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REVIEW PAPER
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Flax Seeds and Walnuts: Biochemical Lipid Homeostasis by Alpha Linolenic Acid and Linoleic Acid in Obviating the Dyslipidemia

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ABSTRACT

Dyslipidemia is extremely prevalent in urban and rural area of developing countries especially India and its high pervasiveness is responsible for causing the complication of cardiovascular disease. Considering this, a review research was conducted on the biochemical mechanisms of essential fatty acids like alpha linolenic acid (ALA) and linoleic acid (LA) through the prescribed intake of flax seeds and walnuts in overcoming the complication of dyslipidemia. ALA and LA modulate the mechanisms in the form of stimulation of Reverse Cholesterol Transport (RCT) process , activation of PPAR receptor, deadlocking the COX2 and TNF operation, dismissing the expression of enzyme stearoyl CoA desaturase 1, promotion of cholesterol 7- alpha- hydroxylase activity, amplification of sterol retinol binding protein cleavage activated protein parameter and cessation of proprotein convertase subtilisin kexin type 9 (PCSK9) operation which sequentially is in charge for bringing down the process of thrombosis and plaque genesis. These thrived biochemical mechanisms can only be sustained by a requisite intake of flax seed and walnuts which assists in advancing the ani-thrombic, anti- erythrogenic, anti-thriving, cholesterol efflux, anti prooxidant biochemical activities. Contemplating mechanical eminence of ALA and LA in lipid stabilization, the present review outlines the biochemical homeostasis of ALA and LA in obviating the intricacy of dyslipidemia through the streamlined intake of flax seeds and walnuts.

Key Words : Dyslipidemia, PUFA, ALA, LA, Biochemical mechanisms, Heptobiliary elimination, Flax seeds and walnuts

INTRODUCTION

Eminence of fatty acids :

Researchers assimilated that the fatty acids have been recognized to be a necessitate nutritional component in the healthy prolongation of metabolic system. They have been tenacious in the preponderance of body components where eminent characters are being executed by them in perpetuating the healthy metabolism (Calder, 2015). Fatty acids have a fundamental engagement in communicating signals from one cell to another, impacting the membrane adaptability and pliability, regulating the antioxidant signaling pathway by substituting with the antioxidants and henceforth directing the erythrogenic process (Calder, 2017). The persistence of functional, dietary and therapeutic properties forges the fats into one of the indispensible constituents for humans.

Biochemical benefaction of ALA and LA towards the healthy metabolic system :

Curtailed derivation of polyunsaturated fatty acids (PUFA) like alpha linolenic acid (ALA) and linoleic acid (LA) have been correlated with the escalated prevalence of several disease and disorders like hypertension, diabetes, cardiovascular disease, skin disorders, renal disorders, malfunctioning of brain activity, depression, anxiety, and decreased immunity (Simopoulos *et al.*, 1999). It has been determined that the reason behind the incidence of metabolic complications is the deficient proportion of metabolic products of ALA and LA like

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Docosahexanoic acid (DHA) , Eicosapentanoic acid (EPA) and Arachidonic acid (AA), respectively. The biochemical conversion of ALA to EPA, DHA and LA to AA entails a catalyst which backs the whole biochemical process by metamorphosing the substrates (ALA and LA) into metabolic products. But the scarcity of catalysts or substrates leads to the dearth of metabolic product formation which successively is interrelated with the occurrence of innumerable metabolic diseases (Sakai *et al.*, 2017).

Maintaining the equilibrium of ALA and LA through the aversion of refined or processed food :

Refined food devoured by the people is elevated in linoleic acid *i.e.* omega 6 and deficient in alpha linolenic acid *i.e.* omega 3. It's habitual and consistent intake results in the scarcity of omega 3 *i.e.* alpha linolenic acid which is an essential fat, vital for the sustainability of healthy metabolic system. Higher proportion of substrate omega 6 or linoleic acid (LA) in the blood imbalances the omega 3 and omega 6 ratio which sequentially is accountable for destabilizing the healthy metabolic system and procurement of omega 6 through excessive junk food consumption causes the aggravation of blood composition. So to avoid this, it becomes extremely vital that metabolic conversion of alpha linolenic acid to EPA and DHA, LA to AA should be unchallenging so as to repress the potency of variant metabolic disease and disorders (Baker *et al.*, 2016; Walker *et al.,* 2015).

Essence of ALA and LA: Subduction of Dyslipidemia:

Besides lessening the contingency of several metabolic diseases, substrates like ALA and LA have an exceptional and noteworthy part to perform in thwarting the issue of inflated and disturbed range of lipid profile. ALA and LA abet in subduing the levels of triglycerides, LDL cholesterol, total cholesterol and succor in enhancing the HDL cholesterol. Systemized and stipulated proportion of food products rich in omega 3 and omega 6 braces the preservation of equilibrated lipid profile which in turn is accountable for mastering the complication of cardiovascular disease like atherosclerosis, ischemia and myocardial infarction (Walker *et al.*, 2015; Baker *et al.,* 2016).

Objectives of study :

Hence, the present study aims to prospect :

i) the biochemical mechanisms of ALA and LA in context with the flax seeds and walnuts in lessening the medical issue of lipidemia

ii) the recommended daily dietary intake of ALA and LA rich foods in flourishing biochemical lipid mechanisms

iii) the nutritional role of ALA and LA in bringing down the impediment of perturbed lipid profile on grounds of verified and authenticated based studies.

Following physiological mechanisms ascertaining the health benefits of ALA and LA by bridling the issue of dyslipidemia-

Alpha linolenic acid (ALA):

Reverse Cholesterol Transport process (RCT): Hepatobiliary Elimination:

ALA has an advantageous potentiality in averting the complication of cardiovascular disease by advancing the operation of Reverse Cholesterol Transport (RCT). RCT is a process which ameliorates the decayed and deteriorated lipid parameters by conveying the excess of cholesterol from circumferential tissues to hepatic cells for hepatobiliary elimination which is then bounded by the equilibrated, nourish able and salutary lipid profile. This mechanism involves the discontinuation of circumferential, subvascular macrophage and chrondocytes procured cholesterol either straightly via HDL or divergently by transmitting cholesterol from constituents of HDL cholesterol to apoB lipoprotein for instantaneous absorption into liver cells through LDL receptors (Siddiqui *et al.*, 2015; Ali *et al*., 2012).

Modulation of cholesterol through extracellular and intracellular pathway:

It was ascertained that synthesis and breakdown process of cholesterol components is bifurcated into an extracellular and intracellular mechanism. In the intracellular mechanics, hepatic and supplemented hepatic tissue manifests and synthesizes the cholesterol parameters and forges into blood circulation in the form of lipoprotein where majority of it is converted into bile components. On other hand, in extracellular pathway, cholesterol is procured from dietary and bilary origins where they are absorbed into the intestine and then making the way into blood circulation (Shephard et al., 2001). Due to the peripheral cholesterol breakdown process, deluge of intracellular cholesterol and conveyance of cholesterol from tissues to liver is eminent

and indispensible so as to avoid the precipitation of cholesterol crystals in arteries which successively averts the complication of dyslipidemia (Phillips *et al.*, 2014).

Active and passive pathways of cholesterol efflux (essential step of RCT):

ATP- binding cassette transporters A1 (ABCA1) and G1 (ABCG1), aqueous diffusion and SR-B1 facilitated cholesterol desorption succors in clearance of cholesterol and other lipid parameters into hepatic tissue.[14] This eminent stride-cholesterol efflux prevents the accumulation of cholesterol and other lipid parameter in the arteries. HDL particles and its residues are crucial conciliators of cholesterol efflux (Cuchel *et al.*, 2006 ; Zhao *et al.*, 2010).

Catabolization of apolipoprotein:

ALA has a favourable impact on disturbed lipid profile as it abets in uprooting and eradicating cholesterol remnants from the circulating pathway. It has been revealed that EPA and DHA- the metabolic products of omega 3 has been considered functionally potential in promoting the breakdown of apolipoprotein B-100, subduing the hepatic apoB production and enhancing the withdrawal of plasma triglyceride through the enzyme lipoprotein lipase which in succession uplifts the elevated transformation of VLDL to LDL, thus lowering the manifestation of LDL components and weakening the process of postprandial lipid synthesis (Chan *et al.*, 2003; Park *et al.*, 2003).

Subjugation of Sterol retinol binding protein:

ALA has a repressive effect on sterol regulatory element binding protein -1 mediated mechanism involving actuation of nuclear transcription factors, hepatocyte nuclear factor-4 alpha, etc which backs in preventing the raised lipid parameters (Calder, 2010). Therefore, it has been deduced that omega 3 (ALA) promotes Reverse Cholesterol Transport process by elimination of cholesterol and other lipid parameters into bilary constituents which sustains in stabilizing the healthy lipid parameters thus, conquering and surpassing the issue of dyslipidemia.

Besides the mechanism of RCT, ALA has an eminent part in the regulating the following lipid biochemical operations:

Stimulation of Perioxisome proliferator actuated alpha and gamma receptor (PPAR):

PPAR performs an eminent role in regulating the lipid metabolism. When the concentration of fatty acid rises, biochemical paraphrase of PPAR's genes gets stimulated by the metabolism of long chain fatty acids like DHA and EPA. Oxidation system like mitochondrial and perioxisomal beta oxidation also gets triggered by the metabolization process. As soon as both the operations get energized, discerning of PPAR in liver increases which heightens the level of catabolized energy and reduces the storage of fats in the form of adipocytes. Activated PPAR's also synchronize the utterance of enzymes which are necessitated with the raised catabolism of mitochondrial fatty acids and transformation of fatty acids into acyl coenzymes (Gervois *et al.*, 2000; Grygiel-Górniak *et al*., 2014).

Demilitarizing the cyclooxygenase-2 (COX-2) and TNF activity:

ALA has the functional ability in decreasing the activity of inflammatory gene— cyclooxygenase-2 which is involved in increasing the process of thrombosis. Cyclooxygenase-2 activates the metalloproteinase's which is accountable for the genesis of angiogenesis and plaque breakage. Cyclooxygenase-2 (COX-2) has been found to be persistent in the fatty streaks and with the assistance of oxidized LDL and IL-1, COX-2 instantly triggers the adherence of monocytes to stimulated endothelial cells. IL-1 α/β, TNF- α and CD40 ligand is taken into the account of the process of induction of COX-2 in monocytes cells which means TNF- α promotes the induction process of COX-2. Hence, it becomes essential to demilitarize the cyclooxygenase (COX-2) and TNF activity which successively assists in stabilizing the aggravated lipid parameters (Cipollone *et al*., 2003).

Inactivation of stearoyl CoA desaturase 1 utterance (SCD1):

Stearoyl COa desaturase 1 is the enzyme utilized in the genesis of MUFA. ALA and Stearoyl CoA desaturase 1 reduces the utterance of stearoyl CoA desaturase 1 in macrophage cells. ALA also triggers the activation of farnesoid –X- receptor which controls the SHP gene execution and sterol regulatory elementary binding protein (SREBP) transcription receptor. This process successively lessens the activity of SCD1 which is responsible for increasing the rate of cholesterol effluence from the hepatic cells and reduces the fats and cholesterol

depository within the cells (Zhang *et al.*, 2012).

Thus, it has been that derived that ALA procurement flourishes these mechanical actions which successively normalizes the ruined lipid parameters.

Linoleic acid (LA):

Cholesterol 7- alpha- hydroxylase activity:

It has been established from a study that suitable and orderly intake of linoleic acid (LA) upgrades the rate of hepatic LDL receptor gene (LDLR) and protein expression which in order surges the removal of LDL cholesterol from liver cells thus, clearing the pathway of mechanical hepatic process. Omega 6 functionally triggers the organization of liver X receptor alpha gene through escalated mushroomed receptor which raises the utterance of cholesterol 7- alpha hydroxylase gene, thus encrypting the enzymatic mechanism of cholesterol transformation into bile constituents. Due to the persistence of activated LDLR receptor, this mechanical process withdraws all the constituents of LDL cholesterol, making the breakdown and eradication of circulating cholesterol. Hence, altering the utterance of liver X receptor alpha gene into cholesterol 7 alpha-hydroxylase gene aids in encrypting and controlling the biochemical process of breakdown of cholesterol components into bile acids. Therefore, it is derived that cholesterol 7- alpha-hydroxylase activity to be ameliorated and boosted which has the capability in overpowering and mastering the disturbed lipid profile (Brown *et al.*, 2018).

Sterol retinol binding protein cleavage activated protein (SCAP):

Researchers made out that organizational parameters – sterol retinol binding protein bears an eminent capability in depressing the high levels of total cholesterol and triglycerides. It was stated that sterol retinol binding protein is a transcription component which abets in arranging the constituents of gene by encrypting the protein. The encryption of protein ingests the cholesterol molecules and integrates the constituents of cholesterol, triglyceride and fatty acid. Sterol retinol binding is only triggered by sterol retinol binding protein cleavage activated protein (SCAP). SCAP is known for activating sterol binding protein activity which successively helps in clearance and removal of cholesterol from hepatic cells. Linoleic acid (omega 6) upgrades the activation of sterol binding protein which advances the utterance of LDLR , hence promoting the removal of hepatic LDL cholesterol. On contrary side, SCAP has a critical task in stabilizing and balancing the cholesterol levels by hindering the synthesis of SCAP-SREBP complex from leaving the endoplasmic reticulum which in order averts the protein catabolization of sterol binding protein molecules hence, equilibrating the hepatic cholesterol levels. So, besides the excess removal of cholesterol levels from liver cell, SCAP mechanism also succors in balancing the healthy range of lipid parameters for maintenance of flourished and and nourished metabolism (Brown *et al.*, 2018).

Cessation of Proprotein convertase subtilisin kexin type 9 (PCSK9) :

Systemized intake of linoleic acid (LA) helps in declining the activity of proprotein convertase subtilisin kexin type 9 (PCSK9) which is responsible for improving the expression of LDLR, hence promoting the clearance of LDL cholesterol from liver cells (Krysa *et al*., 2017). Genetic variation in LDLR and apolipoprotein B causes the deceleration of PCSK9 activity which increases its excretion from various cells and tissues causing the aggravation of healthy lipid profile and welcoming the complication of cardiovascular disease (Guarnieri et al., 2019). To manifest and demonstrate it more adequately, a study was conducted on altered and disarrayed LDLR / PCSK9 gene among Japanese patients where it was revealed that these types of patients had high menace of cardiovascular disease i.e. myocardial infarction (Watts *et al.*, 2014)

Henceforth, it has been acquired that linoleic acid (omega 6) progresses the healthy biochemical mechanism by withdrawing and clearing the elevated levels of LDL cholesterol and other constituents of lipid profile like elevated triglyceride, total cholesterol, etc. from hepatic cells. Stabilization of lipid parameters is only possible if biochemical mechanism perpetuate in a healthy and progressive way as discussed above (Farvid *et al.*, 2014; Pan *et al.*, 2009)**.** The structural outline of biochemical lipid parameters of ALA and LA has been illustrated (Fig. 1).

Concluding remarks :

This review has accentuated that these physiological mechanisms have a beneficiary effect in obviating the elevated lipid parameters which successively has a worthy impact in mastering the complication of dyslipidemia. To sustain and flourish these biochemical mechanisms, it becomes extremely essential to regularly and adequately

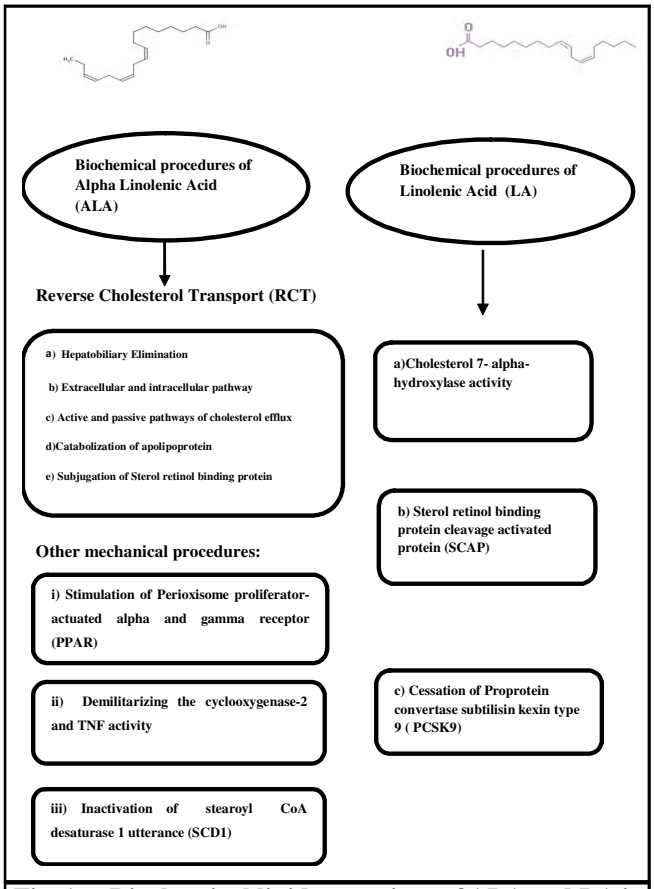

Fig. 1 : Biochemical lipid operations of ALA and LA in overpowering the disturbed lipid profile

consume healthy and nutritious food products rich in both alpha linolenic acid (ALA-omeag 3) and linoleic acid (LA-omega 6). ALA and LA rich food products like soybean, tofu, avocados, fortified food products, eggs, fish, fish liver oil, peanut butter, healthy vegetable oils like olive oil, avocado oil, peanut oil, almond oil, safflower oil, sunflower oil, nut and seeds like chia seed, hemp seeds, pumpkin seed, flax seeds, sunflower seeds, cashew nuts, almonds, walnuts, peanuts, etc have the functional ability in thriving the lipid mechanism by transporting the excess of cholesterol for hepatobilary elimination which in turn is responsible for ameliorating the aggravated lipid profile. Table 1 highlights about the regular consumption of the richest source of PUFA as per the recommended intake which succors in procuring the sufficient proportion of ALA and LA required for the processing of lipid mechanism.

Flax seeds and walnuts:

Flax seeds and walnuts are one of the most indispensable food ingredients augmented with the highest proportion of ALA and LA. Regular consumption of ample proportion of ALA and LA derived from flax seeds aids in strengthening the ani-thrombic, anti- erythrogenic and anti-thriving biochemical operations in effortless way by metabolizing the lengthy fatty acid chains like EPA, DHA and DPA which is responsible for triggering the perioxisome proliferator- actuated alpha and gamma receptor whereas DPA is constituted in demilitarizing the cyclooxygenase I and cellular TNF- alpha operations which enhances the anti- thrombic and anti-erythrogenic activities (Marx *et al.*, 2004). ALA derived flax seeds and walnuts hinders the stearoyl –CoA desaturase-1 activity, lipogenic enzyme through the actuation of nuclear receptor farnesoid X receptor (FXR) progression which causes the effluence of cholesterol from variant macrophages (Caterina *et al.*, 2005; Zhang *et al.*, 2012). Flax seeds and walnuts act as anti prooxidant which subdue and degrade the released free radicals by rummaging the reactive oxygen species and deactivating the oxidative activity which sequentially is blameworthy

Table 1 : ALA and LA rich food products and their recommended intake

Food products	ALA (omega 3) per 100 g (USDA)	LA (omega 6) per 100 g (USDA)	Recommended intake
Flax seeds	22.9 g	5.9 g	15-30 g /day (Mayo health 2015)
Chia seeds	5.84 g	17.8 g	20 g twice /day (Healthline 2017)
Sunflower seeds	0.06 g	23 g	30 g / day (Healthline 2018)
Walnuts	9.08 g	38.1 g	50-60 g /day (Healthline 2018)
Almonds	0.03 g	12.3 g	22-23 almonds/ day (Medical News 2019)
Cashews	0.062 g	7.78 g	28 g / day (Medical News Today 2018)
Canola oil	9.14 g	18.6 g	19 g/day (Very well fit 2020)
Safflower oil	0.096 g	12.7 g	8-9 g /day (Lin L et al 2013)
Avocados	0.125 g	1.67 g	50 g/ day (Huffpost 2018)

for lessening the arousal of redox sensitive proinflammatory transcription factor NF-Kb (Yi *et al.*, 2013). Walnuts reverts the unfavorable changes of SIRTI/ FoxO3a/MnSOD/catalase axis in heart which depresses the proportion of AA/EPA, AA/DHA ratios in the blood plasma (Ros *et al*., 2018). These all biochemical operations are involved in the conflicted thrombic, erythrogenic and propagating mechanical actions. Hence, regular consumption of flax seeds and walnuts succours ALA and LA in balancing lipid mechanisms which abets in overpowering the dyslipidemia (Fig. 2).

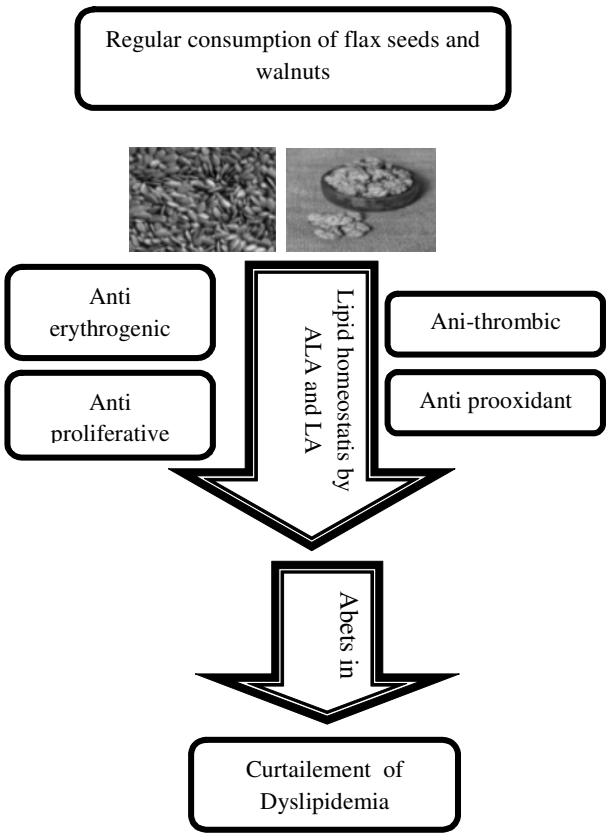

Fig. 2 : Regular consumption of flax seeds and walnuts succours ALA and LA in balancing lipid mechanisms which overpowers the dyslipidemia

Thus, it has been derived that regular and frequent intake of flax seeds and walnuts are extremely helpful in staving off the aggravated lipid profile through the flourished biochemical lipid mechanism. Flax seeds and walnuts have a crucial part in improving the ruined lipid parameters which have been proven scientifically through various clinical trials and systematic reviews or meta analysis represented in Table 2 and 3 where it has been revealed that supplementing flax seeds and walnuts either in whole or milled form, incorporated in food items like biscuits or given solely in specific amount (Edel *et al.*, 2015; Torkan *et al*., 2015 ; Bashan *et al*., 2018) to several dyslipidemic patients has assisted in encouraging the ani-thrombic, anti-thriving and anti-erythrogenic biochemical activities which exceedingly has abetted in depressing the infuriated and elevated levels of total cholesterol, triglyceride, LDL, non HDL cholesterol and enhancing the HDL cholesterol level. Table 2 and 3 have accounted that persistence of ALA and LA in flax seeds and walnuts are chiefly responsible for repulsing the ruined lipid parameters which is only possible through its flourished and progressive biochemical operations.

Persistence of the lipid biochemical parameter in flax seeds and walnuts has made them constituted with bioactive, functional, nutritional and therapeutic properties which bear an immense character in prohibition of dyslipidemia. Progressive and sustained biochemical properties in flax seeds and walnuts have made them extremely appreciable, popular and widely consumed. Due to the uncomplicated mechanical operation of higher proportion of ALA and LA, walnuts and flax seeds are considered to be one of the feasible sources of PUFA.

Conclusion :

In contemporary modernity era, humans are devouring highly processed refined food being raised in saturated fat, trans fat and cholesterol. Its immense consumption on daily basis is making physiological system of human debilitated leading to the inflated incidence of dyslipidemia which is mostly pervasive throughout the world causing the complication of cardiovascular disease. This metabolic suffering of lipid profile is accountable for considering the prominence of nutrients among humans for beneficial health purposes. Among all the nutrients, bioactive components (ALA and LA) of PUFA should be contemplated in obviating the metabolic issue of perturbed lipid profile. ALA and LA have the practical utility in diminishing the biochemical process of artherogenesis and plaque formation by removing the extra cholesterol and other lipid parameters from hepatic tissues into bilary constituents through different physiological mechanisms. These physiological lipid mechanisms can only be flourished by consuming food products rich in ALA and LA.

Flax seeds and walnuts being the richest source of ALA and LA have manifested to enhance the aggravated

Table 2 : Following parameters of variant research studies on flax seeds and blood lipid profile

Type of study	Procedure / Methodology	Outcomes	Presence of bio-chemical constituents	References
Randomzie controlled trial	53 RCT's constipated T2D were fed 10 g of flax seeds pre mixed in cookies for 12 weeks	Curtailment of total cholesterol, triglyceride, LDL cholesterol were observed among dyslipidemic subjects	ALA	Soltanian *et al.* (2018)
Review Article	Literature was collected and analyzed	Total cholesterol ↓	Omega 3 and Omega 6	Parikh *et al.* (2018)
Double bind, randomized, placebo controlled trials	30 g of milled flax seeds incorporated in diet were supplemented to peripheral artery disease (PAD) patients for 12 months.	LDL cholesterol ↓	ALA	Edel *et al.* (2015)
Randomized control clinical trial	30 gram of raw flax seed powder was fed to hyperlipidiemic patients for 40 days	Serum lipids were reduced	Omega 3 and Omega 6	Torkan *et al.* (2015)
Cross sectional Clinical trial	30 g of milled flax seeds were incorporated in regular daily basis diet	Reduction in serum triglycerides level was observed	AA EPA DHA	Ristic-Medic *et al.* (2014)

Table 3 : Following parameters of variant research studies on walnuts and blood lipid profile

Type of study	Procedure / Methodology	Outcomes	Presence of bio-chemical constituents	References
Randomzie controlled supplemented supplemented study	3 different types of diet containing walnuts fed to hyperlipidemic adults for 6 weeks duration	TC LDL cholesterol Non HDL cholesterol ↓	ALA	Tindall *et al.* (2019)
Randomized clinical trial	First group (72 subjects) fed with the regular diet program. Second group (75 subjects) fed with the walnuts.	Second group consisting of 73 subjects which were consuming walnuts showed an elevated reduction in total cholesterol, LDL cholesterol, triglyceride, vldl and enhancement in HDL cholesterol.	LA	Bashan *et al.* (2018)
Meta analysis of 26 clinical trials using databases Pubmed, EMBASE	Comparison of walnut enriched diet with controlled diet on 1059 subjects	TC- 3.25% LDL- 3.73 TG-5.52 ↓	ALA	Guasch-Ferré *et al.* (2018)
Randomized, double bind placebo controlled clinical trial.	100 subjects type 2 diabetic subjects aged 35-75 yrs acquired 15 cc walnut oil for the duration of 90 days.	TC, TG,LDL and TG/ HDL were improved	ALA with an atherogenic property	Zibaeenebad *et al.* (2017)
Literature review analysis	Literature data bases were searched for published trials in which walnut enhanced diet were compared with control diet	Total cholesterol and LDL cholesterol were decreased.	ALA and LA	Banel *et al.* (2009)

lipid profile by stimulating the ani-thrombic, anti-erythrogenic, anti-thriving and anti prooxidant pathways. Numeral interventional studies conducted on walnuts and flax seeds have been proven to be extremely advantageous for patients suffering from hypercholesterolemia, hypertriglyceridemia and hyperlipoproteinemia. This is due to the bioactive presence of ALA and LA in both the food ingredients.

Hence, it can acquired that pervasiveness of dyslipidemia among the majority of population to be perceived by dieticians and nutritionists in interpreting the biochemical significance of ALA and LA by delivering the valuable information to the dyslipidemic subjects about the incorporation of flax seeds and walnuts in daily dietary basis (Hunter *et al.*, 2017).

Thus, it is exceedingly eminent to recognise the

importance of biochemical lipid mechanisms of ALA and LA through the regular and streamlined intake of flax seeds and walnuts which in turn is accountable for bridling the nutritional pathological disorder like dyslipidemia.

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