International Journal of Science and Research (IJSR)
ISSN: 2319-7064
SJIF (2022): 7.942

Managing Caloric Homeostasis and Postprandial Glucose: Strategies for Preventing Metabolic Disorders

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Abstract: ***Calorie or energy management plays a vital role in maintaining metabolic homeostasis and preventing disorders. The gastrointestinal tract serves as the primary control point for nutrient absorption, with glucose being a key factor in the development of ectopic fat deposits, particularly in the liver and pancreas. Unregulated glucose absorption leads to inflammatory conditions, contributing to metabolic disorders like fatty liver disease and atherosclerosis. Effective glucose management strategies, including inhibiting specific transporters and performing pre and postmeal exercises, can help control postprandial glucose levels, reduce ectopic fat deposits, and maintain overall metabolic health. This article explores various management approaches aimed at sustaining calorie balance and preventing metabolic dysfunctions.***

Keywords: caloric homeostasis, postprandial glucose, metabolic disorders, ectopic fats, glucose transporters

Calorie or energy management of the body is most essential for homeostatis of metabolic disorder and required calorie management rather than calorie burnt by exercise. For E. g in River water management, we construct dam in the river origin for quality distribution of water throughout the year. Similarly when we take food, all foods together gather in GI tract, from there it will pass through intestinal lumen (which act as barrier) between GI and enterocyte. Then it passes from enterocyte to blood circulation. Here we can say that intestinal tract is the origin of the river gathering all foods together and intestinal wall serve as dam, the carbohydrates, fats and protein transform to amino acid, fatty acids and glucose, fructose etc by different types of digestive enzymes and then these fatty acids, amino acids and glucose transported to enterocyte and from enterocyte it is again transported to blood circulation as river flow from the dam.

After transport to blood circulation, amino acids, fatty acids and glucose are stored in their defined destination in the body. Fat is stored in adipose tissue and protein in muscles and glucose in liver and muscles. The storage capacity of fat is as per the kcal is 1, 40, 000 and as per the weight 15.5 kg in body. Amino acid storage as per the kcal is 24, 000 and as per weight is 12 kg. Glucose storage as per kcal 800 - 1000 and as per the weight is 250 gm – 400 gm. So any amount of fat and amino acid taken by body can be easily adjusted to their respective storage areas with negligible alteration of storage capacity. But as glocuse storage capacity is 250 gm in normal condition, an average human being of 70 kg weight and 100 mg/dl fasting sugar and in between the meals and 4 hours after meal the glucose amount in blood circulation will be 5 gm only and this change varies as per their weight. So after taking a meal which contains approx.90 gm of glucose the total glucose amount in blood circulation will be 95 (90+ 5) gm and this 95 gm should have to come to basal level to 5 gm within 2 hours of taking a meal (prandial sugar). At best and maximum 3 - 4 hours of post prandial period it should come to basal state i. e 5 gm.

As the glucose is transported to liver according to their concentration gradient in portal circulation and this process is independent of insulin and transport depends upon glut 2 transporter. The glucose taken by liver is deposited as glycogen dependent on availability of insulin and highest capacity of liver for glycogen storage is 100 gm and it is already 80 % fill up before a meal period, so after glucose absorbed in liver the rest 20 % will be filled up as glycogen in the liver and i. e within 2 hours (prandial period), the rest glucose which is not stored as glycogen is immediately transformed to be deposited ectopic saturated fats in liver. Ectopic fatty acids in liver are 16 carbon saturated palmitic fatty acids which is highly pro inflammatory with different cytokines, leucokines, chemokines etc. It attracts macrophages & T cells, lymphocytes by producing IL1, IL6, IL17 etc and the ectopic fats in the liver transferred to a chronic inflammatory cells producing organ by activating immunological process by macrophages and T cells and different hormonal imbalance by adipokines and these are the main culprit to produce metabolism disorder in the body. All these ectopic fats lead to steatosis, steato hepatitis, cirrhosis and carcinoma of liver and also main culprit to produce production of small particle LDL by the liver which when enter in the blood circulation by VLDL deposited in the endothelial tissue of arteries and arterioles etc and thereby dearranging their structure and production of chemokines signaling factors, they attract macrophages, T cells etc thereby producing artherosclerosis of arteries and arterioles. So it is necessary to manage glucose homeostasis at the time of origin in GI tract and during the time of blood circulation after the meal (prandial period) to avoid ectopic fats deposit in liver and pancreas to avoid metabolic disorder.

Management - 1

In the GI tract we can limit glucose absorption from GI tract to enterocytes in small intestines by blocking – inhibiting SGLT - 1 transporter which is 90% responsible for glucose transport from intestinal lumen to enterocyte. SGLT - 1 is expressed in luminal border of GI tract and a non absorbale SGLT - 1 inhibitor can be given with meals to prevent glucose absorption from small intestine to enterocyte without producing any adverse effect as it is not absorbed in blood stream.

Management - II
From enterocyte glucose will be transported (approx.90%) to blood circulation by GLUT - 2 transporter present in baso lateral side of enterocyte and this GLUC - 2 transporter can be inhibited or blunted for a short time say 10 - 15 mins by giving short half life GLUC - 2 inhibitor during the meal time to prevent glucose absorption from enterocyte to circulation.

Pleotrophic effect of GLUT - 2 inhibitor
By inhibiting GLUT - 2 transporter, the glucose which is taken by liver and pancreas are prohibited to enter the hepatocytes and pancreatic cells so there will be no ectopic fat deposition in liver and pancreas and thereby their function will be well maintained and there will be no mitochondrial stress effect & thereby preventing metabolism disorder and moreover due to its action on B cells of pancreas, insulin secretion will be reduced and thereby encouraging B oxidation in cellular level, as it will maintain mitochondrial health by reducing stress.

Management III (exercise after meal - prandial exercise)
Prandial glucose can be managed by calorie burn process thereby to reduce blood glucose level after meal, moderate muscles exercise is required which will utilize 50% max glucose capacity thereby producing energy souce 50 % from glucose and 50% from B oxidation of fatty acids, but if we will improve exercise strength to use 75 % of maximum utilization then 90 % energy will be from glucose source and negligible energy from fatty acids so daily post meal exercise of 10 - 15 mins to reduce prandial glucose level thereby reducing ectopic fats in liver and pancreas. As the normal body use 2000 kcal per day & the source of calorie utilization in a normal condition is 90 % from the fatty acid B oxidation say 1800 kcal from fats and total amount of fats required for this used is 200 gm to get this energy & 200 kcal is used from glucose source i. e 50 gm maximum from glucose source, we are taking a diet as glucose in a meal more than 90 gm, in total a day we are consuming more than 300 gm of glucose so by burning 50 gm glucose all the rest 250 gm use as saturated fat production of 16 carbon palmitic acid and this 16 carbon saturated fatty acid is the main substrate for B oxidation and also its accumulation causes pro inflammatory, chronic inflammatory, auto immune metabolic disorders by producing different cytokines, interlukenis, adipokines etc.

Pleotrophic Effect of Prandial Exercise
Prandial exercise will increase abdominal pressure thereby reducing portal circulation to liver and due to increased abdominal pressure GI lumen diameter will be decreased thereby reducing nutritional absorption, intotal reducing the prandial calorie intake.

Management IV (Pre Prandial Exercise to Improve Prandial Glucose Homeostasis)
Preprandial exercise before 15 - 30 mins of a taking meal can burn glucose source of energy by doing medium to intensive exercise using 50 - 75% maximum oxygen capacity and by doing this it will improve the glycogen storage capacity of the liver & muscles as glucose will be utilized from glycogen store of liver & muscles thereby increasing its glycogen storage capacity in liver & muscles preprandially.

Fat Management
Fat is the main source of cell structure producing its membrane structure by phospholipids, cholesterol & fat is the main source of steroid hormone production including vitamin D & main source of energy in the human body because 90 % energy derived from fat source, only 10% from glucose source, 80 % of saturated fat is used in B oxidation and most of this saturated fats derived from carbohydrate source so there is no need of taking more saturated fats in the food so food should contain more unsaturated fatty acid from monosaturated and ploysaturated fatty acid origin & to avoid pro inflammatory & chronic inflammatory disorder & metabolic disorder of the body omega 3 ploy unsaturated fatty acid should be taken in comparison to omega 6 fatty acids which produces all pro inflammation, chronic inflammation & metabolic disorder so nutrition should be managed accordingly.

The best ideal fat management in the body & muscles is: -
Muscles Fat content: -
1.5 % of total body weight.
1.5 to 5 % is overweight
i. e BMI from 23 - 29.5.

More than 5 % of the total body weight is obese i. e BMI more than 30%.

Body fat Content	
For Males	9 - 15
For Females	16 - 24
Average Fat	
Males	16 - 25
Females	20 - 30
Obese	
Males	>25
Females	>35
Distribution of fats	
Males	Abdominal
Females	Thigh & buttock

Conclusion
From ancient period all placental delivered animals including human were always in a running state in their work due to fear from environmental factors such as animal, insects and etc. Due to their running state they utilize 50 - 90 % of their maximum oxygen utilized capacity & thereby used their energy source from sugar i. e 80% and 20% from fatty acids. Accordingly their genes are made for sugar utilization & they store glycogen and the rest remaining sugar is converted to fat so their main source of energy was on sugar based foods as per that present environmental condition.

At present, we hardly use our calorie requirements from sugar. Rather 90% used from fat reserves i. e aerobic B oxidation in mitochondria so for better B oxidation a low energy condition with low insulin & high oxygen level required. For e. g a person requiring 2000kcal energy per day needs 1800 kcal from fat and 200 kcal from sugar per day. For getting calories 200 kcal from sugar we require 50 gm of glucose per day. In a normal meal at present we are taking 90 gms of sugar, and in 3 meals per day we are taking 270 gms of sugar daily and our total sugar requirement is only 50 gm to give necessity calorie & considering a standard meal

contains 90 gm sugar we require only 15 gms of sugar from each meal out of 90 gms in a meal so 75 gm of sugar should go to dustbin in the prandial management of sugar as they produce ectopic fats & other metabolism disorders so prandial management of sugar to be used to limit as 15 gm only in a meal. For that inhibitors are required to blunt the action SLGT 1, glut 2, preprandial & prandial exercise etc to be required for creating proper environment to meet aerobic B oxidation and to keep low level of insulin, low level energy & high level of oxygen in mitochondria.

Calorie or Energy Homeostasis in Post Prandial Period and in between Two Meals:

After amino acid fats and sugar enter to blood circulation. The master energy regulator in the body is hypothalamic arcuate nucleus. It contains at least two crucial populations of neurons to project to second order target including para ventricular nucleus and these neuronal activity are within few seconds and this manage orexigenoic sensitivity and anorexigenic in a co - ordinated manner. It regulate the peripheral organs involve in the central of nutrition storage and it is independent of food intake status. Insulin, GLP - 1, VIP and Leptin receptors in arc urate nucleus produce negative effect and there by producing less calories and utilizing more calories by activating PONC and de activating AGOUTIC response protein neurons. Here we give the example of the clomiphene on oestrogene receptor of hypothalamus in CNS, which by attaching to the ostrogene receptor produce agonist effect of ostrogene on hypothalamus sensing as if more Ostrogene present in the blood. And this clomiphene has no remarkable effect on peripheral ostrogene receptors such as ovary, Uterus and Mammary gland etc. So maintaining normal ostrogene activity in the peripheral without any agonist effect. Likewise a substrait of chemical or neurological origin (like clomiphene on hypothalamus) may be targated or developed to act only on Aarcurte GLP1 or insulin receptor etc without no remarkable effect on peripheral or other CNS GLP1R or Insulin R to produce energy homeostatis in the body in future.

N. B. – By pleotropic effect of insulin receptor in Arcurate nucleus it will immediately activate in few seconds brown fat (BAT). By regulating myostatins and there by immediately reduce insulin resistance in the body.

Conclusion

In conclusion, managing postprandial glucose and overall caloric balance is essential to preventing metabolic disorders like fatty liver disease and atherosclerosis. Strategies such as inhibiting glucose transporters in the gastrointestinal tract, along with targeted exercises, can effectively control glucose levels and reduce ectopic fat accumulation in the liver and pancreas. These methods help maintain metabolic health and prevent the onset of chronic conditions by promoting better energy utilization. It is crucial to focus on early interventions, starting from nutrient absorption, to sustain longterm metabolic stability.