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**Original Article**

# Bioinformatics analysis of microtubule-associated protein-1 light chain 3 (*MAP1LC3A*) and (*BECN1*) genes in autophagy

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## ABSTRACT

Autophagy is an effective regulatory process for eliminating tumors and worn-out intracellular components. Different groups of enzymes and regulatory elements are involved in the autophagy process. *MAP1LC3A* and *BECN1* genes are the most important gene groups in autophagy. These genes, through the production of beclin-1 and lc3 proteins, are involved in the production of autophagosomes. In general, both *MAP1LC3A* and *BECN1* genes are active in cellular responses and the biological process. The aim of this study was bioinformatics analysis at the level of genome and proteome and to evaluate and compare the expression of *MAP1LC3A* and *BECN1* genes in different human body tissues. The results of this study showed that the expression level of the *BECN1* gene was relatively higher than the *MAP1LC3A* gene in different mammals. Cell analysis of *MAP1LC3A* and BECN1 genes by antibodies that bind to proteins of target genes showed that the protein encoded by the BECN1 gene is more present in the cytosol and the proteins encoded by *MAP1LC3A* gene are locally present in vesicles. It was also found that the protein encoded by the *MAP1LC3A* gene had a higher expression in brain tissues than in other tissues, while the beclin-1 protein in cardiac tissue showed higher expression than in other tissues. Finally, by using this information, it is possible to provide the ground for targeted therapies.

## 1. Introduction

Today, there are various mechanisms for the destruction of cancerous tumors, including inactivation of carcinogenic genes[1], impaired cell homeostasis, removal of cell organs, and autophagy, the most important of which is autophagy. In the autophagy mechanism, part of the cytoplasm is swallowed by the autophagosome vacuole and destroyed by lysosome vesicles[2]. In fact, autophagy is a protected and regulated process in the destructive pathway of lysosomes that is performed by chaperones through macroautophagy, micro-autophagy, and autophagy pathways[3].

Macro autophagy is the selective or non-selective degradation of large organs or molecules. Micro autophagy is the selective or non-selective degradation of small molecules and autophagy is the selective degradation of small soluble proteins. Autophagy usually occurs in response to cellular environmental stresses [4].

In macroautophagy, a bilayer membrane is formed around the molecule known as the autophagosome, and then the lysosomes are fused with these sacs to produce autophagolysosomes and the breakdown of the encapsulated molecule with the help of

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acid hydrolase enzymes and finally the release of the products into the cytoplasm continues. Micro autophagy is based on the direct uptake of cytoplasmic material into endosomes, and It acts by the depression of the lysosome membrane [4]. Autophagy by chaperones involves protein-dependent translocation of lysosome-2 membrane protein (LAMP2) in autophagic substrates which are attached to the cytosolic chaperones of the heat shock protein family via a lysosomal membrane [5]. Macro autophagy has been studied with particular importance because of its effect on organs and its ability to cause and control certain diseases (often diseases related to cell destruction or accumulation of inappropriate proteins that can be of environmental or genetic origin) such as Parkinson's, Alzheimer's, and Huntington's disease.

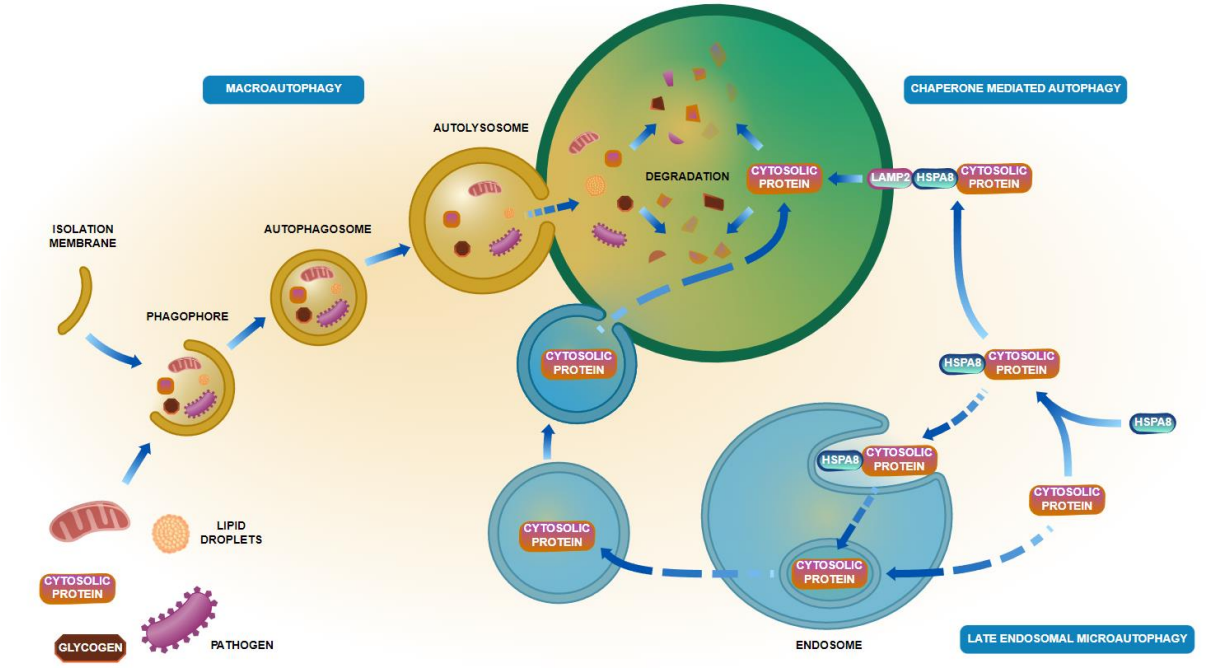


**Fig. 1**. The intracellular degradation process of autophagy. There are three primary types of autophagy - macroautophagy, chaperone-mediated autophagy (CMA), and late endosomal microautophagy. Despite being morphologically distinct, all three processes culminate in cargo delivery to the lysosome for degradation and recycling[4].

Autophagy in tumor cells has two functions: tumor suppressor and tumor induction. In fact, autophagy in the early stages of the tumor suppresses it and causes the survival of tumor cells in the advanced stages. The process of autophagy in cancer cells is induced by the long-term use of chemotherapy drugs and causes cancer cells to become resistant. It is now known that autophagy is an intracellular destruction system and triggers the defense mechanism in the cell by destroying external factors such as bacteria and viruses. Studies have shown that cells degrade their aging materials through autophagy and use them to produce new components. So in other words, autophagy is necessary to remove waste products and reduce cell consumption. Autophagy pathways were first discovered in yeasts. In total, 33 different gene groups and many regulatory elements were identified in the mechanism of autophagy. MAP1LC3A and BECN1 genes are among the most critical gene groups in autophagy in humans.

## 2. Materials and methods

First, the sequences of MAP1LC3A (NM_181509.2) and BECN1 (NM_003766.4) genes were obtained from the NCBI database. The lengths of these proteins were 125 and 450 amino acids, respectively. The exact location of these genes was then determined using the UCSC database. The three-

dimensional structure of the proteins and drawing of a diagram of Ramachandran were determined using the MBC database, and the molecular weight and isoelectric point of the proteins were determined using the ProtScale database. The codon preference trend of these proteins was investigated using the sequence manipulation suite database[6]. Cell comparison of MAP1LC3A and BECN1 genes and expression of these genes were examined by Human Protein Atlas OMIM database.

## 3. Results

### 3.1. *MAP1LC3A* gene

The MAP1LC3A gene is located on chromosome 20 with access number NM_181509.2. Microtubule-associated proteins 1A/1B light chain 3 A are proteins that in humans is encoded by the MAP1LC3A gene. The protein encoded by the MAP1LC3A gene also has the ability to bind to phospholipids, phosphatidylethanolamine, and to the protein ligase ubiquitin. This gene is involved in the signaling pathway and the early stages of autophagy. Also, the MAP1LC3A gene in mammals and yeast is associated with the AGT6 gene[7]. The number of amino acids, molecular weight, and isoelectric point of the number of exons were determined in Table 1 for the MAP1LC3A gene. It was also found that the MAP1LC3A gene is located on the long arm of chromosome 20, the exact location of which is shown in Table 1.

### 3.2. *BECN1* gene

The BECN1 gene is located on chromosome 17 with access number NM_003766.4. Beclin-1 is a protein that in humans is encoded by the BECN1 gene. The protein encoded by the BECN1 gene is capable of binding to GTPase, protein kinase, ubiquitin ligase protein, and phosphatidylinositol 3-kinase. It also shows homodimerization activity and plays a critical role in the regulation of both autophagy and cell death. BECN1 can promote autophagy and suppress the growth of breast cancer cells in vitro. It was showed [8] that nuclear BECN1 might be effective in the functional regulation of autophagic growth control. Table 1 shows the number of amino acids, the molecular weight of the isoelectric point, and the number of exons for the BECN1 gene. The results also showed that the BECN1 gene is located on the long arm of chromosome 17, the exact location of which is shown in Table 1.

**Table 1**. Genes sequence results of *MAP1LC3A* and *BECN1*

| Name | *MAP1LC3A* | *BECN1* |
|---|---|---|
| ORGANISM | Homo sapiens (Human) | Homo sapiens (Human) |
| Accession number nucleotide | NM_181509.2 | NM_003766.4 |
| Accession number protein | NP_852610.1 | AAR06912.1 |
| Gene ID | 84557 | 8678 |
| Chromosome | 20 | 17 |
| Cytogenetic location | 20q11.22 | 17q21.31 |
| Chromosome | 34546854 - | 42810134 - |
| location bp | 34560345 | 42833350 |
| nucleotide length | 994bp | 2167bp |
| protein length | 125aa | 450aa |
| Molecular weight (Da) | 14492.80 | 51896.28 |
| Isoelectric point | 7.78 | 4.83 |
| Total Exon | 5 | 12 |

### 3.3. Biological process in *MAP1LC3A*

MAP1LC3A is involved in biological processes such as autophagosome assembly, macroautophagy, cellular response to lead and copper ions, macrophage, response to nutrient levels, cellular responses to the amino acid, cellular response to hydrogen peroxide, cellular response to nitrogen starvation, and response to morphine [9].

### 3.4. Biological process in *BECN1*

*BECN1* is involved in biological processes such as autophagosome assembly, macromitophagy, endocytosis, regulation of apoptotic process, cellular defense response, lysosome organization, mitotic metaphase plate congression, aging, negative regulation of cell proliferation, response to iron (II) ions, lead, aluminum and copper regulation, positive regulation of autophagy, positive regulation of cardiac muscle hypertrophy, positive regulation of phosphatidylinositol 3-kinase signaling, response to nutrient levels, regulation of cytokinesis, response to vitamin E and drugs, cellular response to amino acid starvation, cellular response to hydrogen peroxide and glucose, early endosome to late endosome transport, late endosome to vacuole transport, neuron development, beta-amyloid metabolic process, regulation of catalytic activity, cell division, defense

response to virus, negative regulation of cell death, cellular response to epidermal growth factor stimulus, response to mitochondrial depolarisation, positive regulation of attachment of mitotic spindle microtubules to kinetochore[10, 11].

### 3.5. Three-dimensional structure

Molecular homologation modeling using the SWISS-MODEL server in Expasy resulted in a three-dimensional structure of MAP1LC3A and BECN1 proteins based on sample 1a02 with the highest similarity (Fig 2). Then Ramachandran diagram related to MAP1LC3A and BECN1 proteins was determined to determine the energy level and stability in terms of two angles φ and ψ in proteins. Considering that in BECN1 protein, the percentage of amino acids in Ramachandran favoured was 92.84% and in MAP1LC3A protein, the percentage of amino acids of Ramachandran favoured was 97.37%. Therefore, the proposed model is suitable for three-dimensional structures for proteins (Fig 3).

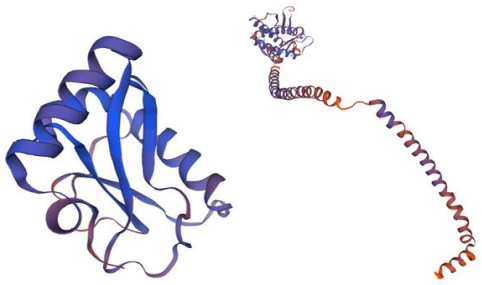

**Fig. 2**. The right side of the three-dimensional structure of *BECN1* protein, left side of the three-dimensional structure of *MAP1LC3A* protein

### 3.6. Gene expression analysis

Gene expression analysis showed that MAP1LC3A gene expression was higher in the olfactory bulb cerebral cortex and heart muscle tissues than in other organs. Also, the expression of the BECN1 gene in skeletal muscle, heart muscle, and small intestine tissues is higher than in other organs. In general, the results of MAP1LC3A and BECN1 gene expression in different tissues showed that the expression in the placenta spleen and thymus in both genes is lower than in other organs and in the MAP1LC3A gene, the expression in brain organs is more than in other organs. The study of gene expression by microarray expression data also confirmed these findings (figure 4-5).

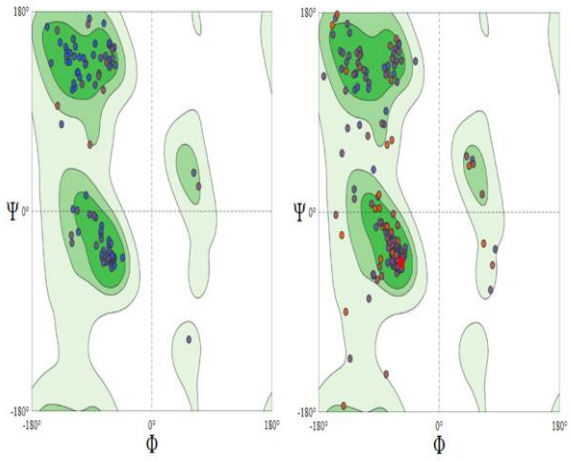


**Fig. 3**. The right side of the *BECN1* protein Ramachandran diagram, left side of the *MAP1LC3A* protein Ramachandran diagram.

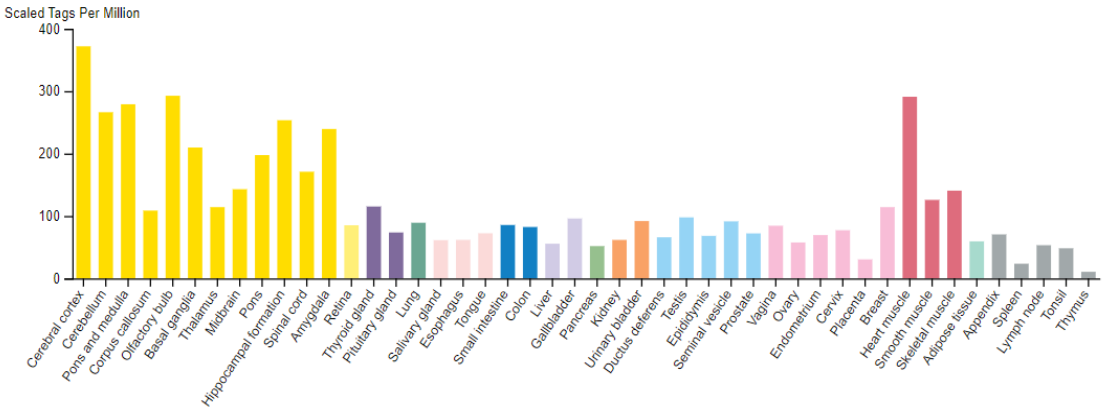


**Fig.** 4. Results of the study of *MAP1LC3A* gene expression

### 3.7. Cellular comparison of *MAP1LC3A* and *BECN1* genes

MAP1LC3A is present in cellular components such as the autophagosome membrane, cytoplasm, cytosol, late endosome, cytoskeleton, microtubule membrane, endomembrane system, cytoplasmic vesicle, and auto lysosomes [12]. BECN1 is present in cellular components such as pre-autophagosomal structure, nucleus, cytoplasm, mitochondria, endosome, Autophagosome, Endoplasmic reticulum, endoplasmic reticulum membrane, Golgi apparatus, trans-Golgi network, cytosol, endosomal membrane, membrane, the extrinsic component of membrane, dendrite, cytoplasmic vesicle, mitochondrial membrane, phosphatidylinositol 3-kinase complex and phagocytic vesicle[13](Figure 6).

### 3.8. Codon usage

The codon preference trend of these proteins was investigated using the sequence manipulation suite database, the results of which for *MAP1LC3A* and *BECN1* genes are shown in Tables 2-3. This information can be used for other research, including the transfer of these proteins. Also, we can use codon usage to assess whether a sequence shows a preference for particular synonymous codons (Table 2-3).

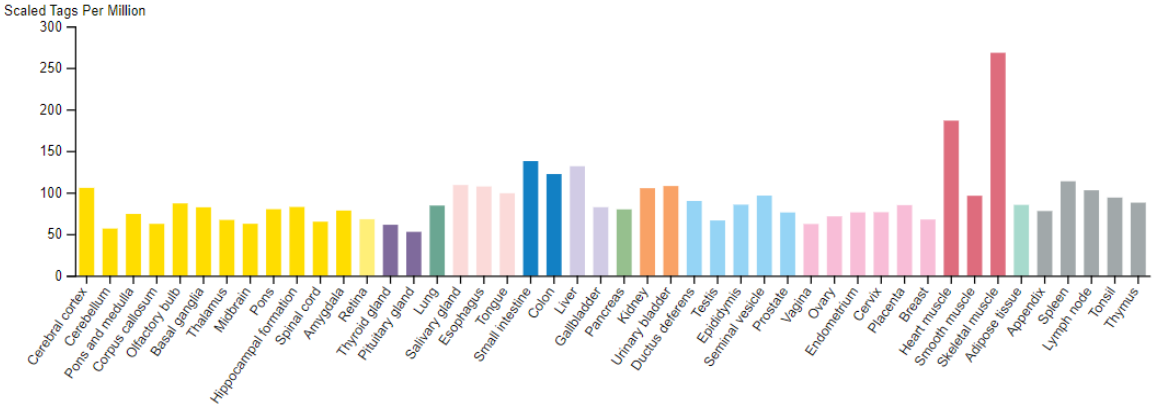


**Fig. 5**. Results of the study of *BECN1* gene expression

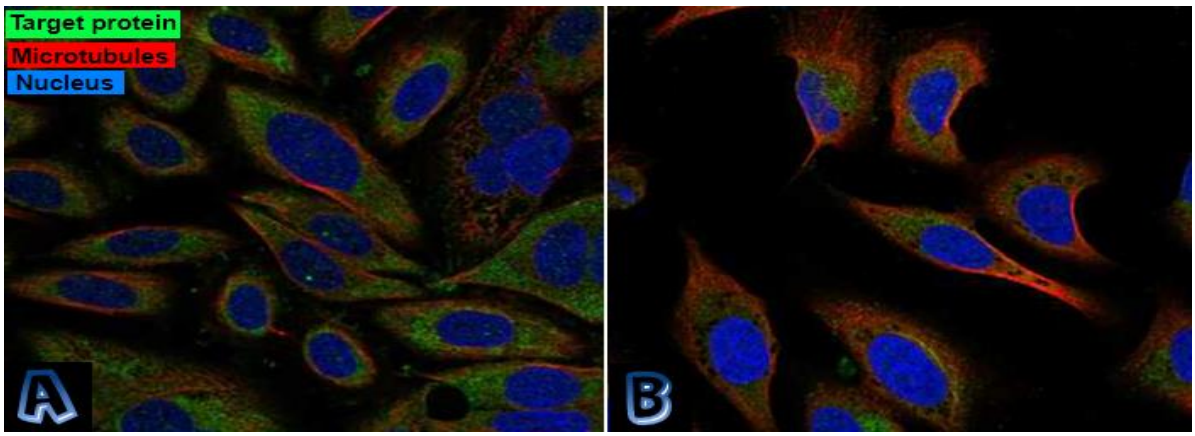


**Fig. 6.** Cell analysis of MAP1LC3A and BECN1 genes by antibodies that bind to proteins of target genes indicates that protein encoded by the BECN1 gene is more present in the cytosol (A) and proteins encoded by the MAP1LC3A gene are locally present in vesicles(B)[14].

**Table 2.** Results of codon usage for gene *MAP1LC3A* (Results for 994 residue sequence " *MAP1LC3A* " starting "GCCAGGTGCA")

| AmAcid | codon | Number | /1000 | Fraction | AmAcid | codon | Number | /1000 | Fraction | AmAcid | codon | Number | /1000 | Fraction |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Ala | GCG | 2 | 6.04 | 0.07 | Lys | AAA | 22 | 66.47 | 0.92 | Ser | AGT | 1 | 3.02 | 0.06 |
| Ala | GCA | 10 | 30.21 | 0.34 | Leu | TTG | 2 | 6.04 | 0.06 | Ser | AGC | 6 | 18.13 | 0.35 |
| Ala | GCT | 7 | 21.15 | 0.24 | Leu | TTA | 1 | 3.02 | 0.03 | Ser | TCG | 2 | 6.04 | 0.12 |
| Ala | GCC | 10 | 30.21 | 0.34 | Leu | CTG | 12 | 36.25 | 0.36 | Ser | TCA | 1 | 3.02 | 0.06 |
| Cys | TGT | 6 | 18.13 | 0.6 | Leu | CTA | 4 | 12.08 | 0.12 | Ser | TCT | 3 | 9.06 | 0.18 |
| Cys | TGC | 4 | 12.08 | 0.4 | Leu | CTT | 6 | 18.13 | 0.18 | Ser | TCC | 4 | 12.08 | 0.24 |
| Asp | GAT | 6 | 18.13 | 0.67 | Leu | CTC | 8 | 24.17 | 0.24 | Thr | ACG | 0 | 0 | 0 |
| Asp | GAC | 3 | 9.06 | 0.33 | Met | ATG | 2 | 6.04 | 1 | Thr | ACA | 1 | 3.02 | 0.14 |
| Glu | GAG | 8 | 24.17 | 0.53 | Asn | AAT | 1 | 3.02 | 0.33 | Thr | ACT | 3 | 9.06 | 0.43 |
| Glu | GAA | 7 | 21.15 | 0.47 | Asn | AAC | 2 | 6.04 | 0.67 | Thr | ACC | 3 | 9.06 | 0.43 |
| Phe | TTT | 5 | 15.11 | 0.63 | Pro | CCG | 6 | 18.13 | 0.13 | Val | GTG | 2 | 6.04 | 0.2 |
| Phe | TTC | 3 | 9.06 | 0.38 | Pro | CCA | 8 | 24.17 | 0.18 | Val | GTA | 1 | 3.02 | 0.1 |
| Gly | GGG | 6 | 18.13 | 0.14 | Pro | CCT | 14 | 42.3 | 0.31 | Val | GTT | 5 | 15.11 | 0.5 |
| Gly | GGA | 14 | 42.3 | 0.33 | Pro | CCC | 17 | 51.36 | 0.38 | Val | GTC | 2 | 6.04 | 0.2 |
| Gly | GGT | 14 | 42.3 | 0.33 | Gln | CAG | 7 | 21.15 | 0.5 | Trp | TGG | 10 | 30.21 | 1 |
| Gly | GGC | 9 | 27.19 | 0.21 | Gln | CAA | 7 | 21.15 | 0.5 | Tyr | TAT | 1 | 3.02 | 0.5 |
| His | CAT | 9 | 27.19 | 0.64 | Arg | AGG | 6 | 18.13 | 0.25 | Tyr | TAC | 1 | 3.02 | 0.5 |
| His | CAC | 5 | 15.11 | 0.36 | Arg | AGA | 3 | 9.06 | 0.13 | End | TGA | 4 | 12.08 | 0.44 |
| Ile | ATA | 0 | 0 | 0 | Arg | CGG | 3 | 9.06 | 0.13 | End | TAG | 3 | 9.06 | 0.33 |
| Ile | ATT | 1 | 3.02 | 0.33 | Arg | CGA | 6 | 18.13 | 0.25 | End | TAA | 2 | 6.04 | 0.22 |
| Ile | ATC | 2 | 6.04 | 0.67 | Arg | CGT | 4 | 12.08 | 0.17 | | | | | |
| Lys | AAG | 2 | 6.04 | 0.08 | Arg | CGC | 2 | 6.04 | 0.08 | | | | | |

**Table 3.** Results of codon usage for gene *BCN1* (Results for 2167 residue sequence " *BCN1*" starting " GTAGCGTCAC")

| AmAcid | codon | Number | /1000 | Fraction | AmAcid | codon | Number | /1000 | Fraction | AmAcid | codon | Number | /1000 | Fraction |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Ala | GCG | 3 | 4.16 | 0.1 | Lys | AAA | 23 | 31.86 | 0.52 | Ser | AGT | 10 | 13.85 | 0.19 |
| Ala | GCA | 5 | 6.93 | 0.17 | Leu | TTG | 13 | 18.01 | 0.15 | Ser | AGC | 6 | 8.31 | 0.12 |
| Ala | GCT | 13 | 18.01 | 0.45 | Leu | TTA | 14 | 19.39 | 0.16 | Ser | TCG | 4 | 5.54 | 0.08 |
| Ala | GCC | 8 | 11.08 | 0.28 | Leu | CTG | 30 | 41.55 | 0.34 | Ser | TCA | 6 | 8.31 | 0.12 |
| Cys | TGT | 13 | 18.01 | 0.57 | Leu | CTA | 3 | 4.16 | 0.03 | Ser | TCT | 15 | 20.78 | 0.29 |
| Cys | TGC | 10 | 13.85 | 0.43 | Leu | CTT | 16 | 22.16 | 0.18 | Ser | TCC | 11 | 15.24 | 0.21 |
| Asp | GAT | 15 | 20.78 | 0.48 | Leu | CTC | 12 | 16.62 | 0.14 | Thr | ACG | 4 | 5.54 | 0.08 |
| Asp | GAC | 16 | 22.16 | 0.52 | Met | ATG | 19 | 26.32 | 1 | Thr | ACA | 18 | 24.93 | 0.37 |
| Glu | GAG | 39 | 54.02 | 0.63 | Asn | AAT | 16 | 22.16 | 0.5 | Thr | ACT | 17 | 23.55 | 0.35 |
| Glu | GAA | 23 | 31.86 | 0.37 | Asn | AAC | 16 | 22.16 | 0.5 | Thr | ACC | 10 | 13.85 | 0.2 |
| Phe | TTT | 25 | 34.63 | 0.66 | Pro | CCG | 5 | 6.93 | 0.21 | Val | GTG | 13 | 18.01 | 0.41 |
| Phe | TTC | 13 | 18.01 | 0.34 | Pro | CCA | 8 | 11.08 | 0.33 | Val | GTA | 2 | 2.77 | 0.06 |
| Gly | GGG | 12 | 16.62 | 0.27 | Pro | CCT | 4 | 5.54 | 0.17 | Val | GTT | 7 | 9.7 | 0.22 |
| Gly | GGA | 11 | 15.24 | 0.24 | Pro | CCC | 7 | 9.7 | 0.29 | Val | GTC | 10 | 13.85 | 0.31 |
| Gly | GGT | 10 | 13.85 | 0.22 | Gln | CAG | 35 | 48.48 | 0.9 | Trp | TGG | 9 | 12.47 | 1 |
| Gly | GGC | 12 | 16.62 | 0.27 | Gln | CAA | 4 | 5.54 | 0.1 | Tyr | TAT | 8 | 11.08 | 0.47 |
| His | CAT | 6 | 8.31 | 0.6 | Arg | AGG | 8 | 11.08 | 0.22 | Tyr | TAC | 9 | 12.47 | 0.53 |
| His | CAC | 4 | 5.54 | 0.4 | Arg | AGA | 9 | 12.47 | 0.25 | End | TGA | 8 | 11.08 | 0.47 |
| Ile | ATA | 7 | 9.7 | 0.27 | Arg | CGG | 2 | 2.77 | 0.06 | End | TAG | 2 | 2.77 | 0.12 |
| Ile | ATT | 8 | 11.08 | 0.31 | Arg | CGA | 5 | 6.93 | 0.14 | End | TAA | 7 | 9.7 | 0.41 |
| Ile | ATC | 11 | 15.24 | 0.42 | Arg | CGT | 4 | 5.54 | 0.11 | | | | | |
| Lys | AAG | 21 | 29.09 | 0.48 | Arg | CGC | 8 | 11.08 | 0.22 | | | | | |

## 4. Discussion

Bioinformatics analyses at the molecular level allow the simultaneous study of many cellular biological and morphological properties of genes and proteins and comparisons between them. These analyses ultimately lead to a better understanding of the reactions and studies of genomics and proteomics[15]. It was Showed [16, 17] that the LC3 protein produced by the *MAP1LC3A* gene, through an autophagy mechanism, reduced the mutant Huntington's protein (mHTT) in flies and mice carrying Huntington's disease and the disease-related phenotypes in disease-containing cells Reduce. As shown, proteins produced by *MAP1LC3A* and BECN1 genes are expressed in various organs and tissues.

The lc3 protein encoded by the *MAP1LC3A* gene in brain and heart tissues and the beclin1 protein produced by the BECN1 gene show the highest expression in cardiac tissues. The three-dimensional structure of the protein encoded by the *BECN1* gene is linear and shows a higher expression in different human tissues than the *MAP1LC3A* gene. The *BECN1* gene is more present in the cytosol, and the *MAP1LC3A* gene is more active in the vesicle[18, 19].

Reports indicate that the *MAP1LC3A* gene in humans and yeast is affected by post-translational changes. One of these changes is the C-terminal cleavage of the protein, which eventually binds the Gly residue to the autophagosome membranes. In humans and rats, three isoforms of *MAP1LC3A*, *MAP1LC3B*, and *MAP1LC3C* were identified, of which only *MAP1LC3B* underwent C-terminal cleavage,

and *MAP1LC3A*, *MAP1LC3C* underwent proteolytic cleavage[20]. It was showed [7, 21] that beclin reduced Sindbis virus replication in rat brains and reduced Sindbis virus-cell death in rat brains. It was concluded [22] [23] that beclin-1 is an autophagy gene in humans, yeast, and mammals that can inhibit tumor genesis and is expressed at reduced levels in breast cancer. They also found that decreased expression of autophagy proteins led to the development of breast cancer and other human malignancies.

## 5. Conclusion

According to these findings, the importance of these genes in the mechanism of autophagy can be mentioned. Also, this information can be used to calculate stable protein conformers and model homology calculations, and blast new sequences.

## Conflict of Interests

All authors declare no conflict of interest.

## Ethics approval and consent to participate

No human or animals were used in the present research.

## Consent for publications

All authors read and approved the final manuscript for publication.

## Availability of data and material

All the data are embedded in the manuscript.

## Authors' Contribution

Main draft of the manuscript was written by A.R.M. and revised by all authors.

## Informed Consent

The authors declare not to use any patients in this research.

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