with initial hunger. *Nutr Metab (Lond).* 2006; 3: 42. <https://goo.gl/92tL4H>
18. Ciampolini M, Sifone M. Differences in maintenance of mean Blood glucose (BG) and their association with response to "Recognizing Hunger". *I J Gen Med.* 2011; 4: 403-412. <https://goo.gl/ydbmfB>
19. Ciampolini M. Editor: Meal by meal dynamic balance of energy in blood. *Research Signpost.* 2011; 37/661(2), Vazhappalli Jn., Fort Post Office, Trivandrum-695 023, Kerala, India. ISBN: 978-81-308-0457-6.
20. Ciampolini M. Requested meals versus scheduled meals. *I J Gen Med.* 2012; 5: 1-9. <https://goo.gl/sjXQhX>
21. Ciampolini M, Brenna JT, Giannellini V, Bini S. Interruption of scheduled, automatic feeding and reduction of excess energy intake in toddlers. *Intern J Gen Med.* 2013; 6: 39-47. <https://goo.gl/9AStXL>
22. Ciampolini M, Lovell-Smith D, Sifone M. Sustained self-regulation of energy intake. Loss of weight in overweight subjects. Maintenance of weight in normal-weight subjects. *Nutr Metab (Lond).* 2010; 7: 1-4. <https://goo.gl/648y4c>
23. Ciampolini M. Medicine versus Science. *EC Nutrition.* 2017; 211: 60-62. <https://goo.gl/H7B2aC>
24. Landrigan PJ, Baker B. The National Children's Study-end or New Beginning? *New England J of Med.* 2015; 372: 1486-1487. <https://goo.gl/vHuDEq>
25. Gregg EW, Shaw JE. Global Health Effects of Overweight and Obesity. *The New England Journal of Medicine.* 377: 80-81. <https://goo.gl/RtwuYJ>
26. Ciampolini M, Bini S, Giommi A, Vicarelli D, Giannellini V. Same growth and different energy intake in chronic non-specific diarrhea children in a four-year period. *Intern J Obesity.* 1994; 18: 17-23.
27. Ciampolini M, Borselli L, Giannellini V. Attention to metabolic hunger and its effects on *Helicobacter pylori* infection. *Physiol Behav.* 2000; 70: 287-296. <https://goo.gl/427Cds>
28. Fisher JO, Birch LL. Eating in the absence of hunger and overweight in girls from 5 to 7 y of age. *American Journal of Clinical Nutrition.* 2002; 76: 226-231. <https://goo.gl/hXY2aC>