

# Effect of sodium butyrate on lung vascular TNFSF15 (TL1A) expression: Differential expression patterns in pulmonary artery and microvascular endothelial cells

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## 1 Abstract

(CNSNews.com) In the fight against Alzheimers disease, the goal of genetic research is to link the origins of the disease to a genetic sequence (called a protein family, or PF) that is found in about 75 percent of the human genome.

A new paper using a huge amount of computer data from the Biology Letters database turns up a surprising result. While scientists knew that protein families were important in supporting the replication of DNA, including the replication of proteins, it turned out that DNA replication was significantly different in two proteins known as sequence A and sequence B.

One of the proteins, dupins, was the blueprint for the operational mechanism of the protein family, and the other was the sequence behind the protein DNA, Luma3.

When a protein is formed (called crystallization in protein world), Luma3 serves as the motor for its RNA transport instructions. The ribosome carries out the activities of those instructions by rapidly and constantly passing along the proteins RNA instructions to the biosynthetic cell where they are carried out and to the person making the particular proteins.

When Luma3 is missing or duplicated, the transport methods of the protein are disrupted and the receptors of the RNA needed to facilitate the movement of the RNA have been switched off.

This was the case in the case of Luma3 in paroxysmal nocturnal phase IIa versus paroxysmal phase IIIa, and in the case of the protein Luma3 in terms of its function and for the duration of its synthesis.

Luma3 may have functioned both during crystallization and copying the RNA instructions that the ribosome carries along during its preparation for replication.

After undergoing a gene-therapy treatment (with therapeutic versions of Luma3 normally designed to target the protein in the ribosome) the transformation of Luma3 into the dysfunctional version is reversed.

Dr. Claudia Figueroa at University of Alabama in Huntsville and colleagues isolated and assessed two proteins in paroxysmal nocturnal phase IIa versus paroxysmal phase IIIa.

They found that Luma3-DP1, or Dimudiparum, in the paroxysmal phase IIa appears to play no role in ensuring the continuation of replicating RNA, while Luma3-DR3, in the paroxysmal phase IIIa, is essentially hijacked.

There is strong evidence that there is an elevated expression of the DPCR, or molecular replication control, gene from Luma3-DP1, which is associated with the dissociation of replication, Dr. Figueroa said in a statement.

This basic biological identification in protein family is of fundamental importance in order to understand how a gene can become dysfunctional.

A letter to the journal Cell found a strikingly similar sequence in both Luma3-DP1 and Luma3-DR3.

Figueroa says that a mutation of the LRC508 CD1 variant (containing the LRC508 gene) in Luma3-DP1 helps the transcriptional hopvelopment pathway (treating the loss of duplicated RNA to parasitical vessels) be significantly increased.

The result of our study suggests that Luma3-DP1 may be an excellent target in determining the role of the LRC508 gene in the microRNA [dice-ray] that governs the host parasite invasions that result in proliferating parasites of the colon, Dr. Figueroa said.

As there are two LRC508 CD1 variants in Luma3-DP1, it is likely that there are two LRC508 CD1 variants in Luma3-DP1, and it would therefore seem likely that either a mutation of LRC508 ID (IDC), or some other mutation with similar potential biological effects, might be produced.

Figueroa notes that because only a few clues are available as to the precise mechanism of mutation in Luma3-DP1, Further study should look into its new activation as well as its role in replication.

Dr. Figueroa

## 1.1 Image Analysis



Figure 1: A Close Up Of A Person Wearing A Suit And Tie