



**INTERNATIONAL JOURNAL OF  
PHARMACEUTICAL SCIENCES**  
[ISSN: 0975-4725; CODEN(USA): IJPS00]  
Journal Homepage: <https://www.ijpsjournal.com>



## Review Paper

# Statistical Study and Analysis of Plasma Proteins

**Bhargavi Reddy Metta\***, Krishna Samalla

*Scientist ICHOR Biologics Ltd*

### ARTICLE INFO

Published: 15 Apr. 2025

**Keywords:**

Plasma proteins  
electrophoresis, Serum  
albumins serum proteins,  
monoclonal gammopathy,  
polyclonal gammopathy

**DOI:**

10.5281/zenodo.15224035

### ABSTRACT

Serum albumins are important in regulating blood volume by maintaining the oncotic pressure (also known as colloid osmotic pressure) of the blood compartment, bovine serum albumin (cattle serum albumin) or BSA, often used in medical and molecular biology labs. Serum albumin is the main protein of human blood plasma. It binds water, cations (such as  $\text{Ca}^{2+}$ ,  $\text{Na}^+$  and  $\text{K}^+$ ), fatty acids, hormones, bilirubin, thyroxin (T4) and pharmaceuticals (including barbiturates), its main function is to regulate the Oncotic pressure of blood. Alpha and beta globulins function as enzymes and proteins that transport compounds in the body. Gamma globulins act as the antibody defense against antigen invasion[1-2] Gamma globulins are manufactured in cells of the immune system known as lymphocytes and plasma cell, Blood plasma, which is usually about 4% of the body weight, consists of many constituents including  $\text{H}_2\text{O}$ , nutrients and metabolites, proteins, various hormones and electrolytes.[1] The normal total plasma protein concentration ranges from 5.5-8.9 gm/dl in domestic animal species, and it is a complex mixture including simple proteins, glyco proteins and various lipo protein hous ands of antibodies (immunoglobulins) are also present in plasma, although under normal conditions the amount of any one is usually quite low.

### INTRODUCTION

Plasma proteins are important complementary constituents in the diagnosis of gastrointestinal, hepatic, renal, or infectious diseases. Determination of plasma proteins seldom leads to a specific diagnosis (e.g., monoclonal gammopathies) but will help the clinician to evaluate the nature, severity, and progress of a

disease. Blood plasma contains 8% solids, which has 7% albumin. The different plasma proteins are albumins, globulins, and fibrinogen. Usually, total plasma proteins are 6 to 8 gms / 100 ml of blood. The proteins present in human blood plasma are a mixture of simple proteins, glycoproteins, lipoproteins [3] and other conjugated proteins called "Plasma Protein". The Functions of Plasma Proteins Plasma proteins are a crucial component

**\*Corresponding Author:** Bhargavi Reddy Metta

**Address:** *Scientist ICHOR Biologics Ltd*

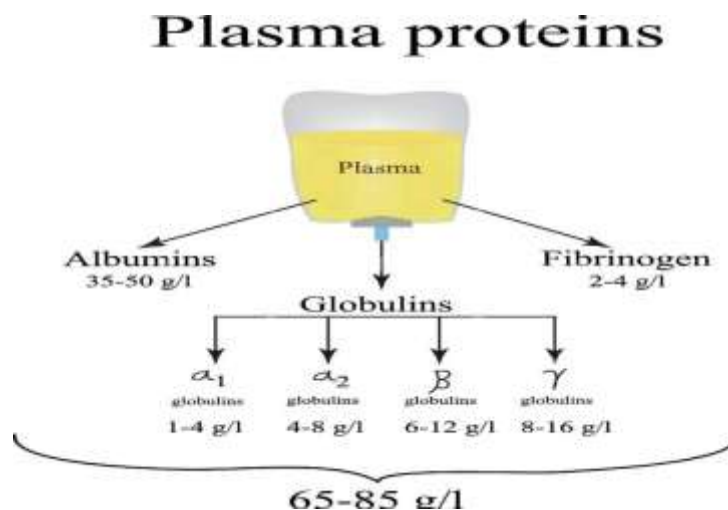
**Email** ✉: [bhargavireddi09@gmail.com](mailto:bhargavireddi09@gmail.com)

**Relevant conflicts of interest/financial disclosures:** The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.



of blood that perform many functions in the human body. They are produced in the liver and circulate in the blood, which is vital in maintaining our

overall health. Plasma protein is vital in our body. Here are the primary functions.



**Fig 1: Illustration of Plasma Proteins**

## 2. Functions of Plasma Proteins

### 2.1 Protein Nutrition

#### 2.1.1 Osmotic pressure and water balance:

The most abundant plasma protein in the blood is albumin, which plays a crucial role in maintaining fluid balance in the body [10]. Albumin helps to regulate the amount of fluid in the blood vessels, preventing excessive fluid build-up in the tissues. It creates an oncotic pressure, which draws fluid back in to the blood vessels. It prevents fluid from accumulating in the tissues, which can lead to edema, a condition where excess fluid builds up in the body [15]. Albumin is also involved in maintaining blood pressure. It helps to prevent a drop in blood pressure by regulating the amount of fluid in the blood vessels [5]. When there is a decrease in blood volume, albumin draws fluid back into the blood vessels, which helps to maintain blood pressure. During protein loss from the body, as in kidney diseases, an excessive amount of water moves in to the tissues, producing edema. Osmotic pressure is the force that drives the movement of water molecules from a region of

low solute concentration to a high solute concentration, while oncotic pressure is the force exerted by proteins in the blood that draws water into the blood vessels [12].

**Immune function** Plasma proteins play a crucial role in the immune system. Globulins are the primary type of plasma protein involved in immune function, as they help to fight off infections and diseases [15]. There are three types of globulins – alpha, beta, and gamma – and each has a unique function. Alpha and beta globulins transport and bind various substances in the blood, such as metal ions and lipids. They also play a role in the immune system by recognizing and attacking foreign invaders, such as bacteria and viruses. Gamma globulins, also known as immune globulins, are the antibodies produced by the immune system in response to an infection or disease [12]. They recognize and neutralize foreign invaders, such as bacteria and viruses, helping to prevent the spread of infection.

## 3. Functions of Plasma Proteins

Buffering action: Plasma proteins help maintain the body's pH by acting as ampholytes. At normal blood pH, they act as acids and accept captions.

**Transport:** One of the essential functions of plasma proteins is to transport lipids and lipid-soluble substances throughout the body [13] Plasma proteins transport various substances in the blood, including hormones, drugs, and fatty acids.

### **Blood Coagulation:**

Fibrinogen is a plasma protein that plays a crucial role in blood clotting. When an injury or trauma occurs, fibrinogen is converted to fibrin [14] which forms a clot to stop bleeding. This process is essential for preventing excessive bleeding and promoting wound healing.

### **Types of Plasma Proteins**

The three significant plasma protein fractions are albumin, globulin, and fibrinogen. Albumin – 55.2%  $\alpha$ 1-Globulin –5.3% ( $\alpha$ 1-Antitrypsin, Thyroxin binding Globulin (TBG), Transcortin, etc.)  $\alpha$ 2-Globulin –8.6% (Haptoglobin, ceruloplasmin,  $\alpha$ 2-macroglobulin, etc.)  $\beta$ -Globulin –13.4% ( $\beta$ 1-transferrin,  $\beta$ -lipoprotein, etc)  $\gamma$  - Globulin –11.0% (Antibodies, etc.) Fibrinogen – 6.5%

### **Albumin**

It is the most abundant class of plasma protein (2.8 to 4.5 gm/100 ml) with the highest electro heretic mobility. It is soluble in water and is precipitated by fully saturated ammonium sulphate. Albumin is synthesized in the liver and consists of a single polypeptide chain of 610 amino acids with amolecular weight of 69,000

**Globulins** By electrophoresis [11] plasma globulins are separated into  $\alpha$ 1,  $\alpha$ 2,  $\beta$ , and  $\gamma$ -globulins. [10] these proteins are synthesized in

the liver, whereas  $\gamma$ -globulins are formed in the cells of the reticuloendothelial system.

**$\alpha$ 1-Globulin.** This fraction includes several complex proteins containing carbohydrates and lipids. Lipoproteins

are soluble complexes that contain no covalently bound lipids [16]. These proteins act mainly as transport carriers for different lipids in the body.

**A2-Globulins:** This fraction also contains complex proteins such as  $\alpha$ 2-glycoproteins, plasminogen, prothrombin, haptoglobin, ceruloplasmin (transports Cu), and  $\alpha$ 2-macroglobulin.

### **B-Globulins:**

This fraction of plasma protein contains these different  $\beta$ -lipoproteins, which are very rich in lipid content. It also has transferrin (siderophilin), which transports non-heme iron in plasma.

**$\gamma$ -Globulins.** These are also called Immunoglobulin's and have antibody activity. Based on their electrophoretic mobility, [17] they are classified as IgG, IgA, and IgM. Immunoglobulins are clinically significant components of globulins. Two different types of lymphocytes involved with immunoglobulin formation are "T-cells of Thymus" and "B-Cells" that originate from "bone marrow."

### **Fibrinogen:**

It is a fibrous protein with a molecular weight of 340,000. It has six polypeptide chains which are held together by disulfide linkages. Thrombin converts fibrinogen into fibrin, which plays a vital role in the clotting of the blood. Fibrinogen is a Complex protein that is involved in the blood clotting process. It is synthesized by the liver and is converted to fibrin during the clotting process [13]. Fibrin helps to form a clot, which is necessary to



stop bleeding after an injury in addition to these three main types of plasma proteins, there are also a number of other proteins that are found in smaller quantities in the blood plasma, including lipoproteins, which transport lipids throughout the body, and enzymes, which catalyze biochemical reactions

C-reactive protein, serum amyloid A, and fibrinogen levels all quickly rise as part of the acute phase response, which is the body's initial reaction to surgical trauma [15] These proteins aid in mobilizing the body's immune response to the site of damage and are involved in tissue healing and inflammation.

#### 4. Acute Phase Response

**Table 1: Plasma Proteins and Clinical Significance Case Study 1**

Protein molecular weight (g/mol)	Concentration in serum (g/L)	Half- life (days)	Function	Common causes for increased (!) or decreased (!) concentrations
<b>Transthyretin</b> (Prealbumin) 54,000	0.2 to 0.4	2	<ul style="list-style-type: none"> <li>binds thyroid hormones and retinol (transport protein)</li> </ul>	↓ malnutrition
<b>Albumin</b> 68,000	35 to 53	15-19	<ul style="list-style-type: none"> <li>most important transport protein</li> <li>major contributor to oncotic pressure</li> <li>protein reserve of organism</li> </ul>	↓ catabolism ↓ hepatopathy (decreased synthesis) ↓ protein loss (nephrotic syndrome)

**Table 2: Plasma Proteins and Clinical Significance Case Study 2**

Protein molecular weight (g/mol)	Concentration in serum (g/L)	Half- life (days)	Function	Common causes for increased (!) or decreased (!) concentrations
<b>Transthyretin</b> (Prealbumin) 54,000	0.2 to 0.4	2	<ul style="list-style-type: none"> <li>binds thyroid hormones and retinol (transport protein)</li> </ul>	↓ malnutrition
<b>Albumin</b> 68,000	35 to 53	15-19	<ul style="list-style-type: none"> <li>most important transport protein</li> <li>major contributor to oncotic pressure</li> <li>protein reserve of organism</li> </ul>	↓ catabolism ↓ hepatopathy (decreased synthesis) ↓ protein loss (nephrotic syndrome)

<b>α<sub>1</sub> zone</b>	<b>α<sub>1</sub>-lipoprotein</b> 180,000–360,000	1.0 to 1.6 (Apo A-I)		<ul style="list-style-type: none"> <li>high-density lipoprotein (HDL)</li> <li>cholesterol transport to liver</li> </ul>	↑ <b>dyslipidemia</b>
	<b>α<sub>1</sub>-antitrypsin</b> 54,000	0.9 to 2.0	4	<ul style="list-style-type: none"> <li>lysosomal protease inhibitor (mostly elastase)</li> <li>hereditary deficit causes hereditary emphysema and cirrhosis</li> </ul>	↑ <b>acute inflammation</b> ↓ hereditary antitrypsin deficiency
	<b>orosomuroid</b> (α <sub>1</sub> -acidic glycoprotein) 40,000	0.5 to 1.2	5	<ul style="list-style-type: none"> <li>binds lipophilic compounds (e.g., progesterone and drugs)</li> <li>regulates the immune response</li> </ul>	↑ <b>inflammation</b>
	<b>α<sub>1</sub>-fetoprotein</b> 69,000	below 7.5 µg/L	3.5	<ul style="list-style-type: none"> <li>physiologically produced by the fetal liver and the yolk sack</li> <li>main protein of fetal serum</li> <li>detectable in serum of pregnant women</li> </ul>	↑ <b>hepatoma</b> ↑ some GIT malignities ↑ pregnancy ↑ in fetus

<b>α<sub>2</sub> zone</b>	<b>Haptoglobin</b> <sup>[p 1]</sup> 85,000–1,000,000	0.3 to 2.0	2	<ul style="list-style-type: none"> <li>binds liberated hemoglobin (in intravascular hemolysis)</li> </ul>	↑ <b>acute inflammation</b> ↓ hepatopathy ↓ intravascular hemolysis (increased consumption of haptoglobin)
	<b>α<sub>2</sub>-macroglobulin</b> 800,000	1.3 to 3.0	5	<ul style="list-style-type: none"> <li>protease inhibitor (thrombin, trypsin, chymotrypsin, pepsin)</li> <li>transport of small proteins (cytokines, growth factors) and divalent ions (e.g., Zn<sup>2+</sup>)</li> </ul>	↑ <b>acute inflammation</b>  cannot be filtered through glomerular membrane even in nephrotic syndrome - marker of nephrotic syndrome
	<b>Ceruloplasmin</b> 160,000	0.2 to 0.6	4.5	<ul style="list-style-type: none"> <li>redox activity (oxidation of Fe<sup>2+</sup> to Fe<sup>3+</sup>)</li> <li>binds copper (up to 90% of all copper in serum)</li> </ul>	↓ <b>Wilson's disease</b> (hepatolenticular degeneration)

**Table 3: Plasma Proteins and Clinical Significance Case Study**

Protein molecular weight (g/mol)	Concentration in serum (g/L)	Half- life (days)	Function	Common causes for increased (↑) or decreased (↓) concentrations
<b>Transthyretin</b> (Prealbumin) 54,000	0.2 to 0.4	2	<ul style="list-style-type: none"> <li>binds <b>thyroid hormones</b> and <b>retinol</b> (transport protein)</li> </ul>	↓ malnutrition
<b>Albumin</b> 68,000	35 to 53	15-19	<ul style="list-style-type: none"> <li>most important transport protein</li> <li>major contributor to oncotic pressure</li> <li>protein reserve of organism</li> </ul>	↓ <b>catabolism</b> ↓ <b>hepatopathy</b> (decreased synthesis) ↓ <b>protein loss</b> ( <b>nephrotic syndrome</b> )

**Table 4: Plasma Proteins and Clinical Significance Case Study 4**

<b>β<sub>1</sub> zone</b>	<b>Transferrin</b> 77,000	2.0 to 3.6	7	<ul style="list-style-type: none"> <li>transport of iron</li> </ul>	↑ iron deficiency ↓ malnutrition ↓ hepatopathy ↓ inflammation
	<b>Hemopexin</b> 57,000	0.5 to 1.1	3-7	<ul style="list-style-type: none"> <li>binds liberated <b>heme</b> (in intravascular hemolysis)</li> </ul>	
	<b>β-lipoprotein</b> 2,750,000	0.7 to 0.9 (Apo B-100)	3	<ul style="list-style-type: none"> <li>low-density lipoprotein (LDL)</li> <li>cholesterol transport from liver to tissues</li> <li>high and variable molar weight</li> </ul>	
	<b>C4</b> (complement protein) 206,000	0.1 to 0.4	1	<ul style="list-style-type: none"> <li>part of <b>complement</b> cascade</li> </ul>	↑ <b>inflammation</b> ↓ autoimmune disorders

<b><math>\beta_2</math> zone</b>	<b>C3</b> (complement protein) 180,000	0.8 to 1.4	1	<ul style="list-style-type: none"> <li>part of <b>complement</b> cascade</li> </ul>	↑ <b>inflammation</b> ↓ autoimmune disorders
	<b><math>\beta_2</math>-microglobulin</b> 11,800	0.001 to 0.002		<ul style="list-style-type: none"> <li>soluble part of leukocyte receptors</li> </ul>	↑ hematologic tumor ↓ tubular resorption disorder
	<b>Fibrinogen</b> 340,000	1.5 to 4.5		<ul style="list-style-type: none"> <li>coagulation cascade, <b>fibrin</b> precursor</li> <li><b>only in plasma, not in serum</b></li> </ul>	↑ inflammation ↑ pregnancy
	<b>C-reactive protein</b> (CRP) 111,000	1.5 to 5 mg/L	1	<ul style="list-style-type: none"> <li>complement activation</li> </ul>	↑↑ <b>acute bacterial inflammation</b> (up to 400 mg/L) (↑) <b>acute viral inflammation</b> (does not have to change, may rise to 20 to 40 mg/L)

## CONCLUSION

In this paper a statistical report has been presented on Plasma Proteins and Clinical Significance Plasma proteins play a dominant role in the pathogenesis of a variety of diseases and clinical syndromes[18] The names of some proteins have become the names of diseases; the specific items discussed in this paper include albumin, fibrinogen, globulins, liver disease, pregnancy, infancy and old age, hemorrhagic disorders, amyloidosis, and other topics.[25] The treatment of these complex subjects is aimed at the person without specialized education Separating serum proteins by electrophoresis is a valuable diagnostic tool as well as a way to monitor clinical progress[16] Current research regarding blood plasma proteins is centered on performing proteomics analyses of serum/plasma in the search for biomarkers. It is observed in this study Blood proteins, also termed plasma proteins, are proteins present in blood plasma. They serve many different functions, including transport of lipids, hormones, vitamins and minerals in activity and functioning of the immune system. Other blood proteins act as enzymes, complement components,

protease inhibitors or kinin precursors. Contrary to popular belief, haemoglobin is not a blood protein, as it is carried within red blood cells, rather than in the blood serum.

## REFERENCES

1. Aarons L. J., Rowland M. Kinetics of drug displacement interactions. *J Pharmacokinet Biopharm.* 1981 Apr;9(2):181–190. doi: 10.1007/BF01068081. [DOI] [PubMed] [Google Scholar]
2. Aggeler P. M., O'Reilly R. A., Leong L., Kowitz P. E. Potentiation of anticoagulant effect of warfarin by phenylbutazone. *N Engl J Med.* 1967 Mar 2;276(9):496–501. doi: 10.1056/NEJM196703022760904. [DOI] [PubMed] [Google Scholar]
3. Banfield C., O'Reilly R., Chan E., Rowland M. Phenylbutazone-warfarin interaction in man: further stereochemical and metabolic considerations. *Br J Clin Pharmacol.* 1983 Dec;16(6):669–675. doi: 10.1111/j.1365-2125.1983.tb02239. x. [DOI] [PMC free article] [PubMed] [Google Scholar]
4. Bjornsson T. D., Meffin P. J., Swezey S. E., Blaschke T. F. Effects of clofibrate and



- warfarin alone and in combination on the disposition of vitamin K1. *J Pharmacol Exp Ther.* 1979 Sep;210(3):322–326. [PubMed] [Google Scholar]
5. Bjornsson T. D., Meffin P. J., Swezey S., Blaschke T. F. Clofibrate displaces warfarin from plasma proteins in man: an example of a pure displacement interaction. *J Pharmacol Exp Ther.* 1979 Sep;210(3):316–321. [PubMed] [Google Scholar]
  6. Brodie B. B. Displacement of one drug by another from carrier or receptor sites. *Proc R Soc Med.* 1965 Nov;58(11 Pt 2):946–955. [PMC free article] [PubMed] [Google Scholar]
  7. Christensen L. K., Hansen J. M., Kristensen M. Sulphaphenazole-Induced Hypoglycaemic Attacks In Tolbutamide-Treated Diabetics. *Lancet.* 1963 Dec 21;2(7321):1298–1301. Doi: 10.1016/S0140-6736(63)90847-X. [Doi] [Pubmed] [Google Scholar]
  8. Fox S. L. Potentiation of Anticoagulants Caused by Pyrazole Compounds. *Jama.* 1964 Apr 20; 188:320–321. Doi: 10.1001/Jama.1964.03060290124046. [Doi] [Pubmed] [Google Scholar]
  9. Faed E. M. Protein Binding Of Drugs In Plasma, Interstitial Fluid And tissues: effect on pharmacokinetics. *Eur J Clin Pharmacol.* 1981;21(1):77–81. doi: 10.1007/BF00609592. [DOI] [PubMed] [Google Scholar]
  10. Gibaldi M., Levy G., McNamara P. J. Effect of plasma protein and tissue binding on the biologic half-life of drugs. *Clin Pharmacol Ther.* 1978 Jul;24(1):1–4. doi: 10.1002/cpt19782411. [DOI] [PubMed] [Google Scholar]
  11. Griner P. F., Raisz L. G., Rickles F. R., Wiesner P. J., Odoroff C. L. Chloral hydrate and warfarin interaction: clinical significance. *Ann Intern Med.* 1971 Apr;74(4):540–543. doi: 10.7326/0003-4819-74-4-540. [DOI] [PubMed] [Google Scholar]
  12. Hansen J. M., Christensen L. K. Drug interactions with oral sulphonylurea hypoglycaemic drugs. *Drugs.* 1977 Jan;13(1):24–34. doi: 10.2165/00003495-197713010-00003. [DOI] [PubMed] [Google Scholar]
  13. Koch-Weser J., Sellers E. M. Binding of drugs to serum albumin (first of two parts). *N Engl J Med.* 1976 Feb 5;294(6):311–316. doi: 10.1056/NEJM197602052940605. [DOI] [PubMed] [Google Scholar]
  14. Liegler D. G., Henderson E. S., Hahn M. A., Oliverio V. T. The effect of organic acids on renal clearance of methotrexate in man. *Clin Pharmacol Ther.* 1969 Nov-Dec;10(6):849–857. doi: 10.1002/cpt1969106849. [DOI] [PubMed] [Google Scholar]
  15. MacKichan J. J. Pharmacokinetic consequences of drug displacement from blood and tissue proteins. *Clin Pharmacokinet.* 1984 Jan;9 (Suppl 1):32–41. doi: 10.2165/00003088-198400091-00005. [DOI] [PubMed] [Google Scholar]
  16. McElnay J. C., D'Arcy P. F. Protein binding displacement interactions and their clinical importance. *Drugs.* 1983 May;25(5):495–513. doi: 10.2165/00003495-198325050-00003. [DOI] [PubMed] [Google Scholar]
  17. O'Reilly R. A., Goulart D. A. Comparative interaction of sulfinpyrazone and phenylbutazone with racemic warfarin: alteration in vivo of free fraction of plasma warfarin. *J Pharmacol Exp Ther.* 1981 Dec;219(3):691–694. [PubMed] [Google Scholar]
  18. O'Reilly R. A. Stereoselective interaction of trimethoprim-sulfamethoxazole with the separated enantiomorphs of racemic warfarin in man. *N Engl J Med.* 1980 Jan 3;302(1):33–35. doi: 10.1056/NEJM198001033020106. [DOI] [PubMed] [Google Scholar]





19. O'Reilly R. A., Trager W. F., Motley C. H., Howald W. Stereoselective interaction of phenylbutazone with [12C/13C] warfarin pseudoracemates in man. *J Clin Invest.* 1980 Mar;65(3):746–753. doi: 10.1172/JCI109722. [DOI] [PMC free article] [PubMed] [Google Scholar]
20. Perucca E., Hebdige S., Frigo G. M., Gatti G., Lecchini S., Crema A. Interaction between phenytoin and valproic acid: plasma protein binding and metabolic effects. *Clin Pharmacol Ther.* 1980 Dec;28(6):779–789. doi: 10.1038/clpt.1980.235. [DOI] [PubMed] [Google Scholar]
21. Pond S. M., Birkett D. J., Wade D. N. Mechanisms of inhibition of tolbutamide metabolism: phenylbutazone, oxyphenbutazone, sulfaphenazole. *Clin Pharmacol Ther.* 1977 Nov;22(5 Pt 1):573–579. doi: 10.1002/cpt1977225part1573.
22. Sellers E. M., Koch-Weser J. Kinetics and clinical importance of displacement of warfarin from albumin by acidic drugs. *Ann N Y Acad Sci.* 1971 Jul 6; 179:213–225. doi: 10.1111/j.1749-6632.1971.tb46901.x.
23. Sellers E. M., Koch-Weser J. Potentiation of warfarin-induced hypoprothrombinemia by chloral hydrate. *N Engl J Med.* 1970 Oct 15;283(16):827–831. doi: 10.1056/NEJM197010152831601
24. Sellers E. M. Plasma protein displacement interactions are rarely of clinical significance. *Pharmacology.* 1979;18(5):225–227. doi: 10.1159/000137256.
25. Stewart C. F., Fleming R. A., Germain B. F., Seleznick M. J., Evans W. E. Aspirin alters methotrexate disposition in rheumatoid arthritis patients. *Arthritis Rheum.* 1991 Dec;34(12):1514–1520. doi: 10.1002/art.1780341207.
26. Tillement J. P., Zini R., Mattei C., Singlas E. Effect of phenylbutazone on the binding of vitamin K antagonists to albumin. *Eur J Clin Pharmacol.* 1973 Jun;6(1):15–18. doi: 10.1007/BF00561795. Toon S., Low L. K., Gibaldi M., Trager W. F., O'Reilly R. A., Motley C. H., Goulart D. A. The warfarin-sulfinpyrazone interaction: stereochemical considerations. *Clin Pharmacol Ther.* 1986 Jan;39(1):15–24. doi: 10.1038/clpt.1986.3.
27. Wilkinson G. R., Shand D. G. Commentary: a physiological approach to hepatic drug clearance. *Clin Pharmacol Ther.* 1975 Oct;18(4):377–390. doi: 10.1002/cpt1975184377.

**HOW TO CITE:** Bhargavi Reddy Metta\*, Krishna Samalla, Statistical Study and Analysis of Plasma Proteins, *Int. J. of Pharm. Sci.*, 2025, Vol 3, Issue 4, 1925-1933. <https://doi.org/10.5281/zenodo.15224035>

