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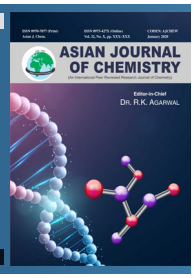

Interaction Between α-Glucosidase Inhibitor with Common Blood Proteins: A Thermodynamic and Spectroscopic Studies

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The interaction of an α-glucosidase inhibitors class of drug acarbose with globular proteins like bovine serum albumin (BSA), human serum albumin (HSA) and haemoglobin studied by fluorescence, circular dichroism (CD) spectroscopic methods. Acarbose is used for the treatment of diabetes mellitus type 2 and in some countries, prediabetes. The quenching constant (k_q) values were calculated by using fluorescence data, higher with haemoglobin (at λ_{ext} = 405 nm). It indicates the quenching process for the acarbose-haemoglobin interaction. Thus, the binding constants (k_b), infers that the electrostatic, hydrogen bonding, and intermolecular interactions play an important role in the proteins, and drug interaction. The number of binding sites (*n*), between BSA, HSA and haemoglobin with acarbose was estimated by fluorescence data, the highest binding sites (15.55) of acarbose-haemoglobin at (λ_{ext} = 405nm) indicates that the strong interaction or high quenching interaction. The interactions between BSA, HSA and haemoglobin with acarbose were confirmed by spectroscopic analysis and thermodynamic determination. The circular dichroism (CD) spectra implied the significant change in the conformation of BSA, HSA and haemoglobin upon binding with acarbose.

Keywords: Globular proteins, α-Glucosidase, Quenching constant, Binding constant, Gibbs free Energy, Circular dichroism.

INTRODUCTION

It is known very well about the interaction between globular proteins with different types of pharmaceuticals is vital for a wide field of pharmacological, biological and clinical applications. Drug and protein interactions play a vital role in pharmacological, biological, clinical and other applications. For an understanding the interaction mechanism in drugs and protein interaction by determining the interaction parameters like the binding constant, location and number of binding sites infers significant information regarding pharmacodynamics and pharmacokinetics studies of the drug [1-3].

In this study, we investigated the association of antidiabetic drug with blood proteins (BSA, HSA and haemoglobin), all proteins play a vital role in the transformation like haemoglobin plays a role transportation of oxygen from the lungs to tissues also help in the transportation of CO_2 and hydrogen ions back to the lungs in the living system. However, BSA and HSA are playing an essential role in holding life, development and metabolism of living. These globular proteins are one of the most abundant water-soluble plasma proteins in the circulatory system. It has several physico-chemical properties (PCPs), like blood pH, maintenance of osmotic pressure and transportation of biological molecules, amino acids, drugs, fatty acids, biopolymers, dyes, dendrimers, lanthanides, ionic liquids and polyphenolic compounds [1].

Furthermore, they have transport properties, so they also responsible for the maintenance of blood pressure and drug delivery [2-6]. Thus, drug and protein interactions act as a pivotal role in drug delivery. Accordingly, the interact-drug could act as a depot while without interactions, the drug develops the anticipated pharmacological effect. Therefire, it is essential for the drug-BSA interactions, which define the pharmacology and pharmacodynamics of drugs in the circulatory system. Thus, various studies have described drugs with globular protein interactions [7,8].

Addition, drug-proteins interactions allocate the surrounding environments, temperature, pH, concentrations and the

presence of other bioactive molecules like biopolymer, dendrimer, surfactants, dyes and ionic liquids, *etc*. [9]. Moreover, an interaction of drug-protein continuously induces the intervention of drug interaction, which changes the binding sites or conformational changes in proteins [10]. Therefore, the distri-bution and metabolism of several bioactive molecules, *e.g.* α-glucosidase inhibitors class of drug acarbose in the blood, been interacted with their strong non-covalent interactions affinities towards proteins. Several researchers [11] have already been discovered that proteins frequently interact association with therapeutically active compounds.

As far as our knowledge, few studies have reported on antidiabetic drugs and drug with proteins [12-19]. Several earlier studies on globular protein with the drug were deter-mined by using different spectroscopic analyses like UV-vis absorbance spectroscopy, fluorescence spectroscopy, Fourier-transform infrared spectroscopy (FTIR), Raman spectroscopy, circular dichroism (CD) spectroscopy, electrochemistry, nuclear magnetic resonance (NMR) spectroscopy and others [20-23]. When the drug interacts with protein with the help of non-covalent intermolecular bonding, hydrophobic-hydrophobic interaction (HbHbI), hydrogen bonding (HB), electrostatic interaction, intermolecular interactions (IMI) and van der Waal interaction than the native structure of a protein is loosened with changed the conformation of the protein structure [24]. As per current study of the interaction study of common blood proteins with the α-glucosidase inhibitor class of drug interaction is a well-known phenomenon. This interaction mechanism is only reflected information related to the area of structural molecular interaction activities of acarbose and a series of common blood proteins, which were confirmed by fluorescence spectroscopy, and circular dichroism (CD) spectroscopy.

EXPERIMENTAL

Acarbose, bovine serum albumin (BSA), human serum albumin (HSA) and human haemoglobin (Hb) were purchased from Sigma-Aldrich (India). Phosphate buffer saline (PBS) (10X PBS) was purchased from Sisco Research Laboratories Pvt. Ltd. (SRL), India.

Several solutions *viz*. 10 uM BSA, HSA and Hb in 0.1 M phosphate buffer (1X PBS) of pH 7.4 were prepared, which were used as stock solutions for variable concentrations 10, 20, 30, 40, 50, 60, 70, 80, 90 and 100 μM acarbose. All these solutions were prepared in an aqueous medium (Milli-Q water) with a specific conductance of 0.71 μS cm^{-1}, pH ~7 values at 298.15 K and p = 0.1 MPa.

General procedure: Using a Jasco spectrophotometer, FP8300, fluorescence studies of proteins (BSA, HSA and Hb) with variable concentrations (10-100 μM) of acarbose were conducted. Two wavelengths at 280 nm for BSA, HSA and Hb while another one at 405 nm for excitation Hb were chosen. The emission spectra were recorded in the 285 to 500 nm range with an interval of 5 nm.

Circular dichroism (CD) of proteins (BSA, HSA and Hb) with different concentrations of acarbose were measured using a Jasco J-815 CD spectrometer. This study was conducted at room temperature over the range of 190-240 nm using a 1 mm optical path of quartz cuvette with a 50 nm/min scanning rate and spectral width of 0.5 nm. All the whole studies were performed at a 20mL/min nitrogen flow rate.

Detection method: The fluorescence quenching constant was calculated using the Stern-Volmer equation. In the current study, fluorescence quenching was determined from the following eqn. 1:

$$\frac{F_0}{F} = 1 + k_q \tau_0 [Q] = 1 + S_{SV}[Q] \quad (1)$$

where F_o and F are the fluorescence intensities before and after the addition of acarbose respectively, and k_q is the quenching constant. [Q] is the quencher (acarbose) concentration, quenching efficiency and Stern-Volmer quenching constant (S_{sv}) are having very close relation to each other, and the τ_o is the average lifetime of biomolecule. The value of k_q is calculated using eqn. 2:

$$K_{SV} = k_q \tau_0 \quad (2)$$

where, $\tau_o = 10^{-8}$ s and S_{sv} is the slope of the linear regression of curve F_o/F *versus* [Q].

The binding constant and binding sites for static processes in the BSA, HSA and Hb with acarbose were calculated [25,26] using eqn. 3:

$$\log\left(\frac{F_0 - F}{F}\right) = \log K_b + n \log [Q] \quad (3)$$

where a plot of $\log\left(\frac{F_0 - F}{F}\right)$ *vs*. log [Q] is a straight line with log K_b as the intercept and *n* as the slope. And K_b is the fluorescence binding constant and *n* is represents the number of binding sites of the proteins, such analysis indicates that the interaction affinity of proteins with acarbose. We could also confirm the type of interactions by a thermodynamic parameter, which is possible in proteins and acarbose interactions [26] using eqn. 4:

$$\Delta G^\circ = -RT \ln K_b \quad (4)$$

The Gibbs free energy (ΔGº) with the help of the fluorescence binding constant (Table-1), which shows that all ΔGº

TABLE-1
STERN-VOLMER QUENCHING CONSTANT (k_{sv}), QUENCHING RATE CONSTANT (k_q), BINDING CONSTANT (K_b), NUMBER OF BINDING SITES (*n*) AND GIBBS FREE ENERGY (ΔG) FOR THE INTERACTION OF ACARBOSE WITH BSA, HSA AND HAEMOGLOBIN

System	λ_{max}	k_q (10^{11} M^{-1} s^{-1}	ΔG (kJ mol^{-1})	k_{sv} (M^{-1})	k_b (M^{-1})	*n*
BSA-Acarbose	280	2.23	-10.2757	2.2	1.80×10^5	2.04
HSA-Acarbose	280	1.87	-11.2462	1.9	1.97×10^5	3.28
Hb-Acarbose	280	1.21	-13.9864	1.2	2.45×10^5	7.32
Hb-Acarbose	405	15.55	-5.82289	15.5	1.02×10^6	14.61

values are negative, which infers that the interaction between proteins and acarbose molecules by the binding process is spontaneous [27].

The acting forces were calculated for proteins with drugs [28] using the van't Hoff eqn. 5:

$$\ln K_a = \frac{\Delta H^\circ}{RT} + \frac{\Delta S^\circ}{R} \quad (5)$$

$$\Delta G^\circ = \Delta H^\circ - T\Delta S^\circ \quad (6)$$

where K_a is the binding constant, T is the temperature in Kelvin and R is the gas constant.

RESULTS AND DISCUSSION

Fluorescence spectra and quenching mechanism: Figs. 1-3 depict the fluorescence spectra of pure proteins and in the presence of acarbose, where these observed spectra suggested the acarbose binding mechanism or phenomena with different proteins. Generally, proteins have intrinsic or extrinsic fluorescence, originating from the different amino acids present in the proteins. When these groups bind with other molecules, intrinsic or extrinsic fluorescence frequently changes with the type or concentration of the molecules [29-31].

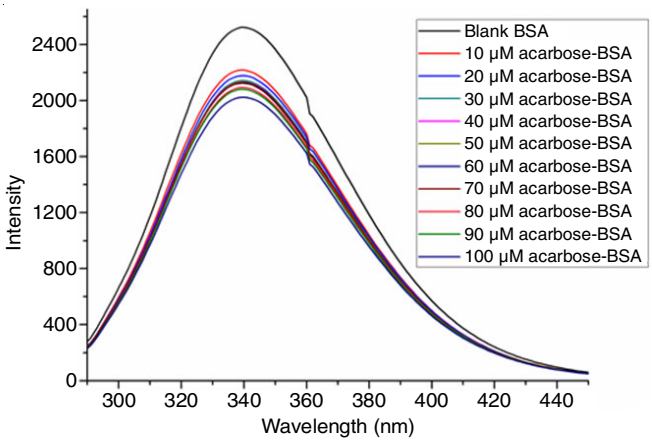

Fig. 1. Effluence of fluorescence intensity of pure BSA with increasing 10 to 100 μM concentration of acarbose

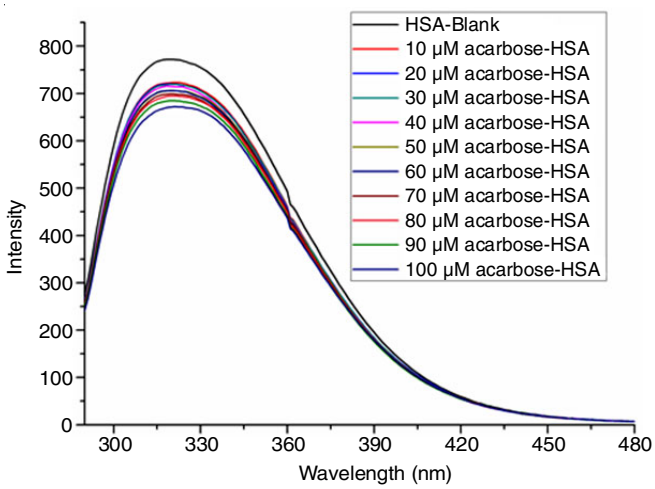

Fig. 2. Effluence on fluorescence intensity of pure HSA with increasing 10 to 100 μM concentration of acarbose

In the current study, the concentration of proteins (HSA, BSA and Hb) was fixed and the binder molecule *i.e.* 10 to 100 μM variable concentrations of acarbose. The intrinsic fluorescence of proteins mainly depends on the high sensitivity of the protein functional groups and their environment [32,33]. Thus, the change in the emission spectra of the proteins is responsible for the conformational transitions, association or dissociation of protein, molecular binding, and denaturation. Hence, the intrinsic fluorescence of the drug-protein can provide useful information for the structural dynamics *via* protein association or dissociation reaction. As shown in Figs. 1-3, the fluorescence intensities of HSA, BSA and Hb were continuously decreased with a blue shift by increasing concentration of acarbose from 10 to 100 μM. These observed results suggest that the fluorescence chromophore groups of BSA, HSA and Hb were placed in a more hydrophilic environment with acarbose with the fluorescence quenching effect *via* the formation of the non-fluorescent complex [34].

The decrease in fluorescence quenching may be due to the variety of intermolecular interactions, energy transfer, excitation state complex formation and a collision quenching mechanism. The observed quenching mechanism is also known as dynamic

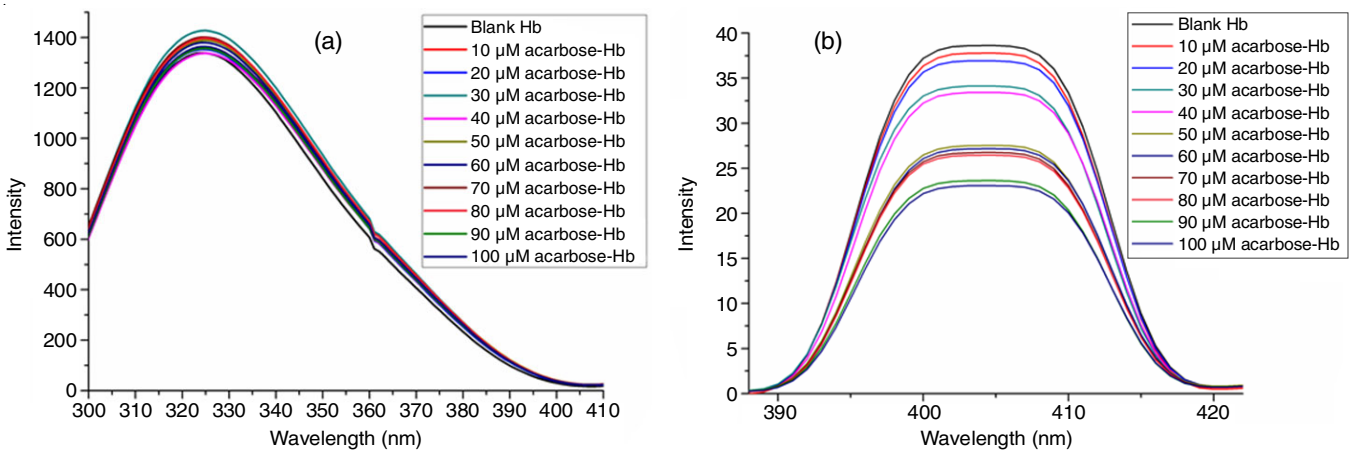

Fig. 3. Effluence on fluorescence intensity of pure Hb at λ_{max} = 280 nm (a) and at λ_{max} = 405 nm (b) with increasing 10 to 100 μM concentration of acarbose

and the static mechanism, which could also be explained by the obtained thermodynamic parameters [35]. To further confirm the structural changes in the protein with acarbose, the UV spectra were also measured at different concentrations [36].

The binding constant (k_b) indicates that proteins interact affinity towards acarbose (Table-1 and Figs. 4-7). These observed spectra revealed that an interaction between acarbose and proteins changed the structure of proteins. The observed results also suggested that, with the addition of acarbose concentrations from 10 to 100 µM, the peptide junctions of BSA and HSA molecules more extended with increasing hydrophilic environment. In addition, for the quenching mechanism elucidation, the observed

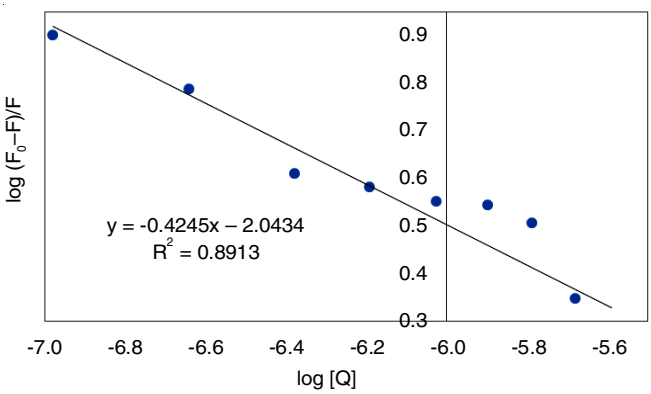

Fig. 4. BSA + Acarbose binding plot of log [(F_0-F)/F] *versus* acarbose log [Q], BSA concentration was fixed (10 µM)

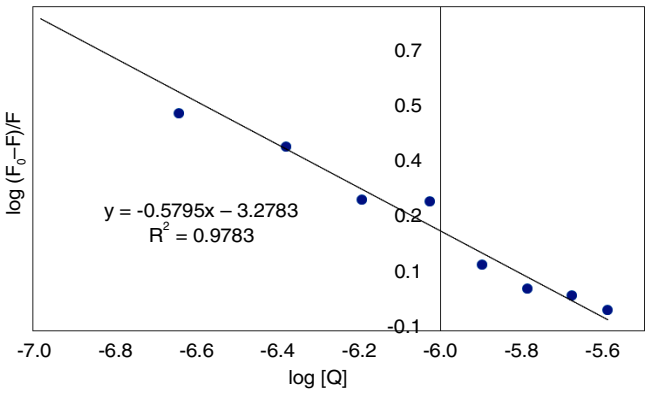

Fig. 5. HSA + Acarbose binding plot of log [(F_0-F)/F] *versus* acarbose log [Q], HSA concentration was fixed (10 µM)

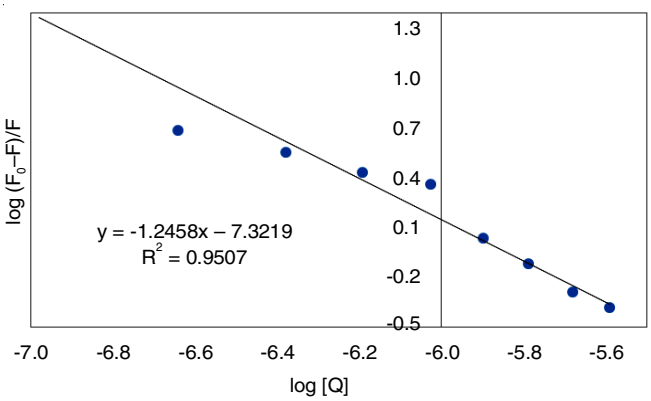

Fig. 6. Hb + Acarbose binding plot of log [(F_0-F)/F] *versus* acarbose log [Q], Hb concentration was fixed (10 µM) at λ_{max} = 280 nm

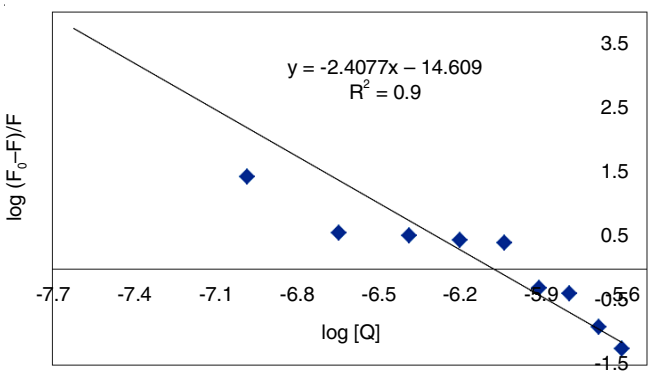

Fig. 7. Hb + Acarbose binding plot of log [(F_0-F)/F] *versus* acarbose log [Q], Hb concentration was fixed (10 µM) at λ_{max} = 405 nm

results are determined by measuring Stern-Volmer equation. Figs. 8-10 show the Stern-Volmer plot of F_o/F *versus* acarbose concentration, where the K_{sv} values obtained as a slope and k_q were calculated based on the fluorescence lifetime. Table-1 shows that k_q of acarbose with different proteins decreased in the order of BSA > HSA > Hb, which suggested the static quenching order of the used proteins [19,31].

The highest k_q observed with BSA reported the diffusion rate constant of various amino acids present in BSA. Hence, the observed results indicate that the quenching occured between acarbose and different proteins *via* complex formation. In

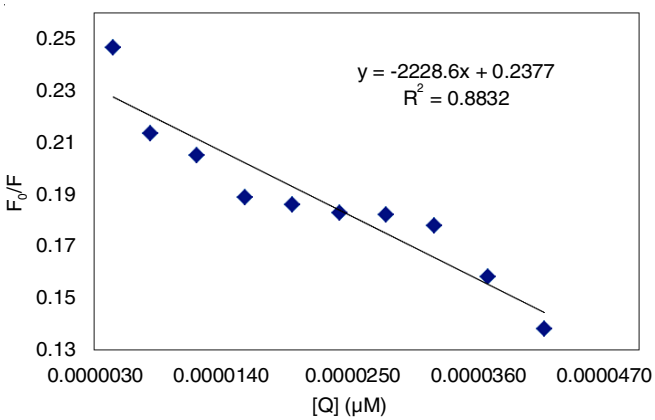

Fig. 8. BSA + Acarbose binding plot: Stern-Volmer plot of F_0/F *versus* [Q], BSA concentration was fixed (10 µM)

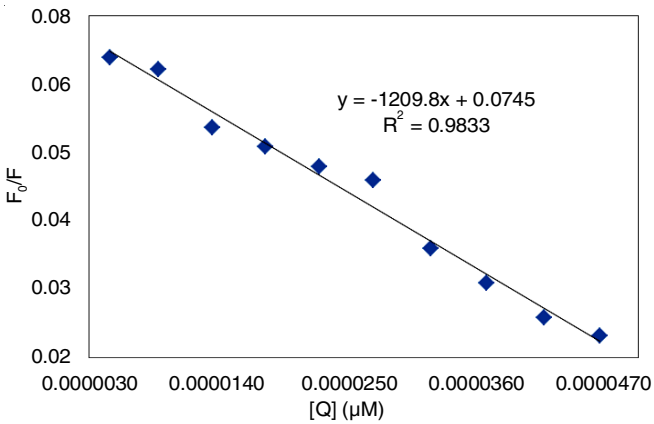

Fig. 9. HSA + Acarbose binding plot: Stern-Volmer plot of F_0/F *versus* [Q], HSA concentration was fixed (10 µM)

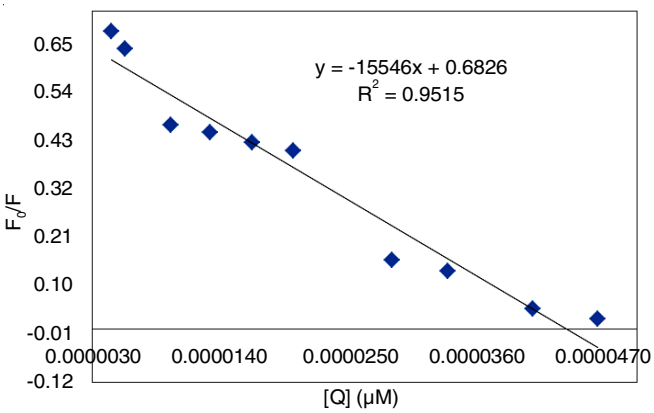

Fig. 10. Hb + Acarbose binding plot: Stern-Volmer plot of F_0/F *vs.* [Q], Hb concentration was fixed (10 μM)

the case of acarbose with Hb, the k_q and K_{sv} values were higher at 405 nm than at 280 nm. These observed results suggested that for acarbose-Hb complexes, the fluorescence quenching intensity of the present amino acid molecules is more effective than that of BSA and HSA.

Acting forces assessments: There are four major non-covalent interactions hydrogen bonds, electrostatic forces, van der Waals forces and hydrophobic interactions play a significant role in drug binding (Figs. 11-13) to proteins could occur [37]. In the present study, the binding modes follow the order:

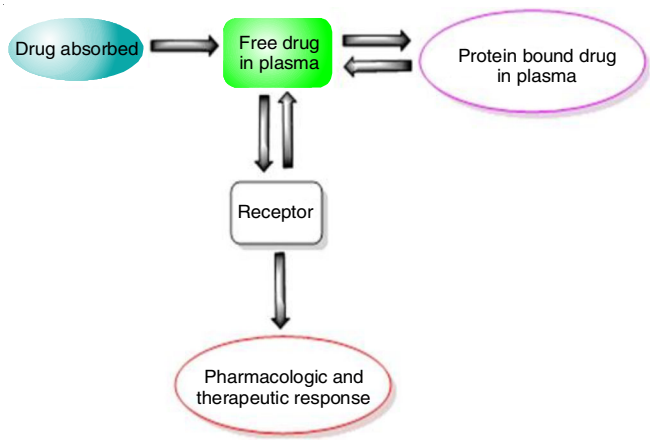

Fig. 12. Protein drug binding process

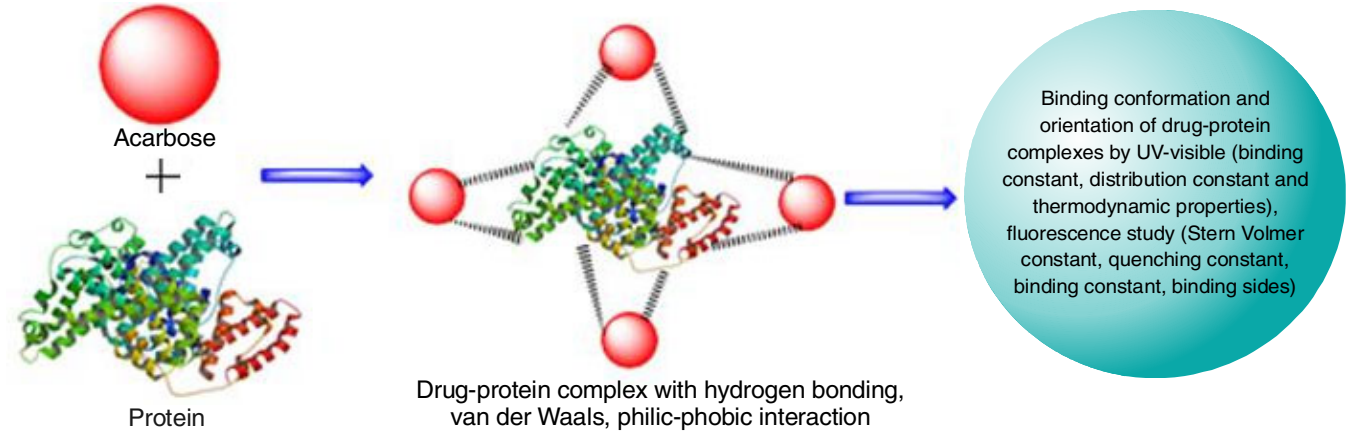

Fig. 11. Graphical representation of drug and protein interactions

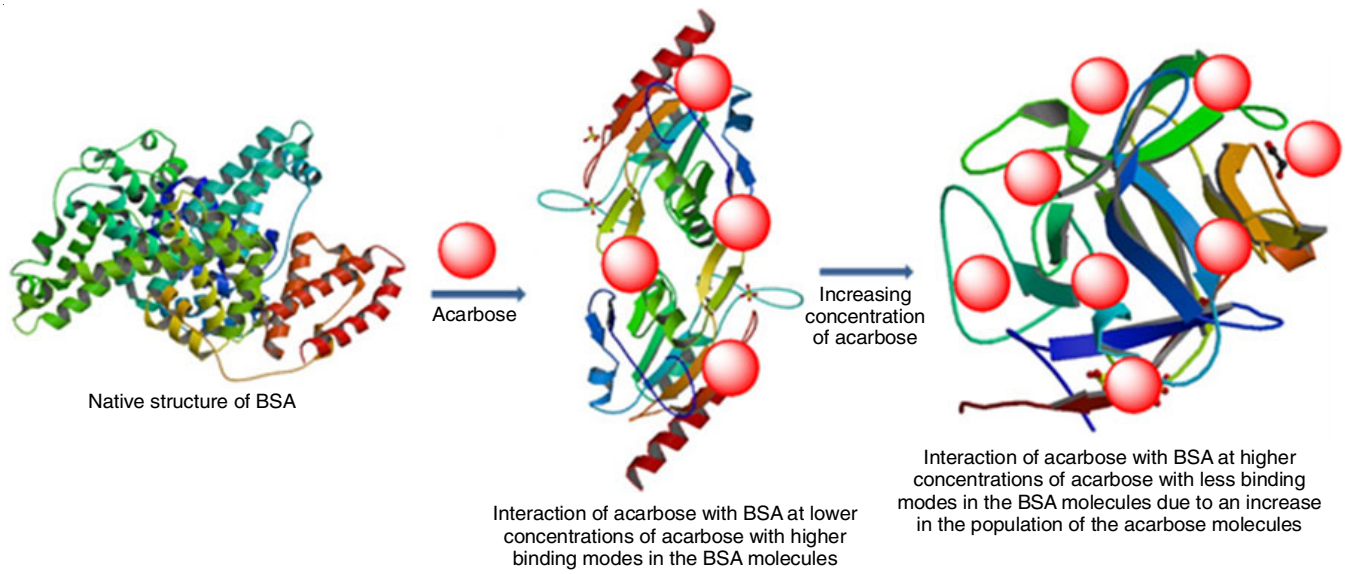

Fig. 13. Influence of drugs on protein structure

Hb λ_{max405} > Hb λ_{max275} > BSA > HSA, such order indicates that λ_{max405} could show stronger interaction than λ_{max405}, BSA, and HSA. Also, increasing the acarbose concentration while the number of binding modes decreases due to increases in the population of acarbose molecules (Table-2 and Figs. 14-15).

TABLE-2
BINDING MODES OF BSA, HSA AND Hb FOR INTERACTION WITH ACARBOSE

Acarbose conc. (μM)	BSA at λ_{max} = 280 nm	HSA at λ_{max} = 280 nm	Hb at λ_{max} = 275 nm	Hb at λ_{max} = 405 nm
0	83.56	76.03	157.07	253.18
10	72.80	74.24	156.72	251.75
20	71.72	74.59	158.87	252.47
30	69.21	72.44	158.51	250.31
40	68.86	71.37	158.87	249.95
50	68.50	69.57	156.00	249.24
60	68.14	68.50	154.21	247.44
70	64.19	67.06	151.34	247.80
80	60.25	68.14	149.18	246.37
90	59.89	66.70	150.26	246.73
100	59.53	65.63	150.62	246.73

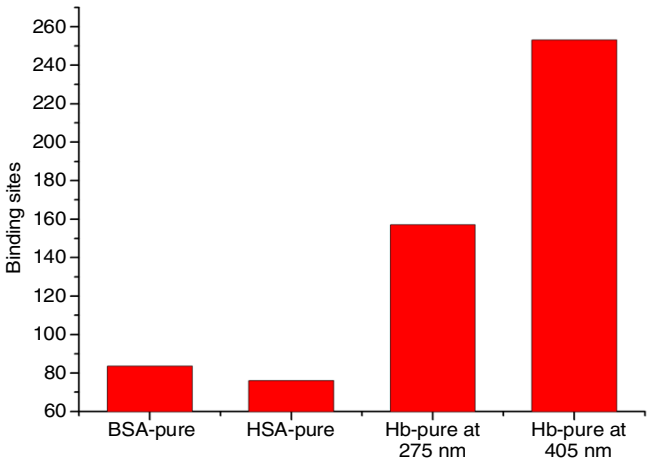

Fig. 14. Number of binding modes of pure-BSA, HSA and Hb

The interactions between acarbose with BSA, HSA and Hb were also explained through thermodynamic parameters [36]. The negative ΔH value infers the exothermic process in the solution whereas the negative ΔG values indicate the thermodynamically favourable process. Thus, the positive ΔS value and ΔH negative values reflected the hydrophobic interaction involved in the drug with protein binding phenomenon [38,39]. In both cases, proteins and acarbose having hydrophobicity which could break the hydrogen bonding. In this situation, an increasing amount of energy is released from the system, more negative values with increasing drug concentration was obtained.

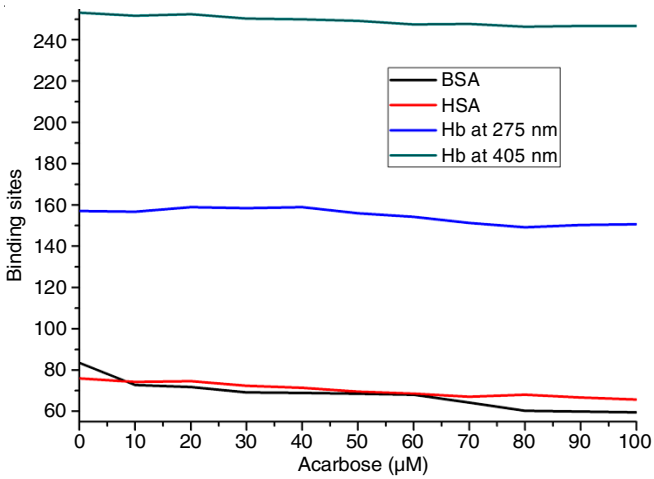

Fig. 15. Number of binding modes of BSA, HSA and Hb with increasing concentration (10-100 μM) of acarbose

Circular dichroism analysis: To understand the structure of biological molecules, circular dichroism (CD) is a well-known spectroscopic analysis for a broad area of biological applications like the characterization of protein structure, α-helix, β-sheet and other sheet determination. In the current study, CD analysis of acarbose and proteins (BSA, HSA and Hb) interactions were determined in the 200-250 nm range with a 0.1 cm path length cell from 10 to 100 μM of acarbose. The secondary structures of BSA, HSA and Hb were estimated from CD spectra (Fig. 16) recorded between 200 and 260 nm, which conforms the secondary structure protein and acarbose complexes. The solution of acarbose and protein complexes with BSA ~ 225 nm minima observed from 10 to 100 μM acarbose concentration confirms the α-helical structure of BSA (Fig. 16c).

Conclusion

The present study provides information related to types of interactions that are involved in drug and protein interactions

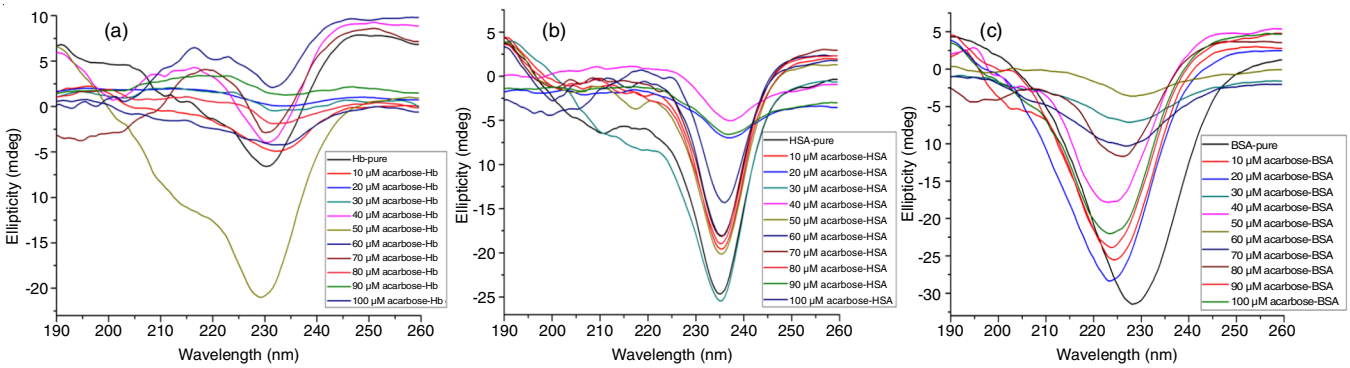

Fig. 16. CD spectra of (a) Hb, (b) HSA and (c) BSA (10 μM in PBS) in the absence and presence of 10-100 μM of acarbose

by using multispectroscopic techniques under thermodynamic conditions. Acarbose significantly quenched the used proteins *via* dynamic and static methods, as confirmed by the Stern-Volmer equation, where the quenching rate constants were measured. The observed thermodynamic parameters of acarbose-protein complexes suggested that hydrogen bonding or other intermolecular interactions play in the interaction of acarbose and proteins with a negative ΔG value of infers the thermodynamically spontaneity in the acarbose-protein interactions. The fluorescence and CD spectra revealed that the structural confirmation was changed by the acarbose addition. An increase in the concentration of acarbose decreases the binding modes of the proteins. Thermodynamic properties indicate that the type of non-covalent bonding between proteins and the acarbose system. Additionally, the negative intercity of the CD spectra different for the free acarbose, which further, suggested the percentage of helices that are a presence in the protein is changed in the presence of acarbose molecules. Fluorescence and CD studies give the depth information about conformational changes in proteins and caused the interactions with drugs. This study could be useful for biological applications, pharmacology and clinical medical trials.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interests regarding the publication of this article.

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