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Lipid and Apolipoprotein Metabolic Homeostasis by Scientifically Targeting Interventional Therapy with Quality & Quantity of HDLc & Chylomicron Produced in GI Tract & HDLc and VLDLc Produced in Liver Cells & Giving Importance to Daily Post Prandial Exercise in Each Meal for Best Nutritional Management of Dietary Food to Avoid Ectopic Fat Deposits from Prandial Sugar

Dr. Bansi Saboo[1], Dr. L Srenevas Murthy[2], Dr. Shilpa Varma[3], Dr. Mridul Bera[4], Dr. Mahendra Prasad Tripathy[5], Dr. Bijay Ketan Dash[6], Dr. Bharat Panigrahi[7], Dr. Rabindra Ku. Nayak[8], Dr. Pratap Ku. Pradhan[9], Dr. Susil Ku. Mohanty[10]

Abstract: ***Lipid, the mother of all metabolic and immunological disorders, even sugar without a part to lipid fats or transfer to 14 and 16 saturated fatty acids have limited or absolute no effect on metabolic or immunological disorders. Sugar related disorders arise only when cells and organs deposits advanced glycation end products inside the cell or organs making it dysfunctional in due course of time and irreversible accumulation of advance glycation end products of lipid and amino acids causing permanent deposits inside cell and producing oxidative stress and ultimate death of the cell. Sugar may periodically produce osmolar effect due to hyperglycemia and Ketoacidosis due to insufficient supply of insulin in blood. Post prandial nutritional containing esters of fatty acids in triglycerides and LDLc play a major part in management of metabolic diseases according to their saturated and unsaturated (both mono and poly) fatty acid contents producing different types of metabolic disorders. And play a vital role in maintaining metabolic homeostasis and 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 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: 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 insulin RA, GIPRA, triglyceride LDLc.

1. Introduction

Brief on HDLc

After APOA-1 secreted from liver cells & GI cells collect free Cholesterol and phospholipids from nearby cells membrane including Gastro intestional, liver & peripheral cell membranes forming NASCENT HDLc as free cholesterol is toxic to every cell, it should be converted to cholesteryl esters by LCAT in HDLc. These HDLc esters are collected from cell membranes they are mostly poly unsaturated fatty acids from phospholipids bilayer and these HDLc cholesteryl esters are taken by SR-B1 receptors of peripheral and liver cells without endocytosis of HDLc with APOA-1. So HDLc remain intact with APOA-1. But otherwise VLDLc remnants (IDLc & LDLc) are endocytosed totally by LDL receptor & LRP receptor. cholesteryl esters taken by SR-B1 are endocytosed to free cholesterol for hormone formations in steroidgenesis cell and PUFA in cholesteryl esters are incorporated to all cell membrane formation, so justifying as good cholesterol and also cholesteryl esters supplied by HDLc of liver cells are mostly poly unsaturated fatty acids and these are essential fatty acids not formed in the body and are necessary for cell membrane formation and cell signalling system. As HDLc with APOA-1 never endocytosed to cell and always remain in blood stream it is not harmful to cell and tissue and benefits by collecting excess free cholesterol from Liver, GI as well as peripheral cells and at the end disposing its content of cholesterol to liver for excretion in bile as bile acids, salt and free cholesterol, justifying the HDLc as good cholesterol.

Non energy producing lipid known as free cholesterol collected from dietary source 300 mg from biles 1000 mg and from GI mucosal source 300 mg in total to be about 1600 mg. And out of this 1600 mg only 800 mg (50%) from GI tract will be absorbed to blood stream by chylomicron and a newly formed APOA-1 lipoprotein converted to HDLc and collect free cholesterol and phospholipid from cell membrane of enterocyte mediated by ABCAI and ABCG-I to produce Nascent HDLc from GI source to supply cholesterol to periphery steroidogenesis cells to produce

steroid hormones and also to other peripheral cells to form their cell membrane structures and this BI-layer of phospholipid which is only formed by PUFA (Omega 3 and Omega 6) and this is delivered to all cells by HDLc mediated by SR-B1 receptors present in liver cells. Steroidogenesis cells and all peripheral cells and in all these processes APOA-1 carrying HDLc remain intact without degradation. So, they are not endocytosed by the above cells and only cholesteryl esters from HDLc are endocytosed.

Brief on VLDLc:

APOB transport container carries triglyceride and cholesteryl esters in liver to form VLDLc and APOB in G.I. tract cell collect triglyceride and cholesterol esters designated as chylomicron. Body uses mostly 80% of energy supplied from Fatty acid. During Feeding State Chylomicrons are the main source of energy supplied to Muscles, Heart, Kidney, Lungs contain all types of dietary Fats, Saturated and Unsaturated Fatty Acid. After supplying to Muscle, Heart etc it is converted to storage Fat in Adipose tissue to supply energy in future use.

Chylomicron's triglycerides can be directly transferred to VLDLc and VLDLc remnants (IDLc & LDLc) facilitate by natural protein exchange in exchange of cholesteryl esters from VLDLc. Whereas VLDLc triglycerides are exchanged to HDLc in exchange of HDLc cholesteryl esters facilitated by CETP only. After losing 60% of triglycerides and gaining more than 40% of cholesteryl ester of chylomicron and VLDLc remnants became dysfunctional and endocytosed by LDLc and LRP receptors in liver cells, with the aim to be excreted in bile as bile acids and free cholesterol.

VLDLc Triglycerides are formed in liver cells at rough and smooth endo plasmic reticulum and there after loaded to APOB-100 which is transporter and structural protein of VLDLc along with cholesteryl esters formed by ACAT in liver cells. As these fatty acids of cholesteryl esters are mostly from adipose tissues, they contain mostly saturated fatty acid which life is more than cholesteryl ester of HDLc.

N.B:- VLDLc received triglycerides from chylomicron and exchanged cholesteryl ester to chylomicron mediated by natural protein exchange. This triglyceride esters are MUFA, PUFA and saturity fatty acid of dietary source. VLDLc loosing cholesteryl ester by transfer to chylomicron and in exchange of triglyceride from chylomicron. Make it more healthy, more functional and produced less LDLc remnants. Thereby LDLc level is reduced in blood circulation.

VLDLc received cholesteryl ester from HDLc in exchange of triglycerides transfer to HDLc. Mediated via CETP, will make both the HDLc and VLDLc dysfunctional impaired and unhealthy producing more LDLc remnants to blood.

Fats (Fatty Acid and Cholesterol):

After fatty acids are collected from liver and dietary fats in GI tract, 3 numbers of Fatty acids are to be esterified with glycerol to produce triglyceride and 1 fatty acids with free cholesterol to produce cholesteryl esters respectfully. Also, free cholesterol collected by HDLc from periphery etc. to be esterified to make it more hydrophobic and they will go to the centre of the HDLc making membrane of HDLc free to accept more cholesterol from periphery etc. and thereby improve HDLc functions and capacity. Also, HDLc collect free cholesterol from macrophage to avoid foam cell formation and HDLc take all these esterified cholesterols to LIVER for excretion in bile, thereby reducing cholesterol level in blood. Triglycerides are used to supply fats for energy production in different cells and organs. And also, this triglyceride are stored in adipose tissues for future energy. Cholesterols carried by VLDLc from Liver and Chylomicron from GI tract are delivered to their destination cells such as steroidogenesis cells to produce steroid hormones and gonadal cells to produce sex hormones also to the skin to produce vitamin D. And in cell it gives structure and also it produces bile acid for digestion.

NB: In room temperature saturated fatty acids take longer time, while mono-unsaturated fatty acids take moderate time and poly-unsaturated fatty acids take shorter time. Such types of disintegration/break down are also manifested in the body.

Triglyceride in blood are esters of saturated unsaturated (mono and poly) fatty acid, and the saturated fatty acids are from 4 to 22 carbon straight chain producing sort chain (4to8) healthy for gut micro biota immunity and health. Medium chain fatty acid 12 to18 carbon where 14 and 16 are atherogenic and 12 and 18 are natural. Triglyceride in blood containing esters of monosaturated fatty acids are better substrate for mitochondrial respiration with cellular health benefit by reducing oxidative stress and cell apoptosis etc.

Triglyceride in blood containing polyunsaturated such as omega 3 are of a linolenic acid, EPA and DHA are most important in cellular functions: linolenic acid derived from plant source converted to EPA and DHA also EPA can be converted to DHA. EPA acts mostly peripherally by improving fatty acid oxidation transportation and activating PPARA receptors and thereby improving fatty acid metabolism and triglyceride turnover. DHA act on CNS producing good memories and improve cognitive Alzheimer's and other functions like improving child brain growth and development in pregnancy and childhood.

Triglycerides containing omega 6 produce arachidonic acid in cell membrane. They produce inflammatory Interleukins and cytokines for acute and chronic inflammation. Cholesterol from Dietary sources and from endogenous sources produced by liver are used in adrenal glands and reproductive organs. But majority are from liver source. Cholesterol from GI tract transported in blood by HDLc. Free Cholesterol produced in liver is transported by HDLc. As free cholesterol is toxic to intracellular structures producing their dysfunction, structural damage, oxidative stress. So, the cholesterol stored in the cells and cholesterol transported in blood are to be esterified to cholesteryl ester for better sustenance and by that reduce toxicity of free cholesterol. Due to their hydrophobic nature they migrate to core portion of HDLc so the membrane will be free from free cholesterol and more cholesterol can be collected from periphery to liver by HDLc.

Cholesterol transported to the liver via VLDLc and HDLc are excreted in bile as free cholesterol and bile acid or used to formed new VLDLc in liver. To form new VLDLc in liver

cell and these Cholesteryl esters may be from saturated unsaturated (mono and poly fatty acids) and work according to their fatty acid quality contains in the cholesteryl ester and are delivered to reproductive organs, adrenal glands including all steroidogenesis cells as cholesteryl esters from monounsaturated acid received by cell is easily taken by mitochondria to produce energy. Cholesterol of saturated fatty acid is difficult to be metabolise and their life span is more than any other cholesteryl ester. They are often impaired and oxygenated and glycosylated etc. Macrophages in endothelial cells cannot take native LDLc but when this saturated small LDLc are impaired or oxygenated they are easily taken by the macrophage. The impaired cholesterol cannot be taken back by HDLc to Liver. So, this saturated cholesterol ester is deposited in macrophage forming a large number of intra cholesteryl esters and there by producing all inflammatory activities and macrophage apoptosis and necrosis producing plaque, fibroatheroma foam cell etc.

Native LDLc are not atherogenic and cannot be taken by macrophage in endothelial cells of arteries and arterioles. But impaired or disfunction LDLc carrying cholesteryl ester along with APOB-100 bind to proteoglycan producing auto immune pro-inflammatory antigen complex. And this antigen complex is suitable to be taken by scavenger receptors of macrophages. And all these activate innate immunity cell such as macrophage, neutrophil, dendrite cells etc. and also activate adaptive immunity cell such as CD4T subsets CDTH1 & TH2, TH17 etc. producing interferon IL1, IL6, IL17, TNFa etc. and changing endothelial cells of arteries and forming it a auto immune inflammatory organs by activating M1 type of macrophages and this process continue for a long period from childhood to old age producing atherosclerosis. But it can be checked by CDTH2 and T-Reg cells by producing IL4, IL10 and IL13 thereby anti-inflammatory and antiatherogenic M2 macrophage. Atherosclerosis fibroatheroma and plaque by accumulating number of cholesteryl esters and other toxic materials producing necrosis degeneration and apoptosis of surrounding cells. It promotes lipid deposit in the surrounding area thereby transformation of macrophages to phenotype M1.

N.B.- High LDLc and high triglycerides in blood are not atherogenic and their atherogenic effects are to be measured in respect to their esters of saturated and unsaturated fatty acids contains producing bad or good effect.

Triglycerides and cholesteryl esters containing unsaturated (mono and poly) fatty acid are easily de-esterified after reaching their target cell or organ producing free cholesterol, free fatty acid and free glycerol but cholesteryl esters containing saturated fatty acid are not easily de-esterified and their long survival period and long-time exposure to epigenetic factors made them impaired and dysfunctional LDLc. This impaired LDLc binds with proteoglycan arterial wall transforms to an antigen complex which activates innate and adaptive immunity. Fat from diet source is better than liver source as they contain all type of saturated and unsaturated fatty acid. But in liver hepatocytes produce mostly 14,16 carbon atherogenic fatty acids from sugar and amino acid. Triglyceride containing esters of mono, and poly unsaturated fatty acids are good for health benefits. HDLc always contains cholesteryl esters of mono, and poly unsaturated fatty acids as because they are collected from cell membrane and good for health benefits and also prevent atherosclerosis. But LDLc containing saturated fatty acid are bad for health benefit and unsaturated fatty acids are non-atherogenic. LDLc receptor recycle Hundred times in normal conditions to transport cholesterol into liver cells but binding PCSK9 enzymes at the receptor will make a complex which cannot be recycled but will be degraded in lysosomes by lysosomal acid lipase. And this PCSK9 enzyme can be inhibited to bind to LDLc receptor by monoclonal antibodies. And PCSK9 enzyme production can be reduced by inhibiting transcription of MRNA of PCSK9 by activation of MiRN in nucleus. But both these two methods will help in reducing the numbers and binding capacity of PCSK9. But it has no effect on the production of LDLc receptor or production of cholesterol in liver and only for the purpose of to complete normal recycling that is hundred times of a LDLc receptor.

Cholesterol Metabolism:

Free cholesterol is toxic to cell and in order to make it to non-toxic and more stable it is converted to cholesteryl ester by LCAT or ACAT. Esterifying a fatty acid from the phospholipid present in cell. APOA-1 is the structural and potent active catalyst for cholesterol metabolism and APOA-1 take up cholesterol and Phospholipid from its production sides such as liver, intestine, macrophages, fibroblast etc. and delivery to targeted cholesterol receiving organs or cells and thereafter at the end to liver for excretion in bile. Whereas APOB-100 & 48 are potent and backbone of structural protein for VLDLc and chylomicron which carry fats soluble vitamins phospholipids cholesterol etc. for delivery to destinated organs or cell. APOE is the delivery boy of helping delivery of these product fat cholesterol etc from VLDLc and HDLc and work as a ligand to respective receptors of designated organ/cells in the body.

Function of HDLc with APOA-1 on cholesterol metabolism:

APOA-1 is the structural protein of HDLc and after its formation from GI and liver cellss endoplasmic reticulum, it collects cholesterol and phospholipids from that cell membrane by the process cholesterol efflux mediated by ABCA1 and ABCG1 present in that cell.

- It produces inert nontoxic cholesterol ester by L-CAT in HDLc collecting a fatty acid from the phospholipid in that cell.
- It transfers cholesteryl ester to VLDLc, IDLc, LDLc etc. of containing APOB-100 and APOB-48

To transfer the cholesterol to be used in targeted required cells as well as for excretion in liver for recycled or to excreted in bile.

HDLc with the APOA-1 distribute cholesterol to different peripheral cells such as steroidgenesis cells, adrenal glands, testis, ovary etc. by SR-B1 receptors. Also, finally all the cholesterols delivered to liver for excretion in bile. APOA-1 is not used or endocytosed by these cells, and it remains intact and for this APOE mediated for the transport and endocytosis of cholesterol.

In arterial wall the deposited LDLc binding to proteoglycans form a antigenic complex to produce innate immunity response by activating macrophages and other adaptive immunity to produce atherosclerosis in that particular wall.

Function of VLDLc with APOB-100:

VLDLc fat transported to all cells, organs and, tissues to supply energy from fat sources and supply of VLDLc fat depends on available triglycerides percentage in volume with respect to total volume in VLDLc. VLDLc triglycerides containing more than 15% of VLDLc volume can transfer fatty acid to different parts. Here only APOE and not APOB-100 take part in transfer and improving fatty acid metabolites transcription factors for VLDLc receptors, lipo-protein lipase receptors, PPRA receptors and thereby improving uptake of fatty acids in adipose tissues, muscle tissue and other cells.

VLDLc carrying less than 15% triglycerides in volume are called LDLc and here APPOB-100 is main ligand with APOE to present total VLDLc particle to LDLc receptor in liver for endocytosis and make them different products such as amino acids, fatty acids, cholesterol to end the VLDLc metabolism. 70-80% of cholesterol is produced in liver and also 70-80% of cholesterol is taken by liver LDLc receptors to be excreted in bile or for recycling. LDLc receptors in cell membrane of hepatocytes are dependent on free cholesterol level available in cytosol of liver cells number of receptors in hepatocytes membrane are inversely proportional to the free cholesterol available in cytosol of hepatocytes i.e., free cholesterol reduced in hepatocytes will increase receptors and vice versa. By reducing free cholesterol in cytosol and increasing receptors will reduce cholesterol in the blood circulation.

Cholesterol Homeostasis or Management:

1) Cholesterol can be managed at the site of their production internally in the liver and externally at GI tract from dietary source and bile source. Bile source 1000 mg and dietary source 300 mg total in the range of 1000- 1500 mg can be blocked by ezetimibe by blocking cholesterol absorbing receptor in GI Tract and bile acid recycling from the GI Tract to portal circulation can be inhibited by cholestyramine. There by giving rise to more production of bile by liver. Above procedure will reduce the cholesterol level in liver cells and enhancing cholesterol receptor numbers in liver cells and decrease the blood level of cholesterol for improving bile production.
2) Utilisation of cholesterol in gonadal and steroidogenesis cells and production of Vitamin D and cellular structure etc. are natural way.
3) Cholesterol formation in liver can be inhibited by statins by inhibiting HMG COA reductase in cytosol of liver cells and thereby reducing the availability of free cholesterol in hepatocytes there by increasing more LDLc receptor and more collection of cholesterol from blood reducing blood cholesterol.

N.B.: Statin and Ezetimibe together produce catalytic effect on reduction of blood cholesterol and increase cholesterol excretion in bile and thereby improving quality, numbers, and function of HDLc to collect cholesterol from liver and periphery cells and transfer to liver for excretion in bile or for recirculation. Improved function of HDLc will decrease the transformation of cholesterol esters from HDLc to VLDLc via CETP enzyme and these VLDLc with low content of cholesteryl esters are easy substrate/ligand for liver cells to be clear and effective excretion in bile in the form of cholesterol and bile acid. As the combination have no effect on fat absorption in GI tract and act only to reduce cholesterol level in blood, so they can be used for longer period if indicated.

Ezetimibe and cholestyramine are catalysts for enhancing natural process of cholesterol excretion in GI tract and adverse to fatty acid absorption. So, they cannot be used for longer periods.

Statin and PCSK9 are used to reduce cholesterol production and increase LDLc receptors life cycle respectively by inhibiting or promoting some enzymes involved in cholesterol production. But their intermediate metabolites products may be ligands for increase or decrease of some cellular products. Cholestyramine should not be used for long period as it adversely reduced fat absorption in GI tract.

Management- 1:

Better Nutritional management with pulse nutrition (Prior pre-prandial and prandial meal) therapy with a combination of protein and mono saturated fatty acid in the diet & minimum 30 mins prior or before to each pre- prandial 3 meals of breakfast, lunch & dinner. This pre-prandial intervention works as a metabolic priming strategy, activating cephalic, gastric, intestinal, and neuro-endocrine phases before carbohydrate exposure, thereby re-programming nutrient partitioning toward oxidation rather than storage

For example, prior 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. Pulse proteins are slowly digestible plant proteins that prolong gastric emptying, enhance antral distension, and produce sustained cholecystokinin (CCK) secretion, leading to early satiety and reduced post-prandial calorie intake. So, taking 2 grams of all pulses plant proteins in three pulses pre- prandial nutrition (breakfast, lunch and dinner). A total of 15 grams of protein will be available in a day from these three-pulse pre-prandial nutrition. Then balance 45 to 60 grams 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.

Combining plant protein with high biological value animal protein improves essential amino acid availability, especially leucine, without causing pathological hyper-insulinemia or excessive mTOR activation, thus preserving insulin sensitivity while maintaining anabolic signalling. Thereby produce better health benefit. These three-pulse nutritional therapy should be differently prepared for better health benefit.

1) Prior pre-prandial breakfast nutrition therapy

Total 20 grams moong and red gram seeds should be germinated in night and that sprouts should be taken with 35 grams of whole milk cheese. Germination enhances enzymatic proteolysis, reduces phytate-mineral binding, increases arginine and glycine bioavailability, and improves nitric-oxide-mediated gastrointestinal perfusion, thereby enhancing nutrient sensing and digestion.

2) Prior lunch pre-prandial nutritional therapy:

'Arhar dal' or 'moong dal of 20 grams and 35 grams of milk 'paneer'. Casein-dominant milk proteins slow gastric emptying, sustain GLP-1 secretion, suppress hepatic glucose output, and reduce post-prandial glycemic variability.

3) Prior dinner pre-prandial nutrition therapy:

20 grams of red gram 'chhatua' and 2 eggs without yolk. Egg albumin provides sulphur-containing amino acids essential for hepatic glutathione synthesis, improving mitochondrial redox balance and reducing overnight oxidative stress.

NB: Water or liquid intake in 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 minutes. After food intake. Excess fluid intake before meals dilutes gastric hydrochloric acid, delays pepsinogen-to-pepsin conversion, and suppresses early protein-induced incretin release. Then food in stomach becomes pasty and semi solid and they pass in a linear row to the small intestine. After these cephalic phases of 20 minutes. 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.

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. 10–15 ml oil provides approximately 9–14 grams of fat and acts as a potent activator of PPAR-α, enhancing hepatic and mitochondrial fatty acid oxidation. Mustard oil, olive oil, and peanut oil contain 60% monounsaturated fatty acid, 21% polyunsaturated fatty acid and 12% saturated fatty acid. Monounsaturated fatty acids stabilize mitochondrial cardiolipin, preserve electron transport chain efficiency, and generate less reactive oxygen species per ATP molecule compared to saturated or polyunsaturated fatty acids. Our body required 200 grams of fatty acid daily producing 1800 kilocalories out of 2000 required calorie per day for a normal human. Total 40 grams fats are available from 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) The addition of mustard oil direct to suitable fruit and salad etc. will improve its taste and flavour.
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 benefits.

Pleotropic effects of monounsaturated pulse nutritional therapy:

1) Reduce gastric motility
2) Increase GIP, GLP1 AND CCK production.
3) Produce satiety and reduce hunger.
4) Reduce insulin resistance in periphery.
5) Decreases triglyceride and LDLc level in blood.
6) Improve diabetes profile.
7) It is a better substrate for mitochondrial respiration, function and health conditions better than poly unsaturated and saturated fatty acid.
8) 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. Protein and fat-induced incretin secretion improves early insulin signalling while preventing late-phase hyperinsulinemia.
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. There are no functional GLP-1 receptors in adipose tissue, making GIP the primary incretin for peripheral fat metabolism.
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 bile, 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 (exercise after meal- Post prandial exercise6):

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 Beta 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 Beta 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 Beta 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).

I.E.: https://www.who.int/news/item/17-07-2023-who-updates-guidelines-on fats- and carbohydrates Published July 2023.

Pleotropic 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 and promote peristalsis movement in GI tract and thereby reducing the prandial calorie intake. Post-prandial moderate intensive exercise will burn triglyceride fatty acids in muscles and rest to be stored in Adipose tissue and muscles for future energy use. Post-prandial moderate intensive exercise will reduce glucose in blood circulation by up taking of glucose in muscle for energy to be used in post-prandial exercise. After exercise, glucose is deposited in the muscles as glycogen for storage. So, there will be no glucose available for ectopic fats production from post prandial sugar. There by reducing the chronic inflammation state in liver and pancreas by avoiding ectopic fat deposit from transferred sugar.

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 Beta 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 the best substitute than saturated fat for mitochondrial respiration and thereby favourable Beta oxidation in mitochondria9. And it also promotes mitochondrial formation and function. Food should contain more monounsaturated fatty acid for better mitochondrial respiration. Omega 3 ploy unsaturated fatty acid should be taken in comparison to omega 6 fatty acids to avoid pro inflammatory & chronic inflammatory & metabolic disorder of the body. Because Omega 6 fatty acids produce all pro inflammatory, chronic inflammation & metabolic disorder so nutrition should be managed accordingly and moreover, long chain OMEGA3 fatty acid EPA is approved by FDA to reduced triglyceride level in blood by improving fat metabolism peripherally.

Oil containing more unsaturated fatty acid (MUFA) will give rise to better Beta-oxidation and improve mitochondrial life and respiration as well as new mitochondrial formation. PUFA contains more omega 3 and less omega6 to be used to avoid inflammation of the body. Dietary fatty acid nutritional foods should be managed targeting more monosaturated oil. Such as Olive oil, mustard oil, peanut oils for cooking purpose. Polly unsaturated Fatty acid (PUFA) containing Omega 3 and Omega 6 should be totally avoided for cooking purpose as these are easily converted to trans-fat fatty acid by simple boiling. Half-life of trans fat fatty acid in body are 3 months and thereby producing oil pro metabolic disorder. PUFA containing OMEGA 3 and OMEGA 6 should be served in salads dishes and add to boiled foods to avoid trans-fat formation. Daily requirement of PUFA is only 5 gr. per day and it is mostly used for cell signalling system of the cell and the most important role HDLC is to collect excess available PUFA from nearest cell microcell phospholipid layer and to distribute these PUFA to other needy cells for cell signalling system. Only HDLC managed these distribution process for proper coordinator cell signalling system to produce enzymes, hormones etc.

The best ideal fat management in the body & muscles is:- Muscles Fat content:-1.5 % of total body weight is normal ie. BMI 18% to 22.9%. Muscles fat 1.5 to 5% is overweight ie. BMI from 23% to 29.5%. Muscles fat more than 5% ie. BMI more than 30% and obesity treatment should be started by present guideline.

2. Conclusion

From ancient period all placental delivered animals including the human beings 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 Beta oxidation in mitochondria so for better Beta 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 grams of sugar , and in 3 meals per day we are taking 270 gm 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 gm of sugar from each meal out of 90 gm 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.

Fatty acid from dietary and liver source to be esterified forming triglyceride in order to supply fat energy to all tissue organs and cells of the body. For transport in blood triglyceride should be incorporated into one APOB molecule (a non-detachable fixed structural protein) for formation of one molecule of VLDLc carrying 1000 to 1500 number of triglycerides. VLDLc with non-detachable fixed APO-B is the transporter of triglycerides to all part of the body and also distribute triglycerides to HDLc for better management and also receive triglycerides from chylomicron in exchange of cholesteryl esters from VLDLc and its remnants. VLDLc with its remnant's (IDLc and LDLc) travel along endothelial lining of circulating system to deliver fatty acids to all parts of the body by attaching to lipoproteins lipids in endothelial cells. VLDLc with its remnants are heterogenic in size and are according to number of triglyceride and cholesterol esters they contained. Heterogenic size VLDLc remnants after distributing (losing) 85% of triglycerides to different parts of the body, the VLDLc remnants with non-detachable fixed APOB are endocytosed by LDLc receptors and LRP in liver cells to excrete as cholesterol and bile acid in bile or make it free cholesterol to be incorporated through HDLc or formation of cholesteryl ester for recycling in VLDLc.

VLDLc remnants smooth passage for degraded end products is affected when VLDLc remnants with APOB are trapped by widening of endothelial gap junctions formed in different locations of arterial wall due to shear forces injury, smoking, diabetes etc. That APO-B is never an atherogenic rather than the most essential for quality distribution of triglycerides in the body. That APO-B containing VLDLc remnants can remain inside macrophages as foam cells from childhood to old age until macrophage foam cells are overloaded above 60% and undergo lipidations and thereby producing proinflammatory cytokines in the local surrounding area and that activate innate and adaptive immunity producing fibrosis, necrosis, apoptosis and chronic inflammation state nearby sites. That APO-B of VLDLc remnants for triglycerides are not atherogenic but when it passes through sub endothelial layer trapped by dysfunctional endothelial wide gap junction and transferred to a antigen signal for activating innate and adaptive immune system.

HDLc formation depends on free cholesterol present in cytosol of hepatic liver cell. High free cholesterol in liver cytoplasm will increase the HDLc. Low free cholesterol will decrease HDLc formation as per the requirement. Saturated fatty acid in diet is natural. Monounsaturated fatty acid like oleic acid is essential and act as a source of clean energy for mitochondrial respiration. PUFA (Omega 3 & 6) are required for formation of cell membrane bi-phosphorylate and helps in cell signalling system, but minimum quantity is required 4-5 gram daily. PUFA (Omega 3 & 6) should not be used as deep fried, as it converts to trans-fat very easily and trans-fat half-life in body is 3 months, producing all metabolic disorder. So, PUFA should be used for dressing salad purposes not for fried purposes. De novo lipogenesis from sugar and protein should be avoided in liver as these are most pro-inflammatory and immunogenic producing atherogenic factors. Each meal post-prandial moderate intensive exercise reduces De novo lipogenesis in liver and for the purpose of best nutritional management. Modern metabolic disease arises from chronic excess glucose intake, persistent insulin elevation, suppressed fat oxidation, and mitochondrial overload. Fat management could thereby be more effective than glucose or protein control alone.

NB: VLDLc endocytosis increases free cholesterol level in liver cytoplasm thereby also increases HDLc formation by reducing VLDLc endocytosis in liver will lower HDLc formation and increase VLDLc remnants in the blood. HDLc cholesteryl esters in hepatocytes of liver are mainly transferred to Biles for excretion in Biles but VLDLc remnants produce more free cholesterol in the cytoplasm of liver and thereby increasing HDLc Production.

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 activities are within few seconds, and this manage orexigenic sensitivity and anorexigenic in a co-ordinated manner. It regulates 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 arcuate 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 produces agonist effect of estrogen on hypothalamus sensing as if more estrogen is 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.

Management-1:
Regular post meal moderate intensive exercise for nutritional management specially to reduce deposit of ectopic fats in liver and pancreatic gland from converted sugar to saturated 14 & 16 carbon most inflammatory saturated fatty acid to be deposited after post prandial meal.

Management- 2 (Nutritional Therapy):
Our dietary food for fatty acid management should contain more mono saturated fatty acid in oil approximately 60% preferably from virgin mustard oil, virgin ground/peanut oil and olive oil, all are equal in containing monounsaturated fatty acid about approximately 60% but ground nut oil is having more smoking index suitable for fried foods. In cooking oil try to avoid poly unsaturated fatty acids as it produces more trans-fat at its boiling point and give rise formation of trans fat and half-life of trans fat is approximately 3 months producing all chronic inflammatory disorders in the body.

Advise: Use only oil containing monounsaturated fatty acids for cooking purposes which also supply clean energy and produce better mitochondrial respiratory health and new mitochondrial formation. The PUFA containing oil should be used in food salads as dressing and add to boiled vegetables and containing more Omega3 fatty acids oil and that will reduce acute and chronic inflammatory disorders in the body and PUFA is most essential fatty acids required for cell membrane formation and cell signalling system in the body and its daily maximum requirement is of 4-5 gram only, so it can be easily collected.

Pharmacological Management:
a) For Only Cholesterol management in blood:
Statin is used to reduce cholesterol production in hepatocyte cells and ezetimibe inhibit cholesterol absorption in GI track that is collected from dietary foods Biles and mucosal cell layer of GI tract. Thereby the best management of cholesterol in circulation is to use stain and ezetimibe combination. Only PCSK9 enzyme inhibitor may be added to combination of statin and ezetimibe on the failure of target achievement.

b) For improvement in hepatobiliary GI tract circulation properly and to reduce cholangiectasis and improve hepatocytes functions in liver and to improve more hepatobiliary GI circulation quality and quantity. Cholestyramine as drug should be used and cholestyramine pleotropic effects are more bile acid production in liver thereby consuming more cholesterol from hepatocyte effecting low cholesterol level in hepatocyte cystosol and this will lead to receiving cholesteryl esters from HDLc and ILDc and LDLc from blood circulation. So, combining statin with cholestyramine only not to reduce cholesterol level in blood but also reduces cholangiectasis in biliary circulation thereby improving hepatocytes functions in liver and improvement of hepato-biliary-GI circulations. And all those pleotropic effects of cholestyramine are due to 80% bile acids combined with cholestyramine are excreted in stool so demanding more bile acid formation from liver cholesterol. Thereby increases cholesterol pool from blood circulation mediated by increasing LDLc receptors in hepatocyte cell membrane.

N.B: By pleotropic effect of insulin receptor in arcuate nucleus, it will immediately activate in few seconds brown fat (BAT). By regulating myostatin and there by immediately reduce insulin resistance in the body and it is irrespective to nutritional status.

N.B: GIP agonists are important in managing fat metabolism both centrally and peripherally Leptin agonist are most effective in fat management both centrally and peripherally.

N.B: Fat management is the most essential effective management than glucose and amino acid management. Producing more than 50 % metabolic disorder reduction than in comparison with management of sugar and amino acid. So FAT management is the best metabolic disorder management to exist all other management.

N.B:
1) Statins with ezetimibe has no effect on dietary fat absorption. So, taking a long time have only cholesterol lowering effect, no effect on fat absorption.
2) Statins with cholestyramine reduces fat absorption with fat soluble vitamins in GI tract. So long term use is prohibited, and intermittent short-term use is only advised, as body requires dietary fats, monosaturated and poly unsaturated fatty acid for clean energy supplied to mitochondrion for energy and PUFA supply for cell membrane formation and cell signalling.
3) Orlistat a lipase inhibitors used for obesity(FDA approved drug)should not be used for a continuous period to reduce obesity, as orlistat inhibits absorption of essential dietary fat MUFA and PUFA and this lead to formation of sugar converted fat of 14 and 16 carbon fatty acid by De Novo lipogenesis from sugar and amino acid and this transferred 14 and 16 carbon fatty acid are most dangerous inflammatory fatty acids producing all chronic inflammatory diseases such as atherosclerosis and arthritis and foam cells etc. So, restriction of dietary fat is totally unhealthy and not ideal in any circumstances as it always led to formation of sugar and amino acid converted fat for store in adipose tissue, producing chronic inflammatory condition of stored fat in body.

Scientific committee members Contact Details:

Sr	Name	Email Id	Mobile No
1	Dr. Abhay Kumar Sahoo	*Drabhay76[at]gmail.com*	6372616678
2	Dr. A K Singh	*Ashoksingh3260[at]gmail.com*	9861010130
3	Dr. A.B Nayak	*Artabandhunayak053[at]gmail.com*	8917276255
4	Dr. Abdul Halim Khan	*Abdulhalimkhan03[at]gmail.com*	8895813258
5	Dr. Abhaya Das	*Abhya0008[at]rediffmail.com*	7978413461
6	Dr. Aditya Narayan Pal	*Apal523[at]gmail.com*	8249124296
7	Dr. Akshaya Kumar Mishra	*omkaku[at]gmail.com*	9437389296

8	Dr. Amitav Mohanty	*dramitav[at]yahoo.co.in*	9937028441
9	Dr. Amiya Prasad Choudhury	*Amiyachoudhury.101[at]gmail.com*	8093767096
10	Dr. Ashok Ku Mahapatra	*Akmahapatra2000[at]gmail.com*	8338004444
11	Dr. Ashok Kumar Das (Dean MGM®, Pondicherry)		
12	Dr. Ashutosh Swain	*draswain[at]gmail.com*	7008298446
13	Dr. B Jagdish	*Masterdoctor.clinic[at]gmail.com*	8093008191
14	Dr. B M MISHRA	*Biswamohan51[at]gmail.com*	9437162710
15	Dr. B N Panda	*brigpandabbsr[at]gmail.com*	
16	Dr. B Panigrahy	*Bharat.panigrahy[at]gmail.com*	9437060977
17	Dr. B S Das (MD, FNASc)	*bhabanisankar[at]gmail.com*	
18	Dr. B. K. Pal	*drbasantpal[at]gmail.com*	8895549441
19	Dr. B.N Mishra	*Biswanath.mishra2010[at]gmail.com*	9437031462
20	Dr. B.P Nanda	*Bishnupn59[at]gmail.com*	8908886166
21	Dr. Bijay Mishra	*Dr_bijaymisra[at]yahoo.com*	9437232824
22	Dr. Bijaya Ketan Dash	*bijayaketandash[at]rediffmail.com*	9437044544
23	Dr. Bijaya Kumar Mishra	*bijayakumarmishra[at]yahoo.co.in*	9437012255
24	Dr. Bijin Kumar Sial	*Bijin_Siallo[at]ifffer.com*	9861066734
25	Dr. Brig Ambika Mohanty (KIMS)	*Ambikamohanty11[at]gmail.com*	9437074292
26	Dr. Dayanidhi Meher	*dayanidhimeher[at]gmail.com*	9937793342
27	Dr. Dinabandhu Sahoo	*Dinabandhu1970[at]gmail.com*	9937219147
28	Dr. G Sahoo	*Sahoog2002[at]yahoo.co.in*	7708949275
29	Dr. G.C Sahoo	*Dcsent99[at]gmail.com*	9861716151
30	Dr. J K Samanataray	*Jagat.dr[at]gmail.com*	
31	Dr. J Kiskore	*dreamclinicQi05[at]yahoo.co.in*	9337102704
32	Dr. J.N. Mishra	*mishrajagdenanda[at]gmail.com*	
33	Dr. Jayaram Patro	*drsangram3132[at]gmail.com*	9337103131
34	Dr. K B Parida	*drkrutibas[at]gmail.com*	9437004948
35	Dr. K.K Jajodia		9437017879
36	Dr. Kailash Biswal	*kailashbiswal[at]yahoo.com*	
37	Dr. Kailash Chandra Agrawal		9437243141
38	Dr. Kedarnath Panda	*K n panda gastro[at]gmail.com*	9861035744
39	Dr. Kshirod Chandr Behera	*brigkcbehera[at]yahoo.in*	7978986210
40	Dr. Kshyama Sagar Sahoo	*Kshyamasagar.sahoo1951[at]gmail.com*	7609044690
41	Dr. L K Meher,Sum Hospital	*drlkmeher[at]gmail.com*	9861184752
42	Dr. L S Nivasa Mohanty	*drlsm[at]lcrc.in*	9448051046
43	Dr. Laxmipriya Tudu	*Drlaxmi_p[at]rediffmail.com*	9437214022
44	Dr. M K Chhotray	*chhotraymanoj[at]yahoo.com*	9437302355
45	Dr. M P Tripathy	*drmptripathy[at]gmail.com*	337014758
46	Dr. M. R Pattnaik	*Manasranjanpattnaik2013[at]gmail.com*	
47	Dr. Madan Sundar Panda	*drmspanda[at]gmail.com*	9438668608
48	Dr. Manoranjan Behera	*manoranjan[at]rediffmail.com*	9437031672
49	Dr. Mridual Bera	*Beramridul2[at]gmail.com*	9830170613
50	Dr. Nibedita Pani	*drnp[at]rediffmail.com*	9437004747
51	Dr. Niranjan Tripathy	*drntripathy[at]rediffmail.com*	7008472820
52	Dr. P C BAHINIPATI	*purnabahinipati[at]hotmail.com*	9861040381
53	Dr. P C Mohanty	*drpcm[at]yahoo.co.in*	9437199837
54	Dr. P C Panda	*Pratap.panda454[at]gmail.com*	7978929310
55	Dr. P K Patnaik		9910027120
56	Dr. P K Sahoo	*Drprasant_s[at]apollohospital.com*	8093060049
57	Dr. P. K Sahu	*Drpksahu4[at]gmail.com*	
58	Dr. P.K Sahoo		9937091686
59	Dr. Pranabandhu Sahoo	*drPronabandhu[at]rediffmail.com*	9348715511
60	Dr. Prasanna Kumar Das	*Dr.prasannadas[at]gmail.com*	9437094944
61	Dr. Pratyush Kumar Ray	*Dr.partyush.ray[at]gmail.com*	9337234860
62	Dr. Pravat K Thatoi	*drpravatthatoi[at]yahoo.co.in*	9437170150
63	Dr. Prem Sagar Panda	*drpspanda[at]gmail.com*	7415658748
64	Dr. Purna Chandra Dash	*Purnadash5865[at]gmail.com*	9437975494
65	Dr. Purna Chandra Rath	*drpurnarath[at]yahoo.com*	9861443398
66	Dr. R. K Choudhury	*Rkchoudhury1950[at]gmail.com*	
67	Dr. R.G Dash	*rgdsanjeevani[at]gmail.com*	
68	Dr. R.K Pati	*Dr.rkpati[at]gmail.com*	9457276133
69	Dr. Rabi Narayan Kar	*Rabinarayankar1943[at]hotmail.com*	9437155260
70	Dr. Raj Ashok Das	*Rrajeshdas12[at]gmail.com*	
71	Dr. Ram Niranjan Sahoo	*Ram_niranjan[at]yahoo.com*	9493977948
72	Dr. Ramakanta Parida		7205474017
73	Dr. Ranjit Kumar Panda	*Drranjitpanda1988[at]gmail.com*	8249652559
74	Dr. Ritesh Kumar Agrawala	*drriteshagr[at]gmail.com*	848050080

75	Dr. S K Bhanja	*Swarupbhanja53[at]gmail.com*	7978049564
76	Dr. S K Mahapatra	*Drskm_med[at]yahoo.co.in*	9437067855
77	Dr. S K mohanty	*dr.skmohantybbs[at]gmail.com*	9437069148
78	Dr. S Kabi	*carexclinic[at]gmail.com*	9861297471
79	Dr. S. C Pradhan	*Satishpradhan65[at]gmail.com*	
80	Dr. S. K Sahu	*sahudrsaroj[at]gmail.com*	9437002424
81	Dr. Sabita Mishra	*Sanjeevani.sabita[at]gmail.com*	7077731131
82	Dr. Sambeet Swain		9777506425
83	Dr. Sambit Das	*drsambitkudas[at]gmail.com*	8093060177
84	Dr. Sanjib Kumar Kar (IIGN, CTC)	*drsajibkar[at]yahoo.com*	9437074292
85	Dr. Santosh Kumar Routray	*Santosh.routray62[at]gmail.com*	9861240793
86	Dr. Saraswata Mishra	*Kuni8422[at]gmail.com*	
87	Dr. Sarat Ch Panigrahi	*Saratpanigrahi[at]nalissmail.com*	9937936499
88	Dr. Sauri Prasad Samantrai	*drspsamntrai[at]gmail.com*	9437304009
89	Dr. Seba Mohapatra	*drsebamohapatra[at]gmail.com*	9437035531
90	Dr. Srikanta Mahapatra	*Dr_smahapatra[at]yahoo.com*	7674006013
91	Dr. Srinibas Sahoo	*Ironman.srinibas[at]gmail.com*	8117087511
92	Dr. Subhendu Dash	*drsdash[at]protonmail.com*	
93	Dr. U K Mishra	*umakantamishra[at]gmail.com*	9437308717
94	Dr. Jayant Panda	*drjayantapanda[at]gmail.com*	9437678788
95	Dr. Basanta Mishra		9437289410
96	Dr. Deepak Ranjan Das	*maildripak[at]gmail.com*	
97	Dr. Bibekananda Kar	*bibekbhu[at]yahoo.co.in*	
98	Dr. Prabhat Mallick		
99	Dr. Biswanath Mohanty		
100	Dr Shilpa Varma	*shilpavarma2008[at]gmail.com*	
101	Prof (Dr) L. SREENIVASAMURTHY	*drlsm[at]lcrc.in*	9448051046
102	Dr. Kailash Ch. Dash	*Kailsashchandradash40[at]gmail.com*	9437001247
103	Prof. (Dr.) Pratap Ku. Pradhan	*Pradhanpratap1951[at]gmail.com*	
104	Dr. Rabindra Ku. Nayak		7809044442

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