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Research Article

CHRONIC OBSTRUCTIVE PULMONARY DISEASE: STRUCTURE PREDICTION OF PULMONARY SURFACTANT ASSOCIATED PROTEIN 1

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ABSTRACT

Biological research is significantly impacted by bioinformatics. Without the bioinformatics component, massive research endeavors like the human genome project would be worthless. After the raw data is available, in silico testing of hypotheses is possible. A form of obstructive lung illness called COPD is characterized by persistently inadequate airflow. Shortness of breath and a cough that produces phlegm are the prominent symptoms. Usually, it gets worse with time. It will eventually be challenging to carry objects or climb stairs. COPD is the primary diagnosis for chronic bronchitis. The most frequent cause of COPD is tobacco use, with other variables such as air pollution and genetics having a lesser impact.

Keywords: Bioinformatics, Tobacco, Hypotheses, COPD, Impact.

INTRODUCTION

Public databanks house biological raw data (such as Gene bank or EMBL for primary DNA sequences). The Internet can be used to access and submit the data (WWW). The most probable translation of all the coding sequences in the EMBL databank is provided by protein sequence databases like trEMBL. In this area of bioinformatics, computer methods are used to forecast and analyse the spatial structure of proteins and nucleic acids. The three-dimensional (3D) structure is frequently uniquely specified by the basic sequence, but the precise rules are not fully understood, and the protein folding problem is still mostly unsolved. From the amino acid composition, several elements of protein structure can already be anticipated. The primary sequence can be used to infer secondary structure using statistics or neural networks. A multiple sequence alignment can predict secondary structure with an accuracy of more than 70%. A form of obstructive lung illness called COPD is characterised by persistently inadequate airflow. Shortness of breath and a cough that produces phlegm are the prominent symptoms. Usually, it gets worse with time (Vestbo and Jorgen, 2013). It will eventually be challenging to carry objects or climb stairs. COPD is the primary diagnosis for chronic bronchitis (John *et al*., 2011). The most frequent cause of COPD is tobacco use, with other variables such as air pollution and genetics having a lesser impact (Decramer *et al*., 2012).

Poorly vented cooking and heating fires are one of the main causes of air pollution in developing nations. Emphysema is a condition marked by the disintegration of lung tissue and restriction of the tiny airways that results from long-term exposure to these irritants (Hurd *et al*., 2007). Poor airflow as evaluated by pulmonary function testing serves as the basis for the diagnosis (Nathell *et al*., 2007). Contrary to asthma, the reduction in airflow does not significantly improve with the use of a bronchodilator. By lessening exposure to risk factors, COPD can be averted in the majority of instances (Pirozzi, 2012). This includes lowering smoking rates and enhancing both indoor and outdoor air quality. There is no cure, however medication can reduce the progression. Treatments for COPD may include quitting smoking, immunizations, respiratory therapy, and frequently oral steroids and bronchodilators. Lung transplants or long-term oxygen therapy may be advantageous for some persons. Increased drug use and hospitalization may be necessary in cases of acute deterioration.

Sputum production, shortness of breath, and a productive cough are the most typical signs of COPD

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(Vestbo and Jorgen, 2013). These symptoms have often gotten worse over time and have been around for a while. It's not clear if there are many kinds of COPD. Emphysema and chronic bronchitis were once considered separate conditions, but today, emphysema is only a term used to describe lung alterations rather than a disease, and chronic bronchitis is just a term used to describe symptoms that may or may not be associated with COPD. A persistent cough is frequently the first sign to appear. Chronic bronchitis is defined as an illness that lasts for more than three months each year for at least two years, is accompanied by sputum production, and has no other known cause. This syndrome may exist prior to the full onset of COPD. Sputum production might vary over the course of hours or even days. Sometimes the cough may not be present or may merely be intermittent and ineffective.

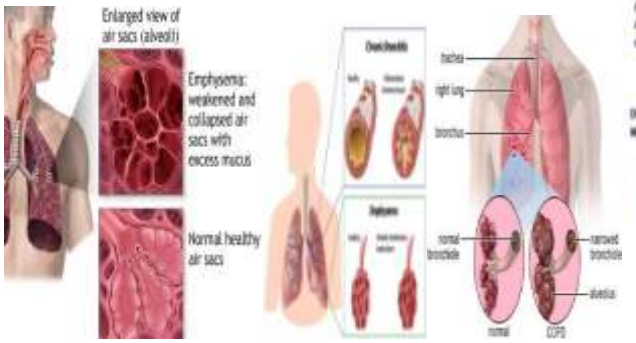
Typically the shortness of breath is worse on exertion of a prolonged duration and worsens over time. In the advanced stages it occurs during rest and may be always present. It is a source of both anxiety and a poor quality of life in those with COPD. Many people with more advanced COPD breathe through pursed lips and this action can improve shortness of breath in some. When you have COPD, breathing in may take longer than breathing out (Gruber and Phillip, 2008). Although it is uncommon, chest discomfort can be brought on by other issues. When the chest is examined with a stethoscope for obstructions in airflow, wheezing or diminished noises with air admission may be audible. A barrel chest is a defining feature of COPD, however it is not frequent. As the condition gets worse, tripod positioning may happen (John et al., 2011). High pressure on the pulmonary arteries caused by advanced COPD puts strain on the right ventricle of the heart (Weitzenblum, 2009). Leg swelling and protruding neck veins are indications of this condition, known as cor pulmonale. COPD is the most prevalent type of lung disease. Due in part to common risk factors, a variety of additional illnesses frequently coexist with COPD (Decramer *et al*., 2012). These ailments include lung cancer, ischemic heart disease, hypertension, diabetes mellitus, muscle atrophy, osteoporosis, anxiety disorder, and depression. People with severe illnesses frequently have an ongoing sense of fatigue. Fingernail clubbing should cause examinations for an underlying lung malignancy because it is not particular to COPD (James *et al*., 2009).

A COPD sufferer experiences an acute exacerbation when their symptoms of breathing difficulty, sputum output, sputum colour change from clear to green or yellow, or coughing worsen. In very severe exacerbations, this may also be accompanied by indicators of increased labour of breathing, such as rapid breathing, a fast heart rate, perspiration, active use of the neck muscles, a bluish tint to the skin, disorientation, or belligerent behaviour (Brulotte, 2012). During a stethoscope examination, crackles may also be audible over the lungs (Stephen, 2012). In addition to occupational exposure and indoor fire pollution, cigarette smoke is the main cause of COPD in most nations. Usually, it takes years or even decades for these exposures to cause symptoms. The risk is impacted by a person's genetic makeup as well. Smoking is the main risk factor for COPD worldwide. About 20% of smokers will get COPD (Helen, 2012). Additionally, roughly half of smokers who smoke continuously get COPD.

In the United States and the United Kingdom, 80-95% of people with COPD are currently or have previously smoked. The more smoke you are exposed to, the more likely you are to get COPD (Goldman and Lee, 2012). In addition, women are more vulnerable than men to the negative effects of smoking (Anita Sharma, 2010). About 20% of cases in non-smokers are caused by secondhand smoke (Stephen, 2013). There is a risk associated with smoking other substances like marijuana, cigars, and water pipes. Smoking during pregnancy may raise the likelihood that the unborn child may get COPD.

Anyone over the age of 35 to 40 with shortness of breath, a chronic cough, sputum production, or frequent winter colds should be evaluated for COPD if they have a history of exposure to risk factors for the condition. The diagnosis is then verified by spirometry. Most cases of COPD are potentially preventable through decreasing exposure to smoke and improving air quality Annual influenza vaccinations in those with COPD reduce exacerbations hospitalizations and death. Pneumococcal vaccination may also be beneficial.

Chronic obstructive pulmonary disease



Systemic effects of COPD

MATERIALS AND METHOD

UNI PORT: FASTA

BLAST

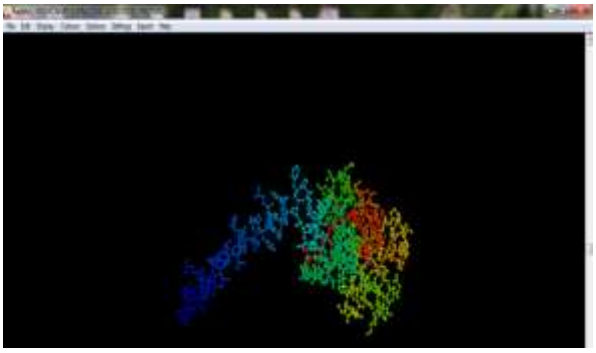
CLUSTAL OMEGA

PROTPARAM

SOPMA

CPH MODEL

RASMOL



RAMCHANDRAN PLOT

RESULTS AND DISCUSSION

Pulmonary surfactant associated protein 1 of *Homo sapiens*

>sp|P35247|SFTPD_HUMAN Pulmonary surfactant-associated protein D OS=*Homo sapiens* GN=SFTPD PE=1 SV=3
MLLFLLSALVLLTQPLGYLEAEMKTYSHRTMPSACTLVMCSSVESGLPGRDGRDGREGPR
GEKGDPGLPGAAGQAGMPGQAGPVGPKGDNGSVGEPGPKGDTGPSGPPGPPGVPGPAGRE
GPLGKQGNIGPQGKPGPKGEAGPKGEVGAPGMQGSAGARGLAGPKGERGVPGERGVPGNT
GAAGSAGAMGPQGSPGARGPPGLKGDKGIPGDKGAKGESGLPDVASLRQQVEALQGQVQH
LQAAFSQYKKVELFPNGQSVGEKIFKTAGFVKPFTEAQLLCTQAGGQLASPRSAAENAAL
QQLVVAKNEAAFLSMTDSKTEGKFTYPTGESLVYSNWAPGEPNDDGGSEDCVEIFTNGKW
NDRACGEKRLVVCEF

Pulmonary surfactandt associated protein 1 of *Rattes norvegicus*

>sp|P35248|SFTPD_RAT Pulmonary surfactant-associated protein D OS=*Rattus norvegicus* GN=Sftpd PE=1 SV=1
MLHFLSMLVLLVQPLGDLGAEMKTLSQRSITNTCTLVLCSPTENGLPGRDGRDGREGPRG
EKGDPGLPGPMGLSGLPGPRGPVGPKGENGSAGEPGPKGERGLVGPPGSPGISGPAGKEG
PSGKQGNIGPQGKPGPKGEAGPKGEVGAPGMQGSAGAKGPAGPKGERGAPGEQGAPGNAG
AAGPAGPAGPQGAPGSRGPPGLKGDRGAPGDRGIKGESGLPDSAALRQQMEALNGKLQRL
EAAFSRYKKAALFPDGQSVGDKIFRAANSEEPFEDAKEMCRQAGGQLASPRSATENAAVQ
QLVTAHSKAAFLSMTDVGTEGKFTYPTGEALVYSNWAPGEPNNNGGAENCVEIFTNGQWN
DKACGEQRLVICEF

Pulmonary surfactant associated protein 1 of *Bos taurus*

>sp|P35246|SFTPD_BOVIN Pulmonary surfactant-associated protein D OS=*Bos taurus* GN=SFTPD PE=1 SV=2
MLLLPLSVLLLLTQPWRSLGAEMKIYSQKTMANACTLVMCSPPEDGLPGRDGRDGREGPR
GEKGDPGSPGPAGRAGMPGPAGPIGLKGDNGSAGEPGPKGDTGPPGPPGMPGPAGREGPS
GKQGSMGPPGTPGPKGDTGPKGGVGAPGIQGSPGPAGLKGERGAPGEPGAPGRAGAPGPA
GAIGPQGPSGARGPPGLKGDRGTPGERGAKGESGLAEVNALRQRVGILEGQLQRLQNAFS
QYKKAMLFPNGRSVGEKIFKTEGSEKTFQDAQQICTQAGGQLPSPRSAAENEALTQLATA
QNKAAFLSMSDTRKEGTFIYPTGEPLVYSNWAPQEPNNDGGSENCVEIFPNGKWNDKVCG
EQRLVICEF

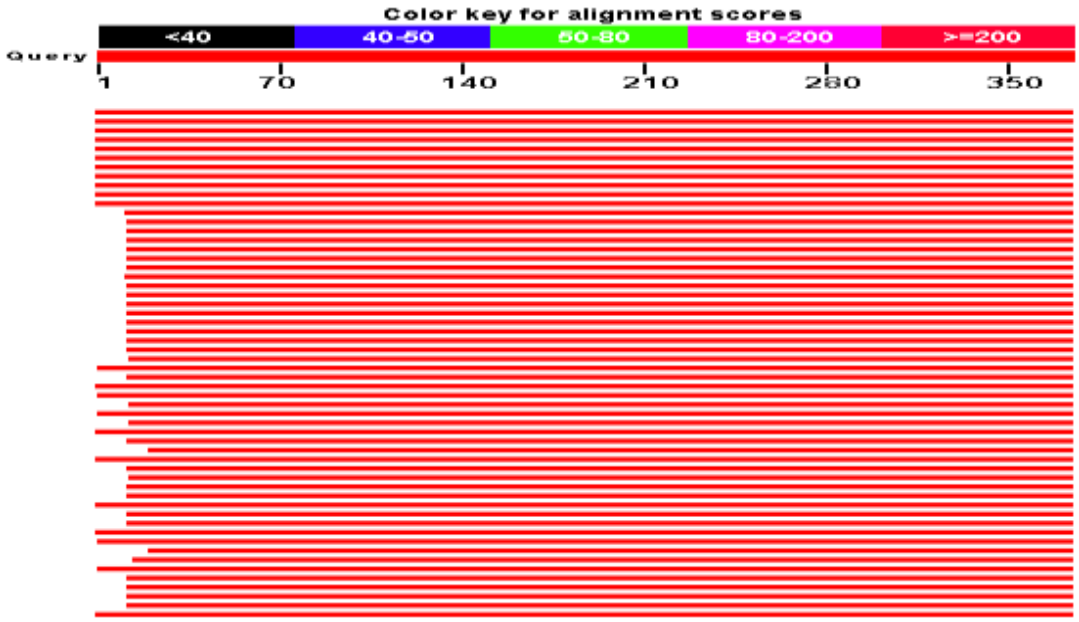
Pulmonary surfactant associated proteion1 of *Sus scrofa*

>sp|Q9N1X4|SFTPD_PIG Pulmonary surfactant-associated protein D OS=*Sus scrofa* GN=SFTPD PE=1 SV=1
MLLLPLSVLILLTQPPRSLGAEMKTYSQRAVANACALVMCSPMENGLPGRDGRDGREGPR
GEKGDPGLPGAVGRAGMPGLAGPVGPKGDNGSTGEPGAKGDIGPCGPPGPPGIPGPAGKE
GPSGQQGNIGPPGTPGPKGETGPKGEVGALGMQGSTGARGPAGLKGERGAPGERGAPGSA
GAAGPAGATGPQGPSGARGPPGLKGDRGPPGERGAKGESGLPGITALRQQVETLQGQVQR
LQKAFSQYKKVELFPNGRGVGEKIFKTGGFEKTFQDAQQVCTQAGGQMASPRSETENEAL
SQLVTAQNKAAFLSMTDIKTEGNFTYPTGEPLVYANWAPGEPNNNGGSSGAENCVEIFPN
GKWNDKACGELRLVICEF

Pulmonary surfactant associated proteion 1 of *Oryctolagus cuniculus*

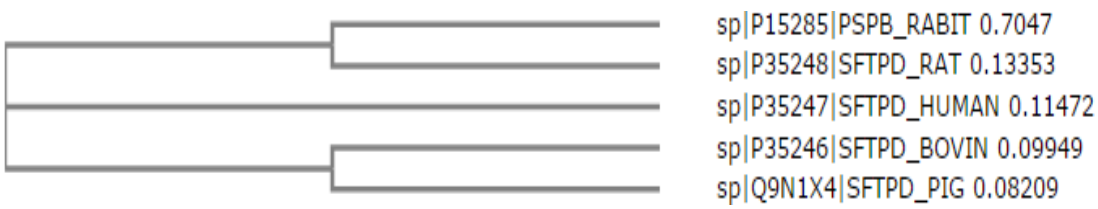
>sp|P15285|PSPB_RABIT Pulmonary surfactant-associated protein B OS=Oryctolagus cuniculus GN=SFTPB PE=2 SV=2
MAKSHLPPWLLLLLLPTLCGPGTAVWATSPLACAQGPEFWCQSLEQALQCKALGHCLQEV
WGHVGADDLCQECQDIVNILTKMTKEAIFQDTIRKFLEHECDVLPLKLLVPQCHHVLDVY
FPLTITYFQSQINAKAICQHLGLCQPGSPEPPLDPLPDKLVLPTLLGALPAKPGPHTQDL
SAQRFPIPLPLCWLCRTLLKRIQAMIPKGVLAMAVAQVCHVVPLVVGGICQCLAERYTVI
LLEVLLGHVLPQLVCGLVLRCSSVDSIGQVPPTLEALPGEWLPQDPECRLCMSVTTQARN
ISEQTRPQAVYHACLSSQLDKQECEQFVELHTPQLLSLLSRGWDARAICQALGACVATLS
PLQCIQSPHF

BLAST: Pulmonary Surfactant Associated Protein 1



Multiple sequence alignment using clustal omega Pulmonary Surfactant Associated Protein 1

PHYLOGENETIC TREE (Cladogram)



PROTEPARAM

Number of amino acids **:** 375
Molecular weight **:** 37728.3
Theoretical pI **:** 6.25
Amino acid composition **:**

Ala (A)	36	9.6%
Arg (R)	14	3.7%
Asn (N)	10	2.7%
Asp (D)	13	3.5%
Cys (C)	6	1.6%
Gln (Q)	20	5.3%
Glu (E)	25	6.7%
Gly (G)	73	19.5%
His (H)	2	0.5%
Ile (I)	4	1.1%
Leu (L)	28	7.5%
Lys (K)	23	6.1%
Met (M)	8	2.1%
Phe (F)	10	2.7%
Pro (P)	39	10.4%
Ser (S)	22	5.9%
Thr (T)	14	3.7%
Trp (W)	2	0.5%
Tyr (Y)	5	1.3%
Val (V)	21	5.6%
Pyl (O)	0	0.0%
Sec (U)	0	0.0%
(B)	0	0.0%
(Z)	0	0.0%
(X)	0	0.0%

Total number of negatively charged residues (Asp + Glu): 38
Total number of positively charged residues (Arg + Lys): 37

Atomic composition:

Carbon	C	**1634**
Hydrogen	H	**2598**
Nitrogen	N	**476**
Oxygen	O	**523**
Sulfur	S	**14**

Formula: $C_{1634}H_{2598}N_{476}O_{523}S_{14}$
Total number of atoms: 5245

Extinction coefficients:
Extinction coefficients are in units of M^{-1} cm^{-1}, at 280 nm measured in water.
Ext. coefficient 18825
Abs 0.1% (=1 g/l) 0.499, assuming all pairs of Cys residues form cystines
Ext. coefficient 18450
Abs 0.1% (=1 g/l) 0.489, assuming all Cys residues are reduced
Estimated half-life:
The N-terminal of the sequence considered is M (Met).
The estimated half-life is: 30 hours (mammalian reticulocytes, in vitro).
>20 hours (yeast, in vivo).
>10 hours (Escherichia coli, in vivo).
Instability index:
The instability index (II) is computed to be **28.61**
This classifies the protein as stable.

Aliphatic index: 59.12
Grand average of hydropathicity (GRAVY): -0.503

Secondary structure prediction for Pulmonary Surfactant Associated Protein 1 using Sopma

```
10        20        30        40        50        60        70
|         |         |         |         |         |         |
MLLFLLSALVLLTQPLGYLEAEMKTYSHRTMPSACTLVMCSSVESGLPGRDGRDGREGPRGEKGDPGLPG
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ccccccccccccccccccccccccccccccccccccccccccccccccccccccccccccccccccccccc
AGPKGEVGAPGMQGSAGARGLAGPKGERGVPGERGVPGNTGAAGSAGAMGPQGSPGARGPPGLKGDKGIP
ccccccccttccccccttccccccccccccccccccccccccccccccccccccccccccccccccccccc
GDKGAKGESGLPDVASLRQQVEALQGQVQHLQAAFSQYKKVELFPNGQSVGEKIFKTAGFVKPFTEAQLL
ccccccccccccchhhhhhhhhhhhhhhhhhhhhhhhhheeecccccchhhheeehttccccccchhhee
CTQAGGQLASPRSAAENAALQQLVVAKNEAAFLSMTDSKTEGKFTYPTGESLVYSNWAPGEPNDDGGSED
ehcttcccccccchhhhhhhhhhhhhhttthhheeeeccccctteeccccceeeeecccttccccttcccc
CVEIFTNGKWNDRACGEKRLVVCEFMLHFLSMLVLLVQPLGDLGAEMKTLSQRSITNTCTLVLCSPTENG
heeeehttcccccttcchheeehhhhhhhhhhhhheehcchhhhhhhhhhhhhcccctteeeeeecccccc
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LLPLSVLILLTQPPRSLGAEMKTYSQRAVANACALVMCSPMENGLPGRDGRDGREGPRGEKGDPGLPGAV
hcchheeeeecccccchhhhhhhhhhhhhhhhhhhheeecccctteccccccccccccccccccttccccc
GRAGMPGLAGPVGPKGDNGSTGEPGAKGDIGPCGPPGPPGIPGPAGKEGPSGQQGNIGPPGTPGPKGETG
cctttttccccccccccccccttccccccccccccccccccccccccccccccccccccccccccccccc
PKGEVGALGMQGSTGARGPAGLKGERGAPGERGAPGSAGAAGPAGATGPQGPSGARGPPGLKGDRGPPGE
cccceeeeecccccccccccccccccccccccccccccccccccccccccccccccccccccccccccccc
RGAKGESGLPGITALRQQVETLQGQVQRLQKAFSQYKKVELFPNGRGVGEKIFKTGGFEKTFQDAQQVCT
ccccccccccthhhhhhhhhhhhhhhhhhhhhhhhhhteeeeccccccccceeeettcccchhhhhhhheeh
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httccccccccccchhhhhhhhhhhttthhheeeeecccctteecccccceeeeecctttcccccccccccc
NCVEIFPNGKWNDKACGELRLVICEFMAKSHLPPWLLLLLLPTLCGPGTAVWATSPLACAQGPEFWCQSL
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HVLDVYFPLTITYFQSQINAKAICQHLGLCQPGSPEPPLDPLPDKLVLPTLLGALPAKPGPHTQDLSAQR
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FPIPLPLCWLCRTLLKRIQAMIPKGVLAMAVAQVCHVVPLVVGGICQCLAERYTVILLEVLLGHVLPQLV
ccccchhhhhhhhhhhhhhhcctthhhhhhhhhhhhhceeeethhhhhhhhhhhhheehhhhhhhcchhhh
CGLVLRCSSVDSIGQVPPTLEALPGEWLPQDPECRLCMSVTTQARNISEQTRPQAVYHACLSSQLDKQEC
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EQFVELHTPQLLSLLSRGWDARAICQALGACVATLSPLQCIQSPHF
hhhhhhccchhhhhhhhttcchhhhhhhhhhhhhhhhcchhheecttc
```
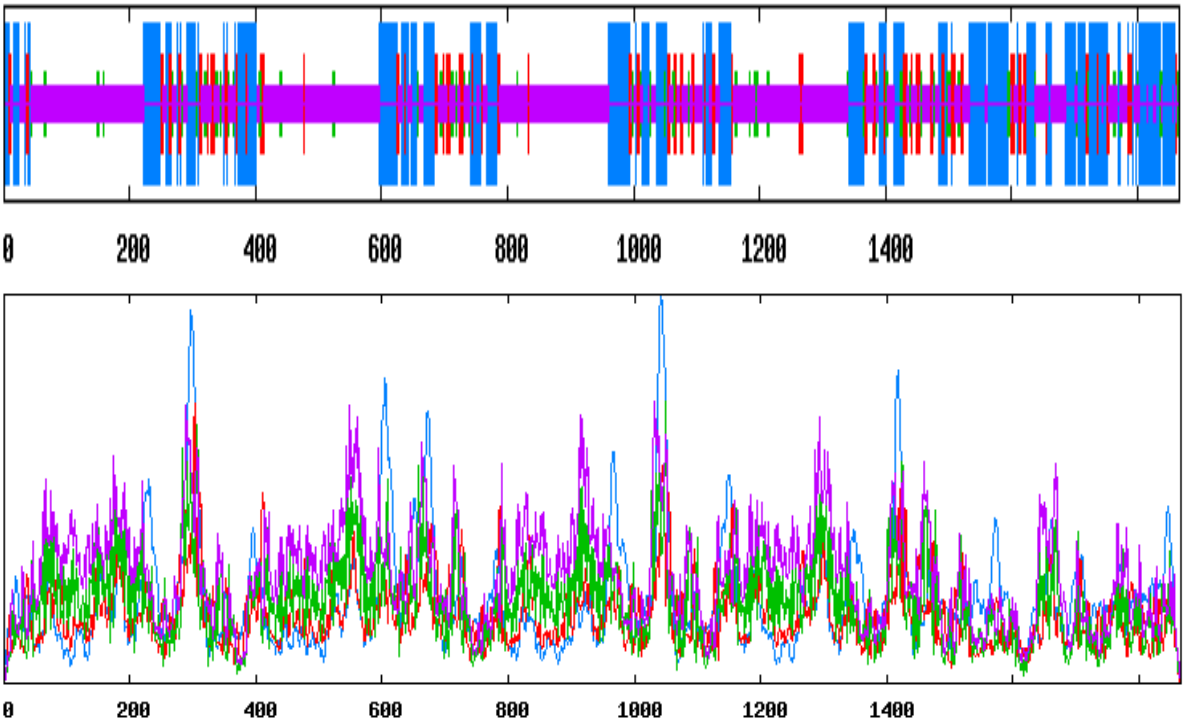
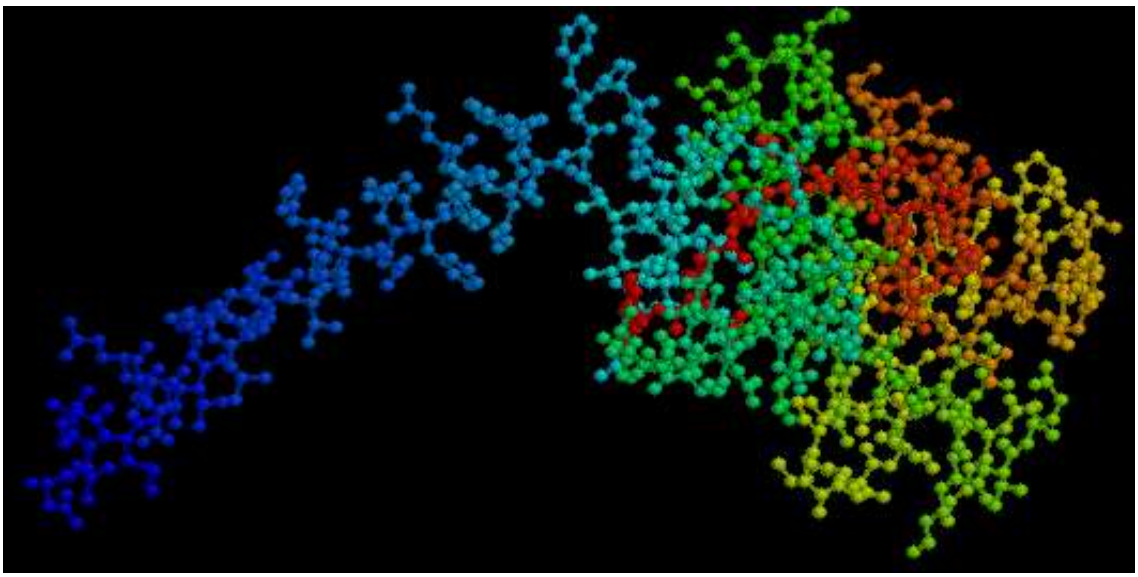

Sequence length : 1866

Alpha helix	**(Hh)**	**:**	**537**	**is 28.78%**
3_{10} helix	(Gg)	:	0	is 0.00%
Pi helix	(Ii)	:	0	is 0.00%
Beta bridge	(Bb)	:	0	is 0.00%
Extended strand	**(Ee)**	**:**	**186**	**is 9.97%**
Beta turn	**(Tt)**	**:**	**101**	**is 5.41%**
Bend region	(Ss)	:	0	is 0.00%
Random coil	**(Cc)**	**:**	**1042**	**is 55.84%**
Ambiguous states	(?)	:	0	is 0.00%
Other states		:	0	is 0.00%

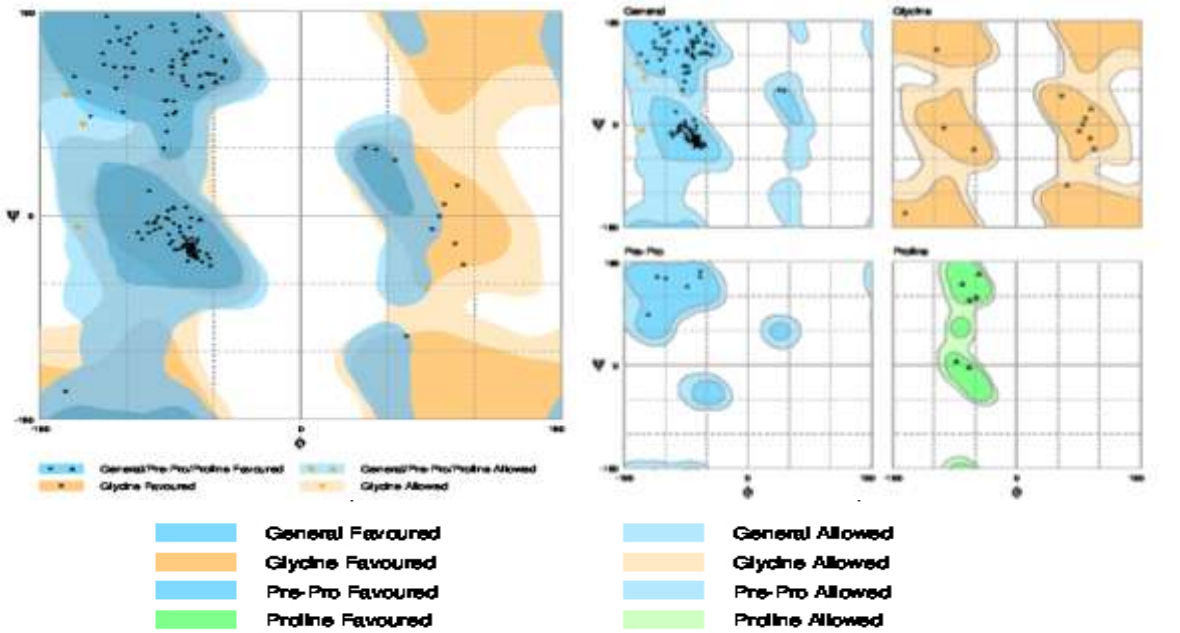
Structural analysis of Pulmonary Surfactant Associated Protein 1



Pulmonary Surfactant Associated Protein 1 (Ball and Stick model)



Rampage: assessment of the Ramchandranplot Pulmonary Surfactant Associated Protein 1



Evaluation of residues

```
Residue [  269 :GLY] (  87.25, -64.88) in Allowed region
Residue [  320 :THR] (-161.08, 106.29) in Allowed region
Residue [  335 :SER] (-149.94,  80.92) in Allowed region
Residue [  348 :SER] (-153.57, -10.25) in Allowed region
Number of residues in favoured region    (~98.0% expected)  :  147 ( 97.4%)
Number of residues in allowed region     ( ~2.0% expected)  :    4 (  2.6%)
Number of residues in outlier region                        :    0 (  0.0%)
```

Utilizing bioinformatics methods, the structure of pulmonary surfactant associated protein 1 was predicted in the current study. The sequence for human pulmonary surfactant related protein 1 was taken from the UNIPORT protein sequence database and submitted to SIB- BLAST. By contrasting pulmonary surfactant associated protein 1, the multiple sequence alignment was carried out. Using CLUSTAL OMEGA, the protein sequence of Homo sapiens was compared to that of Rattus norvegicus, Bos taur, Sus surofa, and Oryctolagus cuniculus. The findings showed that pulmonary surfactant associated protein 1 were present. In the same way that Bovine and Pig are closely related to each other, so are Rat and Human, Rabbit and Rat, and Pulmonary Surfactant Associated Protein 1 Bovine and Pig. The human pulmonary surfactant associated protein 1 has 493 amino acids and a molecular weight of 54756.2, according to analysis of the protein's amino acid composition. Theorectical pI for it is 5.70. The largest concentration of aliphatic amino acids is seen in leusine (57). Additionally, there are more of the amino acids Serine, Valine, Isoleucine, Threonine, and Phenylacanine, with relative counts of 47, 41, 35, 32, and 30. Aspergine, an amino acid that is negatively charged, is more abundant than glutamic acid. Compared to Lysine, the positively charged amino acid Arginine is less abundant. It has an aliphatic index of 59.12 and an instability index of 28.61. Its overall hydropathicity score is 0.503. 7752 atoms make up pulmonary surfactant related protein 1. They contain the atoms of carbon, hydrogen, nitrogen, oxygen, and sulphur in quantities of 2478, 3899, 631, 724, and 20 respectively. Pulmonary surfactant associated protein 1's secondary structure consists of 28.78% alpha helix, 9.97% extended strand, 5.41s% beta twists, and 55.84% random coil. The Ramachandran plot revealed that of the 493 amino acids in the preferred region of the pulmonary surfactant related protein 1, 147 (97.4%) are in the allowed region, and 4 (2.6%) are in the outlier region. This demonstrates the viability of the predicated approach.

CONCLUSION

The sequence of pulmonary surfactant related protein 1 was obtained from UNIPORT (www.expasy. ch). BLAST was used to find comparable sequences (www.ncbi.nlm. nib. Gon:/bast). Using CLASTAL OMEGA, a multiple sequence alignment of the pulmonary surfactant associated protein 1 of Rattus norvegicus, *Bos taurus*, *Sus scrofa*, and *Oryctolagus cuniculus* was carried out. The number of different amino acid and their percentage composition were

determined. Pulmonary surfactant associated protein 1 of Human and Rat is closely related, *Rabbit* and *Rat* are closely related, like wise of pulmonary surfactant associated protein 1 *Bovine* and *Pig* are closely related to each other. Performed PROTPARAM to determine the number, composition of amino acids. Pulmonary surfactant associated protein 1 of Human 1866 amino acid. Glycine present highest number (57). By SOPMA MODAL the secondary structure was determined. Pulmonary surfactant associated protein 1 as 28.78% Alpha helix and 5.41% beta turn. The tertiary structure of pulmonary surfactant associated protein 1 was predicted by using CPH model and visualized using RASMOL The Ramachandran plot was performed for pulmonary surfactant associated protein 1 by using RAMPAGE software.

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