International Journal of Science and Research (IJSR)
ISSN: 2319-7064
Impact Factor 2024: 7.101

Pre and Post Prandial Moderate Intensive Exercise, Prior Pre-Prandial Nutrition, Brain Hypothalamus Arcuate Nucleus are Strategies Management of Post Prandial Glucose with Calorie for Prevention and Reduce Progression of Metabolic Disorder

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Abstract: ***Pre and post prandial moderate intensive exercise, prior pre - prandial nutrition, brain hypothalamus arcuate nucleus 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 & 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 post meal 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: Pre and post prandial moderate intensive exercise, 3 doses of prior pre - prandial nutrition (Break - fast, Lunch and Dinner), brain hypothalamus arcuate nucleus, Metabolic homeostasis, calorie, postprandial glucose, metabolic disorders, ectopic fats, glucose transporters, SGLT1, Glut 2. GLP1R, Insulin RA, GIPRA.

1. Introduction

Calorie or energy management of the body is most essential for homeostasis 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 are transferred 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 amino acid in protein synthesis and glucose in liver and muscles. The storage capacity [1] 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 taken by body can be easily adjusted to their respective storage areas with negligible alteration of storage capacity. But as glucose 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, steatohepatitis, 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 disarranging their structure and production of chemokines signalling factors, they attract macrophages, T cells etc.

thereby producing atherosclerosis 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.

NB - As negligible source of energy from amino acid metabolism required for calorie homeostasis only 0.08 to 1.2 gm / kg of body weight is required for body maintenance purpose & amino acids are building block of protein which are essential for countless biological Process and amino acids are reusable in the body by cycling process & minor amounts of amino acids are metabolised to nitrogen products like creatinine, uric acid & urea etc.

Management - 1

Better Nutritional management with pulse nutritional therapy with a combination of protein and mono saturated fatty acid in the diet & minimum 30 mins prior or before to each pre - prandial $_2$ meal counting approx.3 times of pre - prandial pulse nutritional therapy, minimum 30 min. before each 3 major meals (breakfast, lunch and dinner).

For example, pre - prandial pulse nutrition therapy should contain the type of protein use in the diet. Suppose we select pulses plant protein (moong dal, arhar dal, red gram, green gram etc) the percentage of protein in these are approximately 25%. If each time, we take 20 grams any of these pulses it will give you only 5 grams of available protein as per their 20 - gram weight. So, taking 2 grams of all pulses plant proteins in three pulses pre - prandial nutrition (breakfast, lunch and dinner). A total 15 gram of protein will be available in a day from these three - pulse pre - prandial nutrition. Then balance 45 to 60 gram of protein should be collected from dietary source or adding additional protein such as whole milk cheese, paneer, egg etc to these three pulse pre - prandial nutrition therapy. Thereby produce better health benefit. These three - pulse nutritional therapy should be differently prepared for better health benefit.

1) Pulse breakfast nutrition therapy

Total 20 grams moong and red gram seeds should be germinated in night and that sprouts should be taken with 35 gram of whole milk cheese.

2) Pulse lunch pre - prandial nutritional therapy

Arhar dal or moong dal of 20 grams and 35 grams of milk paneer.

3. pulse dinner pre - prandial nutrition therapy

20 grams of red gram chhatua and 2 eggs without yolk

NB: Water or liquid intake in the pre - prandial nutrition and before meal will delay the cephalic phase of priming the digestive system i. e. in normal condition after taking a meal it's liquid portion with carbohydrate and protein enter the small intestine in the Bolus form for a period of 20 min. after food intake. Then food in stomach becomes pasty and semi solid and they pass in a linear row to the small intestine. After these cephalic phase of 20 min. CCK from neuro - endocrine cells and pepsin in the stomach are produce and they give rise to satiety and also reduce hunger before taking original meal and thereby decrees nutrition calorie intake in breakfast, lunch and dinner.

3) Prior pulse pre - prandial monounsaturated nutrition therapy in breakfast, lunch and dinner per day.

EXAMPLE - 10 TO 15 ml mustard oil adding to the food will give rise to 20 to 40 grams of triglyceride in breakfast, lunch and dinner pulse nutrition therapy. Mustard oil, olive oil, and peanut oil contains 60% monounsaturated fatty acid, 21% polyunsaturated fatty acid and 12% saturated fatty acid.

Our body required 200 grams of fatty acid daily producing 800 kilocalories out of 2000 required calorie per day for a normal human.

Total 40 grams fats are available from 3 breakfast, lunch and dinner pulse nutritional therapy, and rest 160 grams should be from the dietary fats and converted saturated fat from glucose source.

NB:

1) Addition of mustard oil direct to suitable fruit and salad etc. will improve its taste and flavor.
2) Fried food should be prepared from either mustard oil of peanut oil as their smoking point is the higher than other oil.
3) mustard peanut and the olive oil are with same amount mono saturated fatty acids range from 50% to 60% and thereby producing equal health benefit to heart, mitochondria and beta - oxidation so higher metabolic benefit.
4) All oil should be selected from cold processed preparation rather than refined oil to produce more metabolic benefit

Pleo tropic effects of monounsaturated pulse nutritional therapy

a) Reduce gastric motility
b) Increase GIP, GLP1 AND CCK production
c) Produce satiety and reduce hunger
d) Reduce insulin resistance in periphery
e) Decreases triglyceride and LDL level in blood
f) Improve diabetes profile
g) It is a better substrate for mitochondrial respiration, function and health conditions better than poly unsaturated and saturated fatty acid.
h) Reduces inflammation and is an immune modulator

Pleotropic effects of pulse nutritional pre - prandial therapy with protein and monounsaturated fatty acids 30 min prior to meal intake

Will prime digestive system in the following ways:

1) It will increase secretion of GIP and GLP1 as protein & fats are more potent than carbohydrates for incretin secretion such as GLP1 and GIP.
2) GLP1 improves glucose dependent insulin secretion and reduce glucagon secretion in the pancreases.
3) GIP improves fat metabolism in adipose tissue by improving & proliferating GIP receptors in adipose tissue for fat metabolism purpose and there is no GLP1 receptors in adipose tissue.
4) GIP encourage glucagon secretion in pancreatic cells and thereby creating an atmosphere of low energy environment with thermogenic heat production and less production of ATP in electron transfer chain of

mitochondria and hence reduce mitochondrial oxidative stress and increase its function.

5) 30 mins before taking protein and mono saturated fatty acid combination will increase HCL secretion thereby encouraging more cleaving of long chain protein to short chain amino acids protein fractions in the stomach.
6) Fatty acids prime B cells of pancreases to produce more secretion of insulin if it is required in post prandial glucose homeostasis.
7) Fatty acids also prime CCK secretion in producing more bales, pancreatic exocrine secretion and HCL secretion there by enhancing digestive system activities.
8) GIP receptor agonist GIPR is a better substrate for improving aerobic fatty acid B oxidation and improvement of BAT proliferation & improve thermogenic heat production & reducing ectopic fat deposit in different organs thereby reducing chronic inflammatory immune reactions.
9) Monosaturated Fatty acid is a better substrate for aerobic B oxidation than other saturated & ploy unsaturated fatty acid so increasing mono saturated fatty acid consumptions will help for improvised aerobic B oxidation & mitochondrial oxidative stress & improve in total mitochondrial function.

Management - 2

In the GI tract we can limit glucose absorption from GI tract to enterocytes in small intestines by blocking – inhibiting SGLT - 1_3 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 - absorbable 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 - 3

From enterocyte glucose will be transported (approx.90%) to blood circulation by GLUT - 2_4 transporter present in baso lateral side of enterocyte and this GLUT - 2 transporter can be inhibited or blunted for a short time say 10 - 15 mins by giving short half - life GLUT - 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 as aerobic oxidation is healthy for mitochondrial respiration and function and moreover GLUT - 2 transport glucose from renal cells to glomerular capillaries. By blocking or GLUT - 2 action on renal cells will enhance smooth passage of glucose in urine and thereby reduced insulin label, and encouraging B oxidation and mitochondrial health and higher level of insulin required for muscular deposit of glucose whereas normal level of insulin is sufficient to deposit glucose in adipose tissues.

Management 4 (exercise after meal - Post prandial exercise$_6$)

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 source 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, interleukins, adipokines etc. and activating innate and adoptive immunity. In this respect WHO Recommended total sugar from diet as well as added sugar should be 10% of total calories used daily and reducing it to 5% of total calories will give more health benefit $_{11}$ (WHO RECOMMENDATION).

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, in total reducing the prandial calorie intake.

Management 5 (Pre Prandial Exercise to Improve Prandial Glucose Homeostasis)

Pre - prandial exercise$_5$ 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. But during night time for a period of approx.8 hours glycogen stored in liver is significantly reduced as glucose required for resting basal metabolic rate and for brain$_{10}$ is derived from glycogenolysis and gluconeogenesis in liver at night, so pre - prandial breakfast exercise should be replaced by postprandial breakfast exercise for better health benefits.

NB - Pre - prandial exercise is beneficial in all meal except breakfast meal as there is already reduced glycogen storage in liver at night.

Fat Management[7]

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 rather mono saturated fat is better substitute than saturated fat for mitochondrial

Respiration and thereby favourable B oxidation in mitochondria[9]. And it also promote mitochondrial formation and function. 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[8]: -

For Males	for Females
9 - 15	16 - 24

Average Fat:

Males	Females
16 - 25	20 - 30

Obese:

Male	Female
>25	> 35

Distribution of fats:

Male	Female
Abdominal	Thigh & buttock

2. 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 long - term metabolic stability.

NB -

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, pre - prandial & 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, GIP and Leptin receptors in arc urate nucleus produce negative effect and there by producing less calories and utilizing more calories by activating POMC and de activating AGOUTIC response protein neurons. Here we give the example of the clomiphene on estrogen receptor of hypothalamus in CNS, which by attaching to the estrogen receptor produce agonist effect of estrogen on hypothalamus sensing as if more estrogen present in the blood. And this clomiphene has no remarkable effect on peripheral estrogen receptors such as ovary, Uterus and Mammary gland etc. So maintaining normal estrogen activity in the peripheral without any agonist effect. Likewise a sub strait of chemical or neurological origin (like clomiphene on hypothalamus) may be targeted or developed to act only on arcuate GLP1 or insulin GIP LEPTIN receptor etc. with their agonist effect on hypothalamus without no remarkable effect on peripheral or other CNS GLP1R or Insulin R to produce energy homeostasis in the body in future.

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

reduce insulin resistance in the body and irrespective to nutritional status.

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