OPEN ACCESS Freely available online

Organic Chemistry: Current Research

Review Article

Mitochondrial Inner Optic Atrophy 1 (*opa1*) Gene is Necessary for Regulating and Activating Lysosome, related Orphan Receptor a (*rora*) Genes, and *Apol1* Gene Involved in Autophagy Cells for Anti-Inflammation Processes, where *ror-Rlpha* Genes Stored as Lysosomal Security Granules within Autophagy Cells

Ashraf Marzouk El Tantawi*

Department of Biomedical molecular studies, Goldwin Avenue west, Toronto, Canada

ABSTRACT

Tumor Necrosis Factor alpha (TNF-α) subunits deficiency or the involvement in will lead to Sickle Cell Disease (SCD) which marked by a phenotypic variability and inflammation plays the major role in SCD pathophysiology, which linked strongly to *RORA1* genes expression and functions, and also linked to TNF-α subunits expressions and. Activities, where TNF-α subunits are so essential for anti-inflammation processes and linked to ROR-α genes activities and functions, and necessary for regulation of bone homeostasis in several chronic immune and inflammatory joints and tissues diseases.

The inhibition of TNFα due to inhibition or variations in *ROR-alpha* genes lead d to significant inflammations improvements involved in SCD pathophysiology , and also will lead to increasing in Nuclear Factor-κB pathways (NFκB) catabolic pathways and any remaining of TNF-α will be involved in the NF-κB signaling pathway due to inhibition in their mitochondrial activities.

The inhibition or deficiency in presence of Thymine in the Related Orphan Receptor-A (*RORA*) genes can reflect deficiency in mitochondrial synthetase enzyme (where mitochondrial *OPA1* gene depending on ribosomal genes activities), that'll lead to down activities in *ROR - alpha* genes functions, and redu ctions in TNF -α, TXA2, and in VEGF-A subunits.

RORA genes necessary for lipid metabolism where is controlled by mitochondrial phospholipase enzymes productions, similarly, *ROR -α*Genes is necessary for promotion and regulations of hepatic glucose metabolism and is necessary for hepatic activities and for sestrin-Leu carrier synthesis and activities during hepatic metabolic activities. Lysosome and *ROR-α* genes are having so necessary functions of preventing G-protein aggregates associated with neuropathies, and preventing blood platelets aggregations depending on mitochondrial activities through producing its active inflammatory enzymes for acting on any toxic inflammation or on any aggregation for producing the active TXA2 subunits which through feedback will reforme VEGF-A alpha subunits where can be stored in or as lysosomal secretory granules.

Keywords: Endosomes tissue cells; Lysosome; Tetraspanins; Tumor necrosis; Related orphan receptor; Nuclear factor

INTRODUCTION

Endosome and lysosome compartments are characterized by their morphological and functional properties, that their healthy compositions and nutritious can be healthy. For inner cells components, or can be un-useful or toxic to living cells depend on its molecular compositions and their main feedback origin. Endosomes are typically involved with the filtering and delivering of lipid vesicles including sorted digested amino acids to and from the plasma membrane then to inner cells components. Early endosomes in young ages are close to sites of active endocytosis, where they can act as a recycling or re-functioning and transport

Correspondence to: El Tantawi AM, Department of Biomedical molecular studies, Goldwin Avenue west, Toronto, Canada, Email: Ashraf012345f@gmail.com

Received: February 01, 2021, **Accepted:** February 15, 2021, **Published:** February 22, 2021

Citation: El Tantawi AM (2021) Mitochondrial Inner Optic Atrophy 1 (*opa1*) Gene is Necessary for Regulating and Activating Lysosome, related Orphan Receptor a (*rora*) Genes, and *Apol1* Gene Involved in Autophagy Cells for Anti-Inflammation Processes, where *ror-Rlpha* Genes Stored as Lysosomal Security Granules within Autophagy Cells. Organic Chem Curr Res. 10:206.

signals genes and vesicles budding compartments molecules to and from plasma membrane (which are formed from inner cells) to other specific specialized tissue cells. Thus, endosomes provided with a sorted lipid, protein, and extracellular molecules to be filtered and transmitted for cells metabolism. Endosomes, have the transport growth factors and signals transmission functions along the axon, provide a rapid genes signals propagations and regulations of signaling cues.

Endosomes are a type of extracellular gene that originally in young ages are a active micro alpha subunits where can stimulate PPARs genes from plasma for antigen synthesis and for inner cells nourishing, and are responsible for inner cell nutritious and protections including anti-inflammatory effects and more as receiving molecules to be filtered for inner cells metabolism and then transmitted as active signals transmission, but as age increased as the composition of endosomes from fatty acids and amino acids will increased and contaminated that will include necessary and unnecessary fatty and amino acids and may include +ve cationic molecules that will delay many of endosomes functions and activities [1-3].

OPA1 genes is the main gene in mitochondria, that when stimulated for repair inner and outer mitochondrial membrane will cleavage to produce L-*OPA1* & s-*OPA1* genes, where s-*OPA1* is involved in the outer membrane synthesis upon fusion with MN 2 gene for activating mitochondria. Where, decreasing in mitochondrial activities will cause a variants of syndromic intellectual disability with either autism or cerebellar ataxia.

ROR-alpha genes are necessary for *CYP7A* synthesis, which are locally produced in brain and in liver, where necessary in brain to regulate the synthesis of neurosteroids, where in liver is acting for catalyzing the hydroxycholesterol in the regulations of cholesterol synthesis. Where a 7 alpha-hydroxylation is necessary for conversion of both cholesterol and 27-hydroxycholesterol into bile acids, and the *RORA* genes are necessary for *CYP7A* genes productions and activities which are necessary for the conversion of both cholesterol and 27-hydroxycholesterol into bile acids. *RORA* genes considered is imp for controlling the conversion of cholesterol and 27-hydroxycholesterol into bile acids and are so essential for liver protections and are so necessary for protection from sickle cells diseases. Also, deficiency in *ROR-a* gene activities will reflect decreasing in *CYP7A* gene activities, and will lead to decreasing in bile acids and lead to decreasing in liver protections from fibrosis and from inflammation [4].

Also, the high decreasing in *RORA* genes in human will lead to dramatic increasing in basal expression of *NF-κB* regulated genes, due to the high decreasing in *TNFα* and in VEGF-A subunits productions.

METHODS

Tetraspanins characteristically containing 4, 6 or 8 conserved cysteine residues, which indicate that the Cys (TGT, TGC) amino acid and is the main imp amino acid for slowing and control tetraspanins activities in distinct way where Cys present at the head of the game then Thr at the tail of the gene will bonded together for converting the gene to the circled round inactive or in steady position shape, and that bonds only will be broken by phosphorylation process and by increasing polarities by ATPase for practicing their functions. The tetraspanins genes activities can be stopped or slow down by constructing that linkages between Cys and Thr amino acids, or and between Pro and Gly, and between Tyr and Leu amino acids respectively, and then will cause twisting, bending and rotating to the gene to slowdown its activities, but to be activated again those linkages between those amino acids have to be broken down to let and allow the gene to be in active straight linear forms again and that will be done by phosphorylation process whether by ribosomal ATPase or by G_actin ATPase or through MAPK pathways.

Those necessary amino acids Thr, Tyr, Gly, Gly, Leu and Cys are depending on their pyrimidine functions where their re-synthesis are depending on and controlled by mitochondrial synthetase enzyme which regulates the conversion purines to pyrimidine.

The presence of active cysteine with Tyr, Gly, Gly Leu and Thr amino acids in the exosomes and in Tetraspanins.

Are so necessary directly or indirectly for VEGF-A subunits re-synthesis, and for TNF synthesis, consequently for endothelin-1 synthesis, where, Cys, Arg, Tyr, Gly, Gly, Phe, Leu, Ser, Thr are necessary for anti-inflammation cycles through the effect of mitochondrial anti-inflammation enzymes (synthase, phospholipase, Cox2) on inflammations molecules to produce TXA2 subunits, which through feedback will re-produce VEGF-A alpha subunits, and then will resynthesis endothelin-1 during anti-inflammations cycles and processes, then will re-stimulate PPARs genes activities with MAPK pathways for completing the rest of complete cycles [5].

Also we know that Thr with Cys and Ser with Arg amino acids are so necessary for completing phosphorylation processes in several genes during MPAK pathways which controlled and regulated by ribosimal ATPase activities.

Indosomes are imp for regulate metabolic cycles through their specific amino acids arrangements *to* regulate and running anabolic processes and regulate transmitted genes as signals across its polar active receptors in a controlled limited steps depending on ribosomal activities and on surrounding stimulating active sources and factors.

Retinoic Acid-Related Orphan Receptor *α* (*RORα*) activities has a major roles in various biological metabolic processes including the anti-inflammation processes, and involved in autophagy cells, where, Retinoic Acid-Related Orphan Receptor *α* (*RORα*) controls the inflammatory signaling network [6].

RORA genes involved in lipid metabolism where is controlled by mitochondrial phospholipase enzymes (which is necessary for activating *APOL1* gene which involved in autophagy cells synthesis) for producing apolipoproteins, where *ROR-alpha* genes are produced by autophagy cells for acting on inflammations, and also *ROR alpha* genes involved in the regulation of hepat ic glucose metabolism, where is necessary for hepatic activities and is necessary for sestrin-Leu carrier synthesis and activities during hepatic metabolic activities, that is necessary for *PPARs* genes activation.

Also *ROR-alpha* genes activities depending on thymine nucleotide resynthesis which regulated by and depending on mitochondrial synthetase enzyme which regulate the pyrimidine synthesis. Where, the most necessary amino acids in *ROR-alpha* gene are Ser, Arg, Tyr, Gly, Gly, Phe Leu, and Thr amino acids, that are necessary for TXA2 and then for VEGF-A subunits productions and activities, where any deletion within the *ROR-alpha* gene leads to an over-expression of inflammatory cytokines, that reveals the roles of

RORA genes activities in regulations the anti-inflammations processes and cycles.

Lysosomes functions are so essential for degrading G-protein aggregates associated with neuropathies such as Huntington's Disease (HD) and are important for regulating cholesterol homeostasis, where *RORA alpha* genes are also necessary for regulating cholesterol too through conversions cholesterol to bile acids and protecting liver from fibrosis and from inflammation thus *RORA genes* has strong roles in anti-inflammatory cycles, that indicating that links in function between lysosomes and *ROR* families [7].

ROR upregulated antioxidative and anti-inflammatory genes, which ameliorated the symptoms of NASH in the methionine and choline deficient, indicating that *ROR -alpha* gene active sites has strong relations with mitochondrial inner membrane *OPA1* genes which are repaired by acyl-CoA:l-acyl-sn-glycero-3-phosphocholine O-acyltransferase, where that enzyme is so necessary for reactivating brain acetylcholine and for mitochondrial membrane repair and re-activities, consequently, that transferase enzyme necessary for reactivate *ROR-alpha* genes re-expressions for ameliorated the symptoms of NASH in the methionine and choline deficient .

Lysosome and *RORA* genes are both having the necessary functions of preventing G-protein aggregates associated with neuropathies, and preventing blood platelets aggregations depending on mitochondrial optimal activities through producing its active inflammatory enzymes for acting on any toxic inflammation or aggregated molecules to produce active TXA2 subunits which can be stored in or as lysosomal secretory granules, and then transmitted with mitochondrial synthase enzymes and phospholipase enzymes as active signals genes or subunits throughout plasma membrane and endosomes to interstitium fluid between cells for acting on aggregated platelets or G-proteins, and acting on inflammations for producing Thromboxane-A2 then throughout feedback will produce VEGF-A subunits for farther the rest of anti-inflammations cycles activities for reactivate endothelin and Gactin filaments, and removing any other toxicity and impurities from blood vessels.

Note, the lysis of any aggregations whether blood platelets or G-proteins aggregation will need help of the mitochondrial activities to produce its necessary enzymes for acting on the aggregation as synthase, phospholipase, and Cox2 enzymes, where those enzymes are anti-inflammatory enzymes and are so necessary for acting on any inflammations, where their effects on inflammations molecules will produce TXA2 subunits which through feedbacks will produce VEGF-A subunits for increasing the anti-inflammation processes and for reactivate endothelin re-synthesis and Gactin filaments re-activities. So, lysosomes and *RORA genes* functions are linked together and depending on those anti-inflammatory enzymes from mitochondria for lysis the aggregated G-protein, so are having almost same active receptors that can be activated by mitochondrial active enzymes, which are stimulated by inflammations molecules or by G-actin isoforms or by the tetraspanins activities, where tetraspanins functions involved in regulations of cellular signaling.

When, a decrease in NR1D1 will knockdown cells resulted in the reductions of expressions of lysosomal-associated membrane protein 1, LAMP1, commensurate (that indicating the direct strong links between NR1D1 and lysosomes), and also will be a result of reductions in VEGF-A subunits productions, then reduction in anti-inflammations processes.

The overexpression of orphan nuclear receptor (TR3) in Human Umbilical Vein Endothelial Cells (HUVECs) resulted in VEGF-A-independent synthesis, survival, and induction of several cell cycle genes activities. The overexpression of orphan nuclear receptor (TR3) reflect the increasing in mitochondrial activities and increasing in its synthase, phospholipase, and Cox2 enzymes that reflect increasing of the acting of those enzymes on inflammations molecules that will induce increasing in TXA2 alpha subunits productions, and therefore increasing in its feedback to produce VEGF-A subunits productions, and then increasing in anti-inflammations cycles. TxA2 induces platelet degranulation, depending on the mitochondrial activity. The alpha granules or subunits can lysis platelet or G-protein aggregation, and then will produce TXA2 subunits for re-complete its anti-inflammation pathways activities).

ROR α genes involved in regulating anti-inflammatory state of human macrophages, and protect liver from inflammations and from fibrosis, and has main roles in activating brain functions against inflammation and toxicity through feeding and activating enkephalin leu pentapeptides or met pentapeptides and activate many of brain processes to be driven to neuron cells.

Reductions in TNF-*α* subunits productions will reflect deficiency in mitochondria or and ribosomal activities, and will lead to Sickle Cell Disease (SCD) which marked by a phenotypic variability and inflammation plays the major role in SCD pathophysiology, which linked to *RORA1* genes functions and TNF-*α* subunits activities, where TNF-*α* subunits are so essential for anti-inflammation processes, and for regulation of bone homeostasis in several chronic immune and inflammatory joints and tissues diseases.

Also, any variations or mutations or inhibition in *ROR-alpha* genes can reflect deficiency and inhibition in mitochondrial synthetase enzyme lead to deficiency in thymine nucleotides synthesis.

RORα genes has the function of activating AMPK, moreover, *RORα* upregulated antioxidative and anti-inflammatory genes, which ameliorated the symptoms of NASH in the methionine and choline deficient diet mouse model [8].

As i mentioned before that *RORα* genes has the function of activating AMPK, and also *RORα* genes are activated by mitochondrial functions but can re-stimulate to upregulated antioxidative and anti-inflammatory genes activities. Also, RORα genes is a regulator of Treg genes responsible for suppressing allergic skin inflammation through its roles for regulating and participating in anti-inflammation processes.

Localization of *TNF-α* genes are on chromosome 6, where is localized just beside lysosomal secretory granules, that its basic activity depending on the mitochondrial activities, where mitochondrial synthetase enzyme regulate the expression of thymine nucleotides from purines through mitochondrial synthetase enzymes, and is regulating most or all metabolic cycles directly in males, and indirectly in females, where pyrimidine nucleotides are the main for DNA strand in controlling DNA strand in males, but pyrimidine is main in the DNA strand for controlling DNA strand in females where the most nucleotides in DNA strand in females are Gly, Glu, Ala, Arg, Lys, Thr, Gln, Pro, leu and a lesser of Ser nucleotides, where if 1st DNA strand in females contaminated with more pyrimidine nucleotides can lead to males characters or layer can lead to variations of normal genes lead to severe health problems including cancer, so when some mutated active sites DNA strand dominant on DNA strand in males will cause health problems, and also when some variant active sites in DNA strand in females

will dominated on their corresponding DNA strand will cause problems, means mitochondrial synthase enzyme is controlled and stimulated by their main ribosomal genes which corresponding to the related gender [9].

RESULTS

APOL1 protein is a lipoproteins involved in macrophages synthesis and involved in inflammations processes and also involved in cell death in the cause of dysfunction of mitochondria, where *ROR-alpha* has anti-inflammatory effects and involved in DNA damage. There a strong links between APOL1 protein and *ROR alpha* genes during autophagy activities and upon DNA damage and mitochondria dysfunctions. Where during autophagy activities which contain APOL1 protein (lipoproteins) will need phospholipase enz expressed from mitochondrial membrane for activating the autophagic lipoproteins (APOL1 protein) for producing *ROR-alpha* genes which involved within autophagy cells for acting on inflammations molecules, which reveal and i consider that *ROR-alpha* genes are stored within autophagy cells as lysosomal security granules which are involved with the pathogenesis of various disorders, including cancer, neurodegeneration, and inflammatory diseases in the case of deficiency of mitochondrial activities or in case of inhibitions of phospholipase enzymes, which expressed from mitochondrial membrane, which needed for activating *APOL1* gene (which involved in autophagy cells) for producing *ROR- alpha* genes from autophagy cells for fast acting on inflammations molecules.

The anti-inflammatory cycles are conserved with high quality accuracy in healthy tissues, and can start as a full cycles or less fast processes for acting on foreign molecules and on inflammations molecules, whether through Th17 Cells or through mitochondrial synthase, COX-2, and phospholipase (which is necessary for activating *APOL1* gene (the lipoproteins) in autophagy cells for producing *ROR alpha* genes from autophagy for acting fast on inflammations molecules and on foreign cells) enzymes for producing Thromboxane-A2 (TXA2) subunits which through feedback will regenerate VEGF-A subunits productions which will control the endothelin-1 resynthesis and consequently, will reactivate G-actin subunits functions .

Over increasing in sulfur and in +ve cationic units in genes and in plasma membrane will be the result of increasing the idle inactive subunits in genes or in plasma, which will be the result of formation the hard and rigid membrane structure, that will delays signals genes transmission between cells and tissues, and will delay metabolic processes pathways including anti inflammations processes, that can be the result of blood clots in vessels ,specifically blockage in the capillaries that will isolate those cells regions tissue from being connected to other cells tissue then will lead to tumor synthesis.

ROR-alpha genes may are containing Tyr, Ser, Arg, Thr, gln, and Leu, where the optimum active sites nucleotides arrangements in *RORA* straight active genes will be Ser, Tyr, Gly, Gly ,Phe, Leu, Arg which are necessary for feeding and re-activating acetylcholine and enkephalin Leu pentapeptides activities in brain ,and will be reactivated by the mitochondrial repair. Where decreasing in *RORA alpha* genes activities will reflect decreasing in mitochondrial main *OPA1* gene activities, and reflect decreasing in the mitochondrial repair enzymes and consequently will reflect decreasing in APOL1 gene activities in autophagy cells.

RORα genes is so necessary for proliferation for regulating sestrin synthesis in liver through mitochondrial activities, but the main enzyme needed to be expressed for previous steps from mitochondria is the synthetase enzyme which necessary for regulating pyrimidines synthesis from purines for regulating Leu synthesis and activities. Where The deficiency in synthetase enzyme and dysfunction in mitochondria will lead to dysfunction in *RORa* gene and consequently in sestrin-Leu carrier tool, then dysfunction in the conversion of the cholesterol to bile acids through decreasing in *CYP7A* gene synthesis, which are locally produced in brain and in liver, lead to decreasing in catalyzing the hydroxycholesterol in the regulations of cholesterol synthesis, lead to liver fibrosis, and Atherosclerosis, sickle cells disease and more.

Also, as *RORA* genes functions control VEGF-A productionsand functions, as reduction in *RORA alpha* genes activities will lead to decreasing in endothelin-1 synthesis and functions lead to increasing in vain blockage and increasing in inflammation molecules in arteries and vain with decreasing in lipid digestions and metabolism that will lead also to precipitation of lipids arteries that can lead to arteriosclerosis, and cardiovascular disease which are linked to sharp reductions in *RORA alpha* genes functions and full activities.

As the high cholesterol increase as conversion to bile acids decrease as precipitation in arteries walls will increase, and then atherosclerosis and heat disease will be the result, and the liver fibrosis will be formed, and the failure in anti-inflammation processes will be occurred. Patients with diabetes and atrial fibrillation are lacking the *ROR-a* genes activities and decreasing in *CYP7A* genes synthesis and activities.

Based on the participants' scores, the calculated t-test score at every ten domains, which is impatient, happy, frustrated, depressed, hassled, angry, worried, criticized, and tired is statistically not significant at any point/ domain. The finding indicates that the area of living affects people's minds and lifestyles. People from rural areas or urban areas have found equally worried or depressed or even happy and joyful during the lock-down period. No statically significant difference could be seen in their responses. The response percentage found to similar at each domain assessed using a checklist.

CONCLUSION

IN conclusion *RORα* genes regulates pathologic angiogenesis that are necessary for TNF*α* subunits productions, therefore are necessary for anti-inflammation cycles and processes, ROR*α* genes necessary for activating enkephalin Leu pentapeptides, where decreasing in *RORA alpha* gene's activities will cause a variants of syndromic intellectual disability.

Active *RORα* gene in the presence of mitochondrial optimal activities are so imp for protecting liver from inflammations and from fibrosis, and also are so imp for feeding and activating enkephalin pentapeptides in brain for protection from intellectual disability. *RORA* genes are so necessary for *CYP7A* genes productions and activities for conversion the cholesterol to bile acids through catalyzing 27-hydroxycholesterol molecules to cholesterol then to bile acids. Lysosomes functions are so essential for degrading G-protein aggregates where depending on mitochondrial synthase and phospholipase enzymes productivity for acting on aggregation which considered as inflammation (means is responsible for TXA2 subunits productions), and are also important for regulating

cholesterol too, therefore lysosomes functions are so connected and linked to to *RORA* genes functions.

Overstimulation to mitochondria to produce synthetase, COX-2, synthase, and phospholipase enzymes, lead to over digestion to lipid and some genes chains, lead to high catabolic processes in tissue cells, and highly digestion to amino acids in some genes, lead to mission of the characteristics of those amino acids in their genes where lead to high conversion of purine to pyrimidine, and then purines nucleotides will be lesser or may disappeared, then Gly, Cys, Arg, Thr, Ser Leu will be mutated to be tri-coded pyrimidine and will be variable in their biological structure, lead to hardening in arteries and myocardium, and can lead to tumor and cancer.

If *RORA* genes due to any known (or unknown) factors will be mutated will lead to down in liver activities and protection from fibrosis, down in VEGF-A expression, and will decrease the enkephalin pentapeptides activities, and decrease the genes signals transmission, and will lead to reductions in tetraspanins activities.

I would like to give imp note that, any active genes can found*in vivo* as a straight linear chain (active genetic thread), but can be circled or rounded (cell round shape) in a steady the lethargic or in quiet state, where can be circled through covalent linkages between Tyr and Leu, and through linkages between Arg (AGG) at the head of gene and Ser (TCC) at the end part of the gene, where at the time of activating that gene by phosphorylation those bonds between Arg and Ser will be broken through phosphorylation process lead to the return that gene to its active straight form again. Where the active gene forms are found in straight linear chains and depend on the stimulations by phosphorylation to be active. That activity which responsible for changing Circ-RNAs forms to linear active RNA L-RNAs is controlled by Ser&Arg linkages and Thr (ACC) Trp (TGG) linkages Thr (ACA) and Cys (TGT) linkages, where it's necessary to break those linkages through phosphorylation to convert circ-RNAs to to return to its active L-RNAs forms, but in case of deficiency in phosphorylations the circ-RNA will not be able to penetrate living cell for feeding ribosome, and will be present in cytosol as tiny spherical organelles, where that decreasing of previous mechanism can lead to delaying in some metabolic processes and can lead to pathogenic diseases eg un-abilities of autophages to be converted to its L-acrive forms that will force them to release their active security lysosomal *ROR-a* gene for acting on inflammation and helping for running other metabolic processes.

CONFLICT OF INTEREST

The authors declare that the research work was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

REFERENCES

1. Kovalenko OV, Metcalf DG, Hemler ME. Structural organization and interactions of transmembrane domains in tetraspanin proteins. 2005.
2. William GH, David JW. Molecular and cellular endocrinology, in principles of medical biology. 1997.
3. Camilla R. Protein sorting into multivesicular endosomes. Curr Opin Cell Biol. 2003;15(4):446-455.
4. *ROR α* controls inflammatory state of human macrophages. PLoS ONE. 2020;13(11): e0207374.
5. Orphan nuclear receptor TR3/NR4A1 regulates VEGF-A-induced angiogenesis through its transcriptional activity. J Exper Med. 2006;203(3):719-29.
6. Gindhart JG, Weber KP. Lysosome and endosome organization and transport in neurons, in encyclopedia of neuroscience. 2009.
7. Ken AR, Genevieve S. Cyp7b, a novel brain cytochrome P450, catalyzes the synthesis of neurosteroids 7*α*-hydroxy dehydroepiandrosterone and 7*α*-hydroxy pregnenolone. Proc Natl Acad Sci. 1997;13;94(10): 4925–4930.
8. Martin KO, Reiss AB, Lathe R, Javitt NB. 7 alpha-hydroxylation of 27-hydroxycholesterol: biologic role in the regulation of cholesterol synthesis. J Lipid Res. 1997;38(5):1053-1058.
9. Hailiang L, Preeti P, John YL. Cholesterol 7*α*-hydroxylase protects the liver from inflammation and fibrosis by maintaining cholesterol homeostasis. J Lipid Res. 2016;57(10):1831–1844.