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Review on nomenclature and classification of lipoxygenase and its medical applications

R Indhu sri[1], A Afrin[2], A Gowsalya[3], M Neevika[4], S Chozhavendhan[5], N Prabhu[6*]
[1-6] Department of Biotechnology, Vivekanandha College of Engineering for Women, Elayampalayam, Tiruchengode, Tamil Nadu, India

Abstract
Lipoxygenase (LOX) are dioxygenase that catalyze the formation of corresponding hydro peroxide from polyunsaturated fatty acid (PUFA) such as linoleic acid and arachidonic acid. However, in order to study and optimize its application in industrialized settings pure LOX isozymes are required. Such kind of enzyme also present in plants, animals, fruits and vegetables. Soyabean seeds are the richest known source of lipoxygenase, contributing up to 2% total protein content. The present study showed that the 3 lipoxygenase isozymes isolated from defatted soybean flour exhibited inhibition potential by modulating the enzyme to substrate ratio.The LOX enzymes is classified into plant lipoxygenase, human lipoxygenase and mouse lipoxygenase. The LOX enzymes is majorly found in soyabean seed. It took Biotechnological applications such as in baking and later flavor production. The LOX has an enormous functions and pathophysiological conditions, Alzheimer's disease and neuropathic pain and etc.., but it required in pure form. The nomenclature and classification of LOX was described in this article.

Keywords: Lipoxygenase, Hydroperoxide, Polyunsaturated fatty acid (PUFA)

1. Introduction
Lipoxygenase is an enzyme that is used to bring about specific biochemical reaction. This enzyme is produced by all living organism which acts as catalyst. Lipoxygenase it comes under the family of (non-heme) ion group (EC 1.13.11.12, LOX). This enzyme catalyst the deoxygenation of poly unsaturated fatty acids compared to carbon dioxide, lupines albus consumed large quantities of oxygen, in presence of lipids [1, 2, 3]. Craig designated the unsaturated fat oxidase to the responsible enzyme. Plants contain proliferate Isoforms of the LOX enzyme which differs in substrate preference, optimal ph., product formation and stability [4, 5, 6, 7]. High level of Soyabean LOX expression, most knowledge in the enzymology and structural biology of lipoxygenase enzyme are derived more from the studies on soyabean LOX isoforms.
LOX is classified into two types
"Type 1" (mainly extra-plastidial) and
"Type 2" (mainly plastidial),

LOX enzyme is helpful in pharmaceutical process due to their act in inflammation process. The LOX enzyme plays a major role in baking industry. It has baking property of wheat flour dough. Soyabean seed is the major source for the production of LOX enzyme. LOX enzymes consist of 2% of protein content in it. The balls another in 1943 was first extracted the LOX enzyme from soya bean enzyme. LOX enzyme play vital role in its medical properties such as Alzheimer's disease, neuropathic pain and inflammation.

2. Nomenclature
The nomenclature of this enzymes were1st described in the 1st half of the 20th century and the family diversity has expanded to include both the animal and plant kingdoms[1,10].LOX isozymes was named earlier based on their comfort of purification, stability, and optimal pH of catalysis. Multiple isoforms of the LOX enzyme produced by the higher plants. These isozymes distinct in position of optimal pH of catalysis, substrate preference, region specificity, and has the capability in carotenoids pigment bleaching [11, 6, 9, 6]. C18-polyunsaturated fatty acids, linoleic and α-linoleic acid are natural substrate for plant LOX [7, 11, 12]. The nomenclature for LOX enzymes is therefore based on the specificity of the enzyme acting on its substrate [7]. The LOX-1 enzyme is the designated 13-LOX. However, soybean seed LOX- 2, a 9/13LOX, produces equal amounts of both 9S-HPODE and 13S– HPODE [9, 13]. This isozyme is besides unique +++in its knack to utilize esterified unsaturated fatty acids in membranes, when compared to LOX-1 which has an absolute requirement for free fatty acids [14, 9]. LOX-3, also conversely, has a reasonable preference for the production of 9S-HPODE which is resulting in the classification as a 9-LOX. By comparing animal LOX with its plant complements, the chain lengths of the natural substrates (arachidonate vs. linoleic acid) result in the plant 13-LOX equivalent to a 15-LOX in animals [6].Finally, both enzymes act on the ω-6 position of the fatty acid chain. This system of nomenclature has, produces some confusing with growing family diversity. The major reason for this confusion is that the current nomenclature in that period did not take evolutionary and functional similarity into account [15, 16]. Hence, it becomes especially superficial when comparing plant and animal LOX enzymes, since they did not use the similar substrates. Further complications were found when multiple isoforms are compared, for example, mammalian 12-LOX, are present in the same organism [6, 17]. So, the classification procedure which is based on phylogenetic relatedness was established by Ivanov and others (2010). However, there is no blending of LOX nomenclature, which could overcome these difficulties, that method was proposed. Nowadays, different LOX isozymes, catalyzing the same reaction in the

same organism, which is named after the prototypical tissue of their existence with allusion to their regiospecificity [6,18]. For example, there are 3 isoforms of mammalian 12-LOX. These enzymes are therefore designated platelet, leukocyte, or epidermal 12-LOX [6], based on the studies using soybean LOX isoform.

3. Lox- family and its types

Lipoxygenase enzyme are present in both plants and animals (EC 1.13.11.12). They are found in Eukaryotes(plants, fungi, animals, protists);otherwise the third domain of terrestrial life, the archaea, which possess proteins with approximately 20% of amino acid sequence. These proteins lacked iron binding residues, so they are not projected to possess LOX activity. These enzymes are widely present in plants where they are involved in a different number of aspects in plant physiology including growth and development, pest resistance, and senescence to wounding. In mammals number of LOX isozymes are present in the metabolism of eicosanoids (such as prostaglandins, leukotrienes.

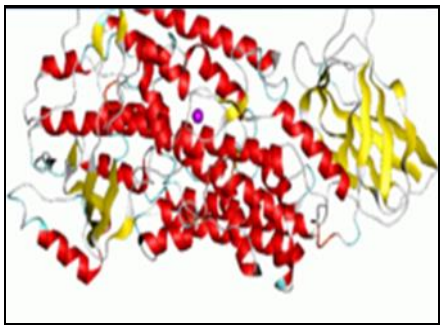

Fig 1: Common structure of lipoxygenase

3.1 LOX in Plants

LOXs are normally present in the seeds. Otherwise, LOXs do not have a clear physiological role in seed development, then it is denoted as that in a soybean line lacking the three seed LOX isozymes, it means that there is no effects on crop performance were detected when compared with a normal line. Hence it supports the seed LOXs in the function of proteinstorage.

Plants express a variety of cytosolic lipoxygenases (EC 1.13.11.12) which is seems like a chloroplast isozyme. Plant lipoxygenase in combination with hydroperoxide lyases are responsible for many fragrances and other signaling compounds. One of the example is cis-3-hexanal, the odor which is like a freshly cut grass.

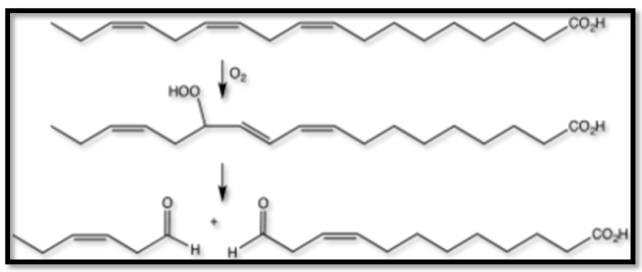

Fig 2: cis-3-hexenal is generated from linolenic acid to hydroperoxide by the action of LOX which is followed by lyase

3.2 Human lipoxygenases

Except the 5-LOX gene which is located on chromosome 10q11.2, all the other six human LOX genes are located on chromosome 17.p13. A single protein chain was coded about 75-81 kilo Daltons and consisting of 662-711 amino acids.Mammalian LOX which contains 14 exons with exon/intron boundaries are as follows:

- ALOX5
- ALOX12
- ALOX15
- ALOX15B

Similarly, the LOX genes which contains 15 exons are as follows

- ALOX12B
- ALOXE3

These boundaries are positioned at highly conserved region. The 6 human lipoxygenases along some significant products which will make genetic diseases.

3.3 Alox5

The ALOX5 gene expression is transcriptionally regulated at a basal level. It is regulated by external stimuli. A potent LOX-5 inhibitor zileuton introduced in the USA blocks an extensive amount of cysteinyl leukotriene fabrication for a short period of time. even though the occurrence is low, a genetic E254K (760G>A) mutation in ALOX5 has been reported in bronchiolar asthma patients. This mutation causes an alteration in the electronic charge of the C-terminal catalytic domain from negative to positive, implicating defective changes in enzymatic activity and protein interaction. Mutations in the Sp1 binding site in the ALOX5 promoter has been associated with air way hyper resonsiveness, but not with asthma. Some evidence Suggests that low 5-LOX expression in tumors found in human might lead to greater 15-LOX expression followed by cancer formation through impaired apoptotic activity [31].

4 Disease caused by alox5

4.1 Inflammation

Immune cells precise leukotriene receptor 1 and 2 as well as cysteine leukotriene receptor 2; as such, the role of ALOX5 could account for the relocation of these immune cells[33].as expected, neurophilic tenderness is one of the fallacious phenotypes in ALOX5-defecient mice.In an ovalbumin-induced asthma model,Alox5-deficient mice exhibited a suppressed methacholine-induced response to air way hyper responsiveness with impaired eosinophilic inflammation in the lung Thus, the production of lipid products from 5-LOXplays an important role under physiological conditions, and its level is forcefully regulated by the balance between LOX and other enzymes, such as COX, as shown in an earlier study.

4.2 Atherosclerosis

Separately from responses in asthma and neutrophilic swelling, research showing that 5-LOX plays a key role in the beginning and/or growth of atherosclerosis. Diminished ex-pression of functional 5-LOX in LDL receptor-deficient mice hasrevealed stifled atherogenesis, proposing that 5-LOX plays a causative role in this disease.Steadily, alternative atherosclerotic ideal convinced by COX-2 interruption lessened disease formation inAlox5 deficient mice. Given that LOX-15 is involved in atherosclerosis, these studies provide examples that atherosclerosis can be

induced by lipid peroxidation products formed from any isoforms. This finding is entirely reliable with clarifications representing that antioxidants generally reveal defensive effects in untried models

4.3 Neuronal disorder

Alox5is recognized to be extremely conveyed in neuronal tissue, chiefly in Alzheimer's disease; thus, its role in neuronal disorders has been dynamically categorized. A recent study testified that matured female Alox5-deficient mice revealed protective effects against disquiet-like behavior on a C57BL/6 genetic background, nurturing the chance that Alox5 could modulate neuronal function. A subsequent study using a transgenic mouse model of Alzheimer's disease demonstrated the efficacy of a 5-LOX inhibitor zileuton and hypothesized that Alox5 could facilitate the initiationor progression of this disease. Using such a disease model, Alox5 defficiency consistently improved disease phenotypes

4.3 Tumor

Colorectal cancer is frequently caused by transmutation of the tumor suppressor Adenomatous polyposis coli (APC) gene. Among man mouse models produced by Apc mutations, ApcΔ468mice precisely tolerate a shortened Apc gene that develops severe polyposis by four months. Amusingly, immune histochemistry showed an increase in Alox5 expression inApcΔ468mice, signifying that LOX-5 influence donate to tumorigenesis in colorectal cancer. Mast cells play an essential role in the development of colorectal cancer in this animal model, as they induce epithelial proliferation. Reliably, the number of mast cells increased in APCΔ468 mice compared to WT controls. In this model, a deficiency in Alox 5 led to impairment, implying that LOX5 acts as an important role in colorectal tumorigenesis

4.5 ALOX12

Since the discovery of the high expression of LOX in platelets, the mechanism of its expression has been studied in detail. A previous study reported that ALOX12expression was attenuated in platelets by the haplo deficiency of RUNX1, a hematopoietic transcription factor associated with familial thrombocytopenia, platelet dys function, and a predisposition to acute leukemia in patients with thrombo cytopenia ALOX12 expression is also precise epigenetically, as indicated by the increase in DNA methylation ofALOX12genes in myelodys plastic syndrome and acute myeloid leukemia patients with mega karyocytic dysplasia. It has been suggested thatALOX12is associated with diminished bone mineral density as well. Given that 12-LOX produces endogenous lipid ligands for nuclear receptors, such as PPAR-γwhich facilitate adipocyte differentiation from mesenchymal stem cells, the number of osteoblasts decreases, followed by impairment of bone mineral density. An earlier study suggested that the 12-LOX-mediated pathway. The best-studied example includes mutation of E261R (8354G), which causes an increase in 12-LOX activity, with a potential link too oesophageal squamous cell carcinoma. This mutation has also been associated with colorectal cancer and breast cancer. An in vitro and in vivo study has shown that 12-LOX plays a vital role in the propagation and antiapoptosis of hepatocellular cells, suggesting that this carcinogenic function of ALOX12 requires endogenously generated lipid mediators

4.6 Alox15

It is widely accepted that cyclooxygenase (COX) inhibitor non-steroidal anti-inflammatory drugs (NSAIDs) induce colon cancer inhumans. One recommended reason is that the balance between COX and LOX determines tumorigenesis critically. Under low COX activity, arachidonic acid released from cell membranes in response to external stimuli is preferentially metabolized by LOXenzymes. There is evidence that a 15-LOX metabolite 13S-HPODE(13S-hydroperoxyoctadecaenoic acid) generated from linoleic acid induces apoptosis in colon cancer cells; thus, defective expression of ALOX15 in colon cancers could promote tumorigenesis.ALOX15 expression itself is controlled, at least in part, by the epigenetic process, as an alteration of methylation in the ALOX15 promoter has been observed in prostate cancer patients Furthermore, the expression of 15-LOX in epithelial cancer cells is tightly regulated by additional mechanisms. As mentioned, STAT6 is a critical regulator ofALOX15expression regulated by its phosphorylation and acetylation, as well as histone modification. Recent studies have also shown the ALOX15 expression can be modulated by the chromatin-dependent STAT6-independent mechanism. Biochemically, the produced 13 S-HPETE interacts with PPAR-δ, followed by the induction of apoptosis prior to carcinogenic conditions. The importance of 15-LOX-derived metabolites has also been defined by its aberrant failure in conditional transgenic mice, expressing it in the mouse prostate, inducing prostatic intraepithelial neoplasia once the apoptotic function is dys regulated.

4.7 LOX15B

Although a link between atherogenesis and ALOX15 expression has long been hypothesized, it is known that the expression of ALOX15B in human carotid plaque macrophages is higher compared to ALOX15. An in vitro experiment of ALOX15B silencing reported an attenuated lipid accumulation in human macrophages, indicating that it is functional for lipid uptake into the cells. Thus, it is suggested that ALOX15B plays an important role in the initiation and development of atherosclerosis in humans. Numerousreadings have shown the down regulation of ALOX15B in epithelial tumors, suggesting that ALOX15B has an antiproliferative role. This reduction of 15-LOX-2 in tumor cells was restored by an inhibitor for COX enzyme, demonstrating that its expression is negatively regulated by prostaglandins, at least in part. PPAR-γ, a nuclear receptor regulated by endogenous LOX products, was upregulated in some epithelial tumors, suggesting that the downregulation of ALOX15B is autonomously controlled by PPAR-γin epithelial cancer cells. In prostate epithelial cells, ALOX15 expression is positively regulated by transcription factorSp1, whereas transcription factor Sp3, which is closely related to Sp1, negatively regulates its expression, suggesting that ALOX15B expression is critically regulated by multiple regulators. Apartfrom epithelial cells, a separate study demonstrated increased ALOX15B expression in tumor-associated macrophages from renalcell carcinoma, suggesting that ALOX15B expression is distinctly regulated in epithelial cancer cells and macrophages. Similarto tumor-associated macrophages, the upregulation of ALOX15B incarotid plaque macrophages has also been described.

4.8 ALOX12B

ALOX12B gene deliversguidelines for making an enzyme called 12R-LOX. This enzyme is part of a group of enzymes called arachidonate lipoxygenase. Most of these enzymes help add an oxygen molecule to a particular fatty acid called arachidonic acid.Arachidonate lipoxygenase add oxygen molecule at different locations on the arachidonic acid molecule, producing a variety of substances called fatty acid hydroperoxides. The fatty acid hydroperoxide are then processed into molecules that play an important role in chemical signalling within cells. Precisely, the 12R-LOX enzymes helps add an oxygen molecule to arachidonic acid to make a substance called 12R-hydroperoxyeicosatetraenoic acid (12R-HPETE). 12R-HPETE is later converted to a signalling molecule that is involved in the formation of the layers of fats(lipids) within the outermost layer of the skin(the epidermis). The lipid layers are necessary to prevent water loss(dehydration) through the skin. Genetic failure of this method leads to ARCI, a heterogeneous skin disease characterized throughlumpy and scabby skin, with anoccurrence of one in 200,000 newborn over the world. the affected skin commonly improves during either childhood or puberty, and they have a normal life span. Among various ARCI diseases, ALOX12B is main cause, but not solely. Mutations, mostly found as missense, termination, and frame shift in ALOX12B, are widely found in its entire molecule, encompassing both in a C-terminal catalytic domain and an N-terminal β-barrel structure. Some mutants have lost enzymatic activity, as established by biochemical assays.

4.9 ALOXE3

Hence, in genetics many mutations in ALOXE3, along with ALOX12B, have been found in ichthyosis. The upshot of disease in ALOXE3 variants seems to be analogous to that found in ALOX12B variants, therefore it showing perfectly that is sequential oxidation by ALOXE3 and ALOX12B are equally imperative. As a substrate, it knows that the LOXE-3favors oxygenated lipids like12R-HPETErather than un oxidized compound such as arachidonic acid. Except ARCI,other diseases are associated with ALOXE3 variants.

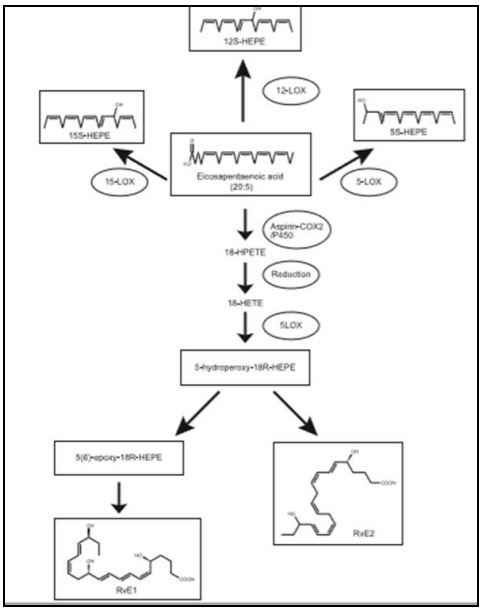

Fig 3: Formation of resolvins from eicosapentaenoic acid:

4. 10 Mouse lipoxygenase

The mouse is a mutual model to analyze lipoxygenase function. Though, there are some vital differences between the lipoxygenases between mice and men which make extrapolations from mice studies to humans difficult [32]. In comparing, the 6 functional lipoxygenases in humans and mice have 7 functional lipoxygenases. I that some of the final one have distinct metabolic activities than their human orthologs. In particular, mouse Alox15, unlike human ALOX15, metabolizes arachidonic acid mainly to 12-HpETE and mouse Alox15b, in contrast to human ALOX15b, is predominantly an 8-lipoxygenase, metabolizing arachdionic acid to 8-HpETE;hence there is no comparable 8-HpETE-forming lipoxygenase in humans [22]. The following some of the genes shows the comparison with with humans:

- Alox5
- Alox12
- Alox15
- Alox15b
- Alox12e
- Alox12b
- Aloxe3

Alox5

Appears to be comparable in function to human ALOX5.

Alox12

They differs from human ALOX12, which favorably metabolizes arachidonic acid to 12-HPETE, but also the considerable amounts of 15-HpETE, in that which metabolizes arachidonic acid almost exclusively to 12-HpETE.

Alox15

They differ from human ALOX15, which under standard inspect conditions metabolizes arachidonic acid to 15-HpETE and 12-HpETE products in an 89 to 11 ratio, which metabolizes arachidconic acid to 15-Hpete and 12-HpETE in a 1: 6 ratios, i.e. its principal metabolite is 12-HpETE. Also, the human ALOX15 prefers linoleic acid over arachidonic acid as a substrate, metabolizing it to 13-HpODE while Alox15 has little or no activity on linoleic acid. Alox15 can metabolize polyunsaturated fatty acids that are esterified to phospolipid and cholestrol which is cholestrol ester. This property along with its dual specificity in metabolizing arachidonic acid to 12-HpETE and 15-HpETE are similar to that of human ALOX15 and has led to both enzymes being termed 12/15-lipoxygenases.

Alox15b

In compare to ALOX15B which break down arachidonic acid principally to 15-HpETE and to a lesser extent linoleic acid to 13-HpODE, metabolizes arachidonic acid principally to 8*S*-HpETE and linoleic acid to 9-HpODE. Alox15b is as useful as ALOX5 in metabolizing 5-HpETE to leukotrienes.

Alox12e

It is an ortholog to the human ALOX12P gene which has suffered damaging mutations and is not expressed. ALox12e desires methyl esters over non-esterfied polyunsaturated fatty acid substrates, metabolizing linoleic acid ester to its 13-hydroperoxy counterpart and to a lesser extent arachidonic acid ester to its 12-hydroperoxy counterpart.

Alox12b

It appears to act similarly to ALOX12B to metabolize the linoleic acid moiety of EOS to its 9*R*-hydroperoxy counterpart and thereby contribute to skin integrity and water impermeability; mice depleted to Alox12b improve a severe skin defect similar to Congenital ichthyosiform erythroderma. Unlike human ALOX12B which cam metabolize arachidonic acid to 12*R*-HETE at a low rate, Alox12b does not metabolize arachidonic acid as free acd but dose metabolize arachidonic acid methyl ester to its 12*R*-hydroperoxy counterpart.

Aloxe3

It appears to act equally to ALOXe3 in metabolizing the 9*R*-hydoperoxy-linoleate derivative of EOS to its epoxy and keto derivatives and to be involved in maintaining skin integrity and water impermeability. AloxE3 deletion leads to a defect similar to congenital ichthyosiform erythroderma.

5 Applications

5.1 Asthma

Highly increased levels of LTC4, LTD4, and LTE4, which are 5-LOX metabolites, have been observed in lung tissues that were challenged with allergens. Up-regulation of these mediators is considered as the main cause of asthma since leukotrienes are potent regulators for smooth muscle contraction in broncho constriction [37]. In addition, cysteinyl leukotrienes can cause plasma leakage from post-capillary venules in respiratory tissues, which can lead to inflammatory edema [33]. In addition, it has been shown that the expression of 15-LOX in lung epithelial cells activates the NF-κB pathway, which demonstrate a connection between LOX activity and NF-κB activation. These findings indicate that the modulation of the assemb, ly of pro-inflammatory leukotrienes victimisation tiny molecule inhibitors has potential for treatment of bronchial asthma.

5.2 Cardiovascular Diseases

Lipoxygenase activity has been concerned within the pathologic process of vas diseases like hardening of the arteries[36]. Lipoxygenases, as aerobic enzymes, are believed to have an important role in the oxidation of low density lipoproteins (LDLs) in macrophages to form foam cells. The formed foam cells will develop plaques of atheroma and their accumulation in the arteries leads to atherosclerosis [36]. In addition, a rise of the 5-LOX metabolites cysteinyl LTE4 levels in urine and LTB4 within the fatty tissue were ascertained in patients with hardening of the arteries. In addition, it's been shown that the 15-LOX-1 and 15-LOX-2 matter 15-hydroxyeicosatetraenoic acid (15-HETE) promotes arterial blood vessel inflammation via activation of the NF-κB pathway, which leads to increased expression of the 15-LOX enzymes during a regeneration loop. This demonstrates that inhibition of lipoxygenase activity will offer a treatment strategy for this disorder

5.3 Rheumatoid Arthritis

Since 5-lipoxygenase is the main catalyst for the formation of LTB4, its role in the development of rheumatoid arthritis becomes apparent with the identification of high LTB4 levels in the synovial fluid of arthritis patient. This leukotriene is produced mainly by neutrophils, which are the most abundant leukocytes in rheumatoid joints. A crucial role of LTB4 during arthritis induction and severity has been disclosed in a mouse body fluid transfer model of inflammatory inflammatory disease[34]. Importantly, the inflammatory responses are reduced in mice with 5-LOX and leukotriene A4 hydrolase enzyme deficiency. In accumulation, another type of lipoxygenase, namely 15-lipoxygenase, is also involved in the pathogenesis of rheumatoid arthritis via the NF-κB pathway [35]. It has been represented that the 15-lipoxygenase matter, 15-(S)-HETE increases the IκBα degradation and the nuclear translocation of NF-κB subunit. It has been ascertained that the NF-κB pathway is activated within the early stage of joint inflammation and NF-κB DNA binding activity is enhanced in atrophic arthritis patients [35]. These results indicate NF-κB activity and LOX activity are also closely linked in rheumatoid arthritis and that inhibition of lipoxygenases could also find a therapeutic application in this field.

5.4 Inflammatory Bowel Disease

The role of leukotrienes in inflammatory viscus malady (IBD) has been explored. A colonic check diagnostic assay} test from patients with IBD showed 3-7-fold improvement of 5-lipoxygenase, FLAP and LTA4 hydrolase expression within the colonic mucous membrane and also the body part dialysates, which form the cellular basis for LTB4 synthesis[33]. More recently, Cys-leukotiene E4 (LTE4) was considered as a biomarker for IBD since the urinary excretion of LTE4 was significantly increased in patients with IBD. All these data together suggest that inhibition of lipoxygenase activity and leukotriene bio-synthesis can be a valuable approach for treatment of such inflammatory diseases.

5.5 Lipoxygenase in Cancer

Lipoxygenases and their catalysis products are associated with carcinogenic processes such as tumor cell proliferation, differentiation, and apoptosis. Several lines of evidence have proven the crucial role of lipoxygenases in cancer. In human prostatic adenocarcinoma cells, the overexpression of platelet 12-lipoxygenase (p12-LOX) has been observed, which is a trigger for angiogenesis and tumor growth. The increased expression of the 5-LOX enzyme and the LTB4 receptors were observed in pancreatic cancer. In addition, 5-LOX expression levels were suggested as indicator for early neoplastic lesions. Leukotriene LTB4 could be a potential stimulator for neoplastic cell growth and additionally plays a job within the formation of ROS in response to drive. It has also been shown that the 5-LOX metabolite LTB4 is capable of activating the transcription factor NF-κB in cancer cells, which suggest a tumor promoting role via this route. The roles of 15-LOX-1 metabolites square measure according within the development of carcinoma by promoting the invasion of neoplasm cells into the humor vessels and also the formation of bodily fluid node metastasis. In colon cancer cells it has been shown that 15-LOX-1 expression suppresses the metastatic phenotype of these cells and this enzyme is linked to increased NF-κB transcriptional activity [31]. Contrary to a neoplasm promoter role of 15-LOX-1 a neoplasm suppressor role of 15-LOX-2 has been represented in glandular carcinoma. For 15-LOX-2, however, no reference to NF-κB communication has been represented to date [32]. These studies indicate that the lipoxygenase expression is related to the event of cancer. For 5-LOX and 15-LOX-1 the activity is connected to NF-

κB activity, whereas such a connection has not been described for the other lipoxygenases. Taking all this proof along, lipoxygenases square measure AN rising cluster of cancer targets.

6. Conclusion

The role of LOX in human, plants and mouse have been studied extensively. In order to develop new medicinal products, for achieving higher rates and levels of extraction, or improve quality of drugs or medicines in terms of its purity. Without knowing the roles of lox in that organism, the cure of diseases which related to lipoxygenase is difficult. Hereby, the lox containing organism, its types and the disease caused was described widely.

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