International Journal of Innovative Research in Computer Science & Technology (IJIRCST)
ISSN: 2347-5552, Volume-9, Issue-5, September 2021
https://doi.org/10.55524/ijircst.2021.9.5.16
Article ID 219516, Pages 79-82
www.ijircst.org

An Overview on Lipids

Chandra Shekhar Yadav

Professor, Department of Life Sciences, Vivekananda Global University, Jaipur, India

Correspondence should be addressed to Chandra Shekhar Yadav; chandra.yadav@vgu.ac.in

ABSTRACT- Instinctive heftiness, dyslipidemia, or insulin obstruction are all important for metabolic syndrome (MetS) (IR). As IR propels, the statement of lipoprotein lipase (LPL), a basic controller of lipoprotein digestion, diminishes. metabolic syndrome manifestations are brought about by an increment of glucocorticoid receptor initiation, which is considered to assume a huge part in metabolic syndrome pathogenesis. Glycyrrhizic corrosive (GA), a bioactive part of licorice roots (Glycyrrhiza glabra), hinders 11-hydroxysteroid dehydrogenase type 1, which catalyze the hydrolysis the initiation of glucocorticoids. As a result, it's recommended that taking GA orally may help with metabolic syndrome. GA was displayed to postpone the development of IR related to tissue steatosis and to forestall the arrangement of stomach stoutness yet, in addition, improve dyslipidemia in rodents under various physiological conditions by specifically attempting to actuate tissue LPL articulation as well as a positive change in serum lipid boundaries, as well as to forestalling the movement of IR related with tissue steatosis.

KEYWORDS- Dyslipidaemia, Glycyrrhizic Acid, Lipids, Syndrome.

I. INTRODUCTION

Since lipids, like cholesterol and fatty oils, are insoluble in water, they should be moved in the circulation system related to proteins (lipoproteins). To stay away from poisonousness, a lot of unsaturated fats from suppers should be moved as fatty substances [1]–[5]. These lipoproteins are significant for dietary lipid assimilation and transport in the small digestive tract, lipid transport from the liver to fringe tissues, and lipid transport from fringe tissues to the liver and digestive system (switch cholesterol transport). Poisonous unfamiliar hydrophobic and amphipathic compounds, like bacterial endotoxin, are moved from areas of intrusion and contamination as an optional capacity [6-9].

A. Metabolic syndrome (MS)

The metabolic syndrome (MetS), otherwise called insulin opposition condition or condition X, is a gathering of hazard factors for atherosclerotic cardiovascular sickness (CVD) and Type 2 diabetes mellitus (T2DM). Insulin obstruction (IR) is believed to be the hidden reason for the condition's different metabolic irregularities, which incorporate stomach corpulence, hyperglycemia, atherogenic dyslipidemia, and hypertension. The disorder puts individuals at a 2-and 5-overlap higher danger of creating cardiovascular illness and diabetes mellitus, separately. In glucose and lipid digestion, glucocorticoids (GCs) assume a significant part [10-13].

B. Lipid Metabolism in its Natural State

Because lipids are involved in energy balancing, reproductive and organ function, and several other aspects of cellular biology, they are essential for life. Because lipids are often insoluble in aqueous settings, they are transported through the body as soluble proteins complexes known as lipoproteins (Crook, 2006). Lipoproteins are spherical particles having non-polar neutral lipids in their core, including triacylglycerol (TAG) and cholesterol esters (CE), that make them hydrophobic, and polar lipids at their surface, also including phospholipids as well as free cholesterol, that make them hydrophilic. Lipoproteins vary in their combination of core lipids or surface apolipoproteins. The four basic forms of lipoprotein are chylomicron, very lower density lipoproteins (VLDL), lower-density lipoprotein (LDLs), or high-density lipoproteins (HDLs) (HDLs).

C. Dyslipidemia Pathophysiology

Dyslipidemia, which is defined by a clustering of related plasma lipid and lipoprotein abnormalities, is the hallmark of the Mets. I increased FFA flux, (ii) elevated TAG level (hypertriglyceridemia), (iii) reduction HDL level, (iv) a predominance of small, dense LDL, but also (v) elevated apolipoprotein (apo) B level, with both the elevated plasma FFA concentration seen as the major cause in the growth of dyslipidemia aforementioned.

Hypertriglyceridemia is caused by obesity-induced secretion of adipocytokines, the most significant of which is tumor necrosis factors alpha (TNF-), which inhibits LPL in adipose tissue, leading to increased flux of adipose-tissue derived FFA towards the liver via the portal vein, stimulating the synthesis of hepatic TAG, and eventually increases VLDL particle secretion [14-19].

D. LPL (Lipoprotein Lipase):

1) Structure and Function of LPL

LPL is a 55,000-dalton glycoprotein with two structurally distinct domains connected by a flexible peptide: a large amino (NH2)-terminal domain and a smaller carboxyl (COOH)-terminal domain. The LPL structure was obtained from the three-dimensional crystallographic foundations of human pancreatic lipase (PL), which has a lot of disulfide linkage homology or sequence homology, especially in the catalytic region. LPL functional studies supported a revised LPL structural model that matched the homodimeric structure of the enzyme. In this model, which displays a head-to-tail arrangement of LPL monomers, the COOH-terminal domain on one subunit is close to the NH2-terminal domain catalytic cleft on the other subunit [20-22].

2) LPL Syntheses, Processes, Degrades, or Secretes

Notwithstanding the way that LPL action happens at the luminal surface of slim endothelial cells, vascular endothelial cells don't deliver it. Parenchymal cells produce useful LPL, which is in this way moved to the site of activity. The LPL mRNA was produced in the core of these parenchymal cells by the record of the LPL quality, which was therefore converted into an early polypeptide in the unpleasant endoplasmic reticulum (RER). LPL is at first delivered as an inert monomeric proenzyme in the RER. LPL is then remembered to be actuated on its course to the Golgi mechanical assembly, bringing about a functioning homodimer.

3) LPL regulation: Tissue-specific regulation

The LPL gene is found on chromosome 8p22 (short arm), which contains 10 exons as well as 9 introns but also spans roughly 30 kb. Even though LPL is expressed by just one gene, it has a tissue-specific expression pattern. As a consequence, LPL is very tissue-specific, shifting in response to tissue TAG needs as a consequence of physiological or pathological conditions.

During physiological conditions such as feeding, there is substantial LPL activity in the adipose tissue and minimal LPL activity in the muscles. As a result of this, TAG is partitioned to the adipose tissue for storage. Fasting, on the other hand, decreases LPL activity in adipose tissue while increasing it in muscle. Because of this inverse control of LPL, energy storage might well be enhanced when food is available, as well as energy available for muscle could be maximized during exercise. LPL activity is also elevated in brown adipose tissue during cold exposure to manage thermogenesis and even in the mammary gland throughout lactation to ensure that circulating TAG is used for dairy production [23-25].

E. Control of Transcription

When specific DNA molecules that bind (transcription factors) bind to cis-acting sequences in the regulatory regions of genes, ligand-mediated LPL gene transcription occurs.

Transcription factors including such peroxisome proliferator-activated receptor (PPAR) alpha as well as gamma bind to the PPRE motif as a PPAR/retinoid X receptor (RXR) heterodimer in response to fatty acids, glucose, fibrates, or thiazolidinediones (Nagao or Yanagita, 2008). PPARs have tissue-specific expression patterns based on their biological function. This leads to tissue-specific induction of LPL in tissues based on the location of the distinct PPARs. PPAR and PPAR activation might thus modulate LPL mRNA as well as exercise levels in a tissue-specific manner. In the presence of additional modifying cues, PPAR sites, on the other hand, may decrease LPL gene transcription.

F. Control of the Post-Transcriptional Process

Nutritional status, growth hormone, insulin, estrogen, glucocorticoids, or inflammatory mediators all have a role in LPL expression post-transcriptionally. These several components are identified to interact at various moments throughout the synthesis, processing, and translocation of LPL.

G. Obesity and LPL

Since LPL is a rate-limiting enzyme in the delivery of NEFA to muscles or connective fat, alterations in its tissues specific expression profile are expected to have an impact on obesity development and progression. Under particular genetic predispositions, a high ratio of adipose tissue (AT) to skeletal muscle (SM) reveals increased LPL activity in AT, which might also result in FFA being trafficked to AT, leading to obesity. It's known as the gatekeeper hypothesis. Low AT: SM-LPL activity, as per the substrate steal hypothesis, inhibits FFA from being delivered to AT, resulting in decreased fat deposition in AT. Since muscle clears roughly 30percent of circulating TAG, an enhancement in muscle LPL should aid in obesity prevention [26-28].

H. LPL's role in Dyslipidaemia

Three variables contribute to hypertriglyceridemia in dyslipidemia: Owing to TAG storage lipolysis through adipose tissue, I enhanced FFA flow to the liver, (ii) inhibited chylomicron or VLDL lipolysis owing to reduced LPL levels, or (iii) boosted hepatic de novo TAG production in the insulin resistance condition. Because LPL is required for the hydrolysis of TAG-rich lipoproteins as well as the generation of fatty acid, boosting LPL activity in either situation would reduce TAG and elevate HDL levels, relieving dyslipidemia. This is supported by the fact that patients with human mutations associated with higher LPL activity have lower TAG but also high HDL, protecting them against myocardial infarction, whereas peoples with familial LPL deficiencies, a rare genetic condition, have hypertriglyceridemic.

I. Drugs that increase LPL

The fibrate family of lipid-reducing drugs includes one of the most effective serum TAG reducing medicines. An increase in LPL activity mediates the hypotriglyceridaemic impacts of this, and also the thiazolidinedione (TZD) family of insulin-sensitizing medications. Both kinds of drugs activate the PPAR nuclear receptor family, increasing LPL mRNA. The fibrates work as PPAR agonists, reducing LPL

inhibition by reducing Apoc III level, whereas the TZDs act as PPAR agonists, enhancing LPL expression in rat adipose tissue but also improving FFA clearance. Fibers generate a slight decline in LDL cholesterol, which might be attributable to the reduction in serum TAG, in addition to their hypotriglyceridaemic effects. Anti-diabetes drugs called as TZDs also called glitazones, have been shown to reduce TAG levels in diabetic or non-diabetic rats and humans. In T2DM patients, rosiglitazone, for example, lowered postprandial FFA or TAG levels, including the increase in postprandial TAG in the VLDL1 %.

J. Glycyrrhizic Acid (GA)

Glycyrrhizic acid is the principal bioactive element identified in the roots of the plant's Glycyrrhiza glabra (GA). These roots, which also are called in Traditional Chinese medicine (TCM as 'Gan Cao' as well as licorice (licorice), give the licorice root its sweet taste (Isbrucker or Burdock, 2006). It has been used in the pharmaceutical, confectionery, or culinary sectors for over 4000 years.

K. Confectionery and the Use of GA for Medicinal Purposes

Licorice is mostly utilized in the culinary and confectionery sectors as a condiment. It's found in diet soda, sweets, chewing gum, gelatine, pudding, cream, and alcoholic drinks, among other things. GA's sweetness may help conceal the bitterness of medications. As a result, cough syrups, linctus, and other medications include GA in their ammoniated form.

1) GA Pharmacokinetics

GA is a saponin compound made up of glycyrrhizic acid (GE), a triterpenoid aglycone, coupled to glucuronic acid, a disaccharide. In both rats and humans, however, GA has a low oral bioavailability. GA is systemically deglucuronidated by intestinal flora (Eubacterium sp., Ruminococcus sp., or Clostridium inoculum) but also entirely absorbed as GE following oral ingestion. GA is only detected in rat or human plasma at significant oral doses of 50-500mg/kg and 100-1600mg/kg.

2) Glucocorticoids

The hypothalamic pituitary adrenal (HPA) axis regulates the release of glucocorticoids (GCs) from the zona fasciculate of the adrenal cortex. The catabolic activities of GCs are involved in the metabolism of carbohydrates, lipids, or proteins. They help the body deal with stress by mobilizing stored energy, and their activities are the polar opposite of insulins. By stimulating gluconeogenesis and glycogenolysis in the liver, GCs raise blood glucose levels. The catabolic action of GC on muscles and adipose tissues causes the release of amino acids and glycerol, both of which are used in the liver for gluconeogenesis. The downregulation of glucose transporters occurs as blood glucose levels rise. This explains why muscles and fat tissues have lower glucose absorption.

II. DISCUSSION

Lipids are hydrocarbon-based molecules required for the construction and functioning of living tissue. Fats, waxes, some vitamins oils, (such as A, D, E, or K), hormones, as well as the non-protein component of the cell membrane are all examples of lipids. The most prevalent types include fats or oils, waxes, phospholipids, as well as steroids. Triacylglycerols, commonly known as triglycerides, are something of energy that can be stored in the body. Fats are formed when fatty acids are mixed with glycerol or sphingosine. Triacylglycerols, sometimes known as triglycerides, are formed when glycerol is joined with three fatty acid molecules (also known as lipids). NMR Spectroscopy: NMR spectroscopy is frequently used to measure the overall lipid content of meals. The lipid content is determined by looking at the area underneath a peak in an NMR chemical shift spectra that correspond to the lipid fraction.

III. CONCLUSION

Under diverse physiological conditions, GA administration resulted in an increase in LPL expression throughout most tissues, but also a reduction in the size of adipocytes in both VAT and SAT. This might suggest that GA directs FFA away from pathological visceral depots but towards oxidative regions, facilitating healthy fat formation. Moreover, the smaller adipocytes seen in histological inspection might be the result of adipogenesis, but these smaller, more insulin-sensitive adipocytes could suck up circulating FFA more easily, reducing circulating FFA flow and enhancing insulin sensitivity.

GA altered blood lipid levels as well, with a comparable pattern of improvement within every lipid parameter, including serum FFA, TAG, total cholesterol, HDL cholesterol, or LDL-cholesterol, showing that GA might generate another less atherogenic lipid profile. Despite increased LPL expression, lipid did not accumulate in the non-adipose tissues examined, perhaps owing to an associated increase in ß-oxidation of LPL-generated FFA. As a result, GA may help to prevent the onset of IR linked to tissue steatosis and dyslipidemia. As a result, GA seems to be a promising drug for treating dyslipidemia by increasing tissue LPL expression, resulting in a favorable lipid profile and decreased tissue lipid buildup, as well as lowering hyperglycemia.

REFERENCES

[1] M. A. Kamal et al., “Tubulin Proteins in Cancer Resistance: A Review,” Curr. Drug Metab., 2020, doi: 10.2174/1389200221666200226123638.

[2] A. Agarwal, S. Agarwal, A. Verma, and R. C. Shukla, “Total basal ovarian volume as a marker of ovarian reserve: Myth or reality,” J. Clin. Diagnostic Res., 2018, doi: 10.7860/JCDR/2018/31482.11211.

[3] A. K. Srivastava et al., “Real time detection and quantification of Epstein Barr virus in different grades of oral gingivitis and periodontitis patients,” J. Exp. Ther. Oncol., 2018.

[4] Z. Chowdahry, S. Mehrotra, N. Swarup, F. Chowdhary, and A. Chowdhary, "Hemangioma-like telangiectatic granuloma: A diagnostic pitfall," J. Exp. Ther. Oncol., 2018.
[5] N. Swarup, M. T. Nayak, Z. Chowdhary, and S. Chandarani, "Necrotising sialometaplasia: A diagnostic perplexity? An innocent entity to malignant masquerade," J. Exp. Ther. Oncol., 2018.
[6] A. Remmerie and C. L. Scott, "Macrophages and lipid metabolism," Cell. Immunol., 2018, doi: 10.1016/j.cellimm.2018.01.020.
[7] M. M. Gaschler and B. R. Stockwell, "Lipid peroxidation in cell death," Biochemical and Biophysical Research Communications. 2017, doi: 10.1016/j.bbrc.2016.10.086.
[8] L. H. Wong, A. Čopič, and T. P. Levine, "Advances on the Transfer of Lipids by Lipid Transfer Proteins," Trends in Biochemical Sciences. 2017, doi: 10.1016/j.tibs.2017.05.001.
[9] Y. Sunami, A. Rebelo, and J. Kleeff, "Lipid metabolism and lipid droplets in pancreatic cancer and stellate cells," Cancers. 2018, doi: 10.3390/cancers10010003.
[10] A. Gupta et al., "Evaluation of microleakage with total etch, self etch and universal adhesive systems in class V restorations: An in vitro study," J. Clin. Diagnostic Res., 2017, doi: 10.7860/JCDR/2017/24907.9680.
[11] A. Singh, V. Goyal, and S. Goel, "Sputum collection and disposal perceptions and practices among pulmonary tuberculosis patients from Northern India," J. Clin. Diagnostic Res., 2016, doi: 10.7860/JCDR/2016/21673.8978.
[12] A. Agarwal, S. Mahajan, S. Verma, P. Bhardwaj, and G. Sharma, "Evaluation of the stress induced in tooth, periodontal ligament & alveolar bone with varying degrees of bone loss during various types of orthodontic tooth movements," J. Clin. Diagnostic Res., 2016, doi: 10.7860/JCDR/2016/17206.7235.
[13] P. Jain et al., "A comparative evaluation of the effectiveness of three different irrigating solution on microorganisms in the root canal: An invivo study," J. Clin. Diagnostic Res., 2015, doi: 10.7860/JCDR/2015/13133.6991.
[14] J. M. Dean and I. J. Lodhi, "Structural and functional roles of ether lipids," Protein and Cell. 2018, doi: 10.1007/s13238-017-0423-5.
[15] A. A. Domínguez-Avila and G. A. González-Aguilar, "Lipids," in Postharvest Physiology and Biochemistry of Fruits and Vegetables, 2018.
[16] E. Fahy et al., "A comprehensive classification system for lipids," J. Lipid Res., 2005, doi: 10.1194/jlr.E400004-JLR200.
[17] C. R. Santos and A. Schulze, "Lipid metabolism in cancer," FEBS Journal. 2012, doi: 10.1111/j.1742-4658.2012.08644.x.
[18] M. Raman, A. Almutairdi, L. Mulesa, C. Alberda, C. Beattie, and L. Gramlich, "Parenteral nutrition and lipids," Nutrients. 2017, doi: 10.3390/nu9040388.
[19] T. Skotland, K. Sandvig, and A. Llorente, "Lipids in exosomes: Current knowledge and the way forward," Progress in Lipid Research. 2017, doi: 10.1016/j.plipres.2017.03.001.
[20] R. Khatoon, N. Jahan, H. M. Khan, T. Rabbani, and S. Ahmad, "Evaluation of different staining techniques in the diagnosis of trichomonas vaginalis infection in females of reproductive age group," J. Clin. Diagnostic Res., 2014, doi: 10.7860/JCDR/2014/9765.5261.
[21] P. Singh et al., "Pharmacognostical study and establishment of quality parameters of aerial parts of Costus speciosus-a well known tropical folklore medicine," Asian Pac. J. Trop. Biomed., 2014, doi: 10.12980/APJTB.4.2014C1103.
[22] S. Chandak and C. M. Shetty, "Comparative study of dentascan and radiography for radiological evaluation of impacted teeth," J. Clin. Diagnostic Res., 2014, doi: 10.7860/JCDR/2014/7997.4618.
[23] H. Kaur, B. Singh, and A. Sharma, "Assessment of blood glucose using gingival crevicular blood in diabetic and non-diabetic patients: A chair side method," J. Clin. Diagnostic Res., 2013, doi: 10.7860/JCDR/2013/7705.3854.
[24] D. Agarwal, F. Huda, N. Arathi, and S. Awasthi, "A hydatid cyst of the appendix which mimicked a tubo-ovarian mass: A case report and review of the literature," J. Clin. Diagnostic Res., 2013, doi: 10.7860/JCDR/2013/5737.3264.
[25] S. Agarwal, D. Goel, and A. Sharma, "Evaluation of the factors which contribute to the ocular complaints in computer users," J. Clin. Diagnostic Res., 2013, doi: 10.7860/JCDR/2013/5150.2760.
[26] A. Mimery and S. De Clercq, "Primary pancreatic lymphoma presenting as recurrent idiopathic pancreatitis: a diagnostic dilemma," ANZ Journal of Surgery. 2020, doi: 10.1111/ans.15309.
[27] S. B. Singh and B. Singh, "Plasma malondialdehyde levels in age related macular degeneration," J. Clin. Diagnostic Res., 2010.
[28] T. Kumar and A. Takalkar, "Study of the effects of drinking water naturally contaminated with fluorides on the health of children," Biomed. Res., 2010.