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A Bioinformatics Approach for the Treatment of Thalassemia using Molecular Docking

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ABSTRACT: Thalassemia is a major blood disorder that results from significantly reduced or absent synthesis of either the α- or β-*globin* chains. *Alpha Thalassemia* is most prevalent in people of African and Southeast Asian descent whereas *beta thalassemia* in people of Mediterranean descent. The drugs commonly used for the treatment of thalassemia are deferasirox, deferiprone, desferrioxamine, hydroxyurea, and luspatercept. In this study, we have determined that bioactive compounds from the plant species Coptidis Rhizome can also be used for the treatment of thalassemia. The identification of the specific ligands that bind to specific receptor binding sites and identification of the most favorable ligand with the help of molecular docking was attempted. Docking studies revealed that the best hit molecule for *alpha thalassemia* was the drug Deferasirox and the bioactive Chilenin from CoptidisRhizome and for *beta thalassemia* was the drug Indicaxanthin and the bioactive Berberine from CoptidisRhizome.Due to very limited information available for the mechanism of action of the bioactive compounds of Coptidis Rhizome and risk factors, the present study provides some clues for the treatment of thalassemia.

Keywords: α- and β-*globin* chains, Auto Dock, Coptidis Rhizome, Drugs, Mutation, Thalassemia.

INTRODUCTION

Thalassemia is a genetically inherited disorder which is caused by the defect in the synthesis of one or more of the hemoglobin chains. It is an autosomal recessive disorder which results in excessive destruction of red blood cells lead to mild to severe anemia. *Alpha thalassemia* occurs when there is an absence of *alpha globin* chains which increases the beta globin chains. *Alpha globin* chains are controlled by two genes on chromosome number 16. The deletion of one or more genes causes a deficiency in the *alpha globin* chain production. *Beta thalassemia* occurs when there is an absence of *beta globin* chains which increases the *alpha globin* chains. One gene on each chromosome 11 is controlled by *beta globin* synthesis. Mainly point mutations are responsible for *beta thalassemia* and rarely deletion of two genes (Muncie *et al.*, 2009). Thalassemia cases, in general, have been often found in persons of African, Mediterranean, Southeast Asian descent. Genetic defects leading to *alpha thalassemia* is mostly prevalent in African population. In Sub-Saharan Africa: 0-12% of the population is affected by *beta thalassemia*. In Europe: 0-19% of the population is affected by *beta thalassemia* and 1-2% by *alpha thalassemia*. The Maldives has a particularly high prevalence of thalassemia in Asia (18% of the population), alongside other countries with tropical climates such as India and Thailand (Wong *et al.*, 2011). India has a huge burden with an estimated 100,000 patients with *β thalassemia* syndrome and around 150,000 patients with sickle cell disease, but few among them are optimally managed. The *β-thalassemia* gene carrier rate varies from 1 to 3% in Southern India to 3% to 15% in Northern India. Certain communities in India, such as Sindhis and Punjabis from Northern India, Kutchis, Bhanushali's, Lohana's from Gujarat, Koli's and Agri's from Maharashtra, Neobuddhist's, and Gowda's and Lingayat's from Karnataka, etc. have a higher carrier rate than other communities in India. The Malaysian Indians who were migrants, mainly from Southern India were rarely found to have thalassemia (Colah *et al.*, 2011).

Various types of mutations have occurred in alpha and beta thalassemia. The $\alpha^{3.7}$ is characterized by the deletion of 3804 base pairs. The $\alpha^{4.2}$ is characterized by Leftward crossover between misaligned homologous X boxes, which are 4.2 kb apart on the 2 chromosomes which produces one chromosome with the 4.2 kb deletion and one functional *α-globin* gene (HBA1) (Ou-Yang *et al.*, 2004). The rare ^{SA}(South African deletion) of *alpha thalassemia*, is characterized by a 23-kb deletion which involves the psi zeta, psi alpha 2, psi alpha 1, alpha 1, alpha 2, and theta 1 genes, are present in 13 members [6 simple heterozygotes, 5 with Hb H

disease of the --(SA)/-alpha(-3.7 kb) type, and a pair of Hb H disease of the (SA)/-alpha(-4.2 kb) type]. IVS 1-5 G → C is the most common *beta thalassemia* mutation. The mutations appear to activate the utilization of three "cryptic" donor sites, two in exon 1 and one in Intervening sequence 1, which are used preferentially to the mutated donor site. The 619 bp-deletion, abolishes the 3′ region of the *β-globin* gene and comprises more than 50% of β-thalassemia in some Indian subpopulations. The IVS1-110 G → A is a primary base substitution identified in *β-thalassemia* gene and very common in the Mediterranean population. The G to A substitution creates an alternate acceptor AG, 19 bp 5′ to the conventional acceptor AG of Intervening sequence 1 (Thein *et al*., 2013).

There are mainly six drugs available for the treatment of thalassemia. The most widely used drug is Hydroxyurea. Hydroxyurea could be a cell-cycle specific agent which blocks DNA synthesis by inhibition of the ribonuclease reductase, the enzyme which converts ribonucleotides to deoxyribonucleotides. It's been seen that chronic daily low dose administration of hydroxyurea will enhance gamma globin synthesis, increase red cell production, and partially or substantially correct the anemia in patients with homozygous beta-thalassemia. Hydroxyurea can be a good treatment option for *β-thalassemia* patients as it helps to reduce blood transfusion burden and also prevents disease complications by shifting body metabolism towards normal. Hydroxyurea is also one of the most cost-effective options available in the market (Iqbal *et al*., 2018; Ravangard *et al.,* 2018). Limitations for Hydroxyurea include a rise of % HbF level, hemoglobinand reduce of HbS, WBC, platelet count, serum bilirubin, and LDH levels (Dehury *et al.,* 2015).

Desferrioxamine (DFX) is the main iron-chelating treatment of transfusional iron overload. This medication is prescribed for hemochromatosis due to blood transfusion. It is recommended first-line therapy for hemochromatosis in people with thalassemia major. Deferiprone or deferasirox are used for treatment of iron overload when desferrioxamine is counter positioned or inadequate (Waheed*et al*., 2014; Fisher *et al.,* 2013). Limitations for Desferrioxamine includes major hindrance in achieving optimal therapeutic results. Limitations may also arise related to toxicity, dose, cost, hemochromatosis of the patients, and ineffective removal of excess iron from the heart and other toxic metal absorption (Kontoghiorghe *et al*., 2016). Luspatercept helps to treat anemia in patients who are receiving blood transfusions. It also helps to treat certain sorts of myelodysplastic syndrome. It works by increasing the amount and quality of red blood cells. Limitations for Luspatercept include an increased incidence of bone pain, arthralgia, dizziness, hypertension, and hyperuricemia. Bone pain was generally of short duration and low grade (Cappellini *et al*., 2020). Deferasirox (DFX), which is an oral iron-chelating agent, can be used for both transfusion-dependent and non-transfusion-dependent thalassemia. It binds to iron within the ratio 2:1 and removes it from the bloodstream. It also has a long half-life of 8-16 hour which provides sustained chelation coverage of 24 hours which is a unique feature of DFX. Deferasirox is used for both transfusion-dependent (TDT) and non-transfusion dependent (NTDT) thalassemia. DFX features a favorable side-effect profile in patients with transfusion-dependent thalassemia (TDT), with treatment-related adverse events comprising gastrointestinal, renal, and dermatologic effects that were generally mild and reversible on cessation of treatment. Also, mild, non-progressive increases in serum creatinine levels are often observed (Ricchi*et al*., 2015). Deferiprone is an oral iron chelator. It is used as a second-line agent for the treatment of transfusional iron overload in patients with thalassemia in the United States. Deferiprone is indicated as second-line treatment in patients with thalassemia major, for whom deferoxamine therapy is contraindicated or in patients who present with serious toxicity to deferoxamine therapy (Belmont *et al*.,2017).Some limitations are that it can cause diarrhea, abdominal pain, and joint pain. Deferiprone can cause agranulocytosis which is one of the most concerning side effects and it may also cause milder forms of neutropenia, which need an appropriate monitoring and patient/provider education. Indicaxanthin could be a pigment derived from the cactus pear fruit and might be used as an antioxidant. Dietary indicaxanthin has been shown to own protective effects on RBCs in people with *beta thalassemia*. Indicaxanthin can reduce the preferred-Hb, a reactive intermediate, back to met-Hb. the general effect of this step is that Hb degradation is prevented, which helps prevent the accelerated breakdown of RBCs. Besides, indicaxathin has been shown to scale back oxidative damage in cells and tissues and does so by binding to radicals. The mechanism of its function, however, continues to be unknown. Indicaxanthin has high bioavailability and minimal side effects, like vomiting or diarrhea (Tesoriere *et al*., 2006)

Coptidis Rhizome (CR), is a Rhizome of species *Coptischinensis, C. deltoidea or C. teeta*. It belongs to the family of Ranunculaceae. This CR is consumed largely in Asian countries like China, Japan, Malaysia, Singapore and India, and only a small amount is used in European countries. The Coptidis Rhizome is commonly used in the Traditional Chinese Medicine (TCM) for treating various diseases including typhoid, tuberculosis, pertussis, bacillary dysentery and other

inflammatory related disorders. In the recent studies, it also is known to have showed wide pharmacological activities, such as antimicrobial, anti-hepatic steatosis, anti-myocardial ischemia, antidiabetic, anti-inflammation, antioxidation and anti-tumor effects. There have been many compounds isolated from CR and they include alkaloids, ligands, flavones, phenylpropanoids, volatile oils, and others (Wang *et al*., 2019). The Coptidis Rhizome is known to contain bioactive compounds such as alkaloids, phenylpropanoids, flavonoids and other compounds. The most abundantly found alkaloids bioactive molecules are berberine, coptisine, palmatine, epiberberine, columbamine, jatrorrhizine, chilenin; phenylpropanoids include ferulic acid, woorenoside I, chlorogenic acid, woorenogenin; flavonoids include rhamnetin, wogonin; other compounds such as vanillic acid. All these bioactive compounds tend to have pharmacological activities, especially berberine is known to have anti-pathogenic and antibacterial effects. Along with berberine, coptisine, palmatine and jatrorrhizine also have these antibacterial activities (Meng *et al*., 2018). They not only have anti-pathogenic and antibacterial activities they are also known for their anti-obesity, anti-hyperlipidemic effect, anti-atherosclerotic effect, antidiabetic, anticancer and other pharmacological activities including the study of Alzheimer's disease as well as on pharmacokinetics research. Considering the above background an attempt was made to identify the specific ligands that bind to specific receptor binding sites and also to identify the most favorable ligand with the help of molecular docking.

METHODOLOGY

Our work plan is recognizing the particular ligands that bind to specific receptor binding sites and recognize the foremost favorable ligand with the assistance of molecular docking. Molecular docking explores the behavior of small molecules in the binding site of the targeted protein and is used as a very important tool for drug discovery (Pagadala *et al.*, 2017). Docking against homology modeled targets can be done for proteins whose structures are not known. Molecular docking tools help to get a collection of various ligand binding poses and use a scoring function to estimate binding affinities for the generated ligand poses to see the most effective binding mode. Computational docking may be utilized to predict bound conformations and free energies of binding for small-molecule ligands to macromolecular targets. Docking is widely used for the study of biomolecular interactions and mechanisms, and it's applied to structure-based drug design. The AutoDock semi-empirical field of force includes intramolecular terms, a "full" desolvation model, and also considers directionality in hydrogen bonds (Morris *et al*., 2008).

To perform AutoDock, AutoDock 4.2, and MGI Tools were installed. The software has been downloaded fromhttp://autodock.scripps.edu/. The method of AutoDock was completed as mentioned below.

(a) Preparation of the Protein: The SDF file of our target protein was downloaded from the PubChem database and it had been converted to PDB format using Openbabel software. Then, we prepared our target protein by the addition of hydrogen atoms, Kollman charges, and Gasteiger charges within the PDB file of our protein. Then we saved a PDBQT file of our target protein.

(b) Preparation of Ligand: The SDF file of the varied bioactive from Coptidis Rhizome and also the available drugs were downloaded from the Pubchem database and that they were converted to PDB format using Openbabel software. Then we prepared the PDBQT get into the identical manner by choosing torsions and detecting the basis and showing root expansions.

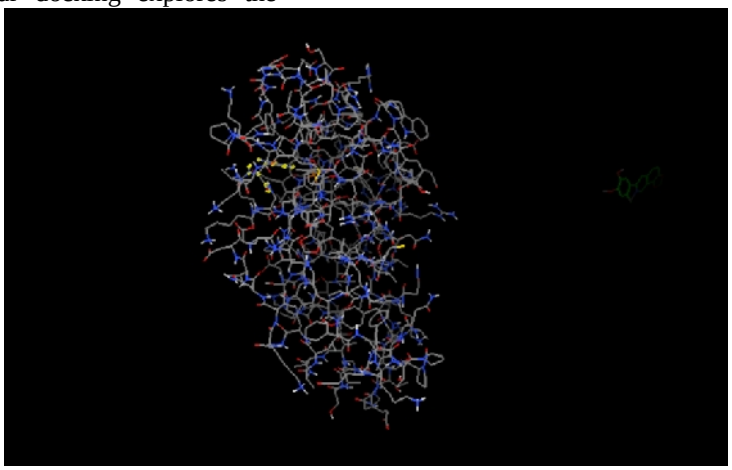

Fig. 1. Preparation of hemoglobin alpha chain with ligand epiberberine from Coptidis Rhizome.

(c) Generating a grid parameter file: Next, we've got to define the 3D space that AutoDock considers for the docking, typically, a volume around the potential binding site of a receptor.

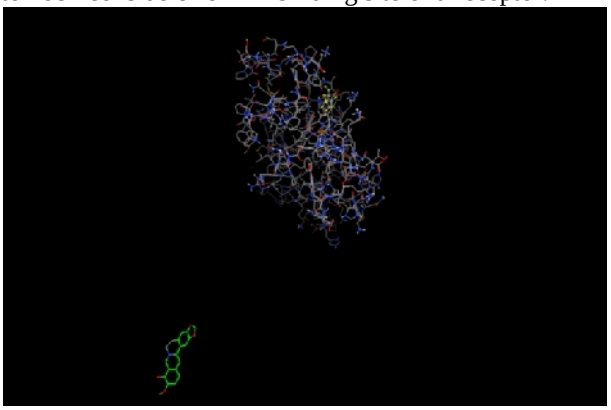

Fig. 2. Preparation of hemoglobin beta chain with ligand berberine from CoptidisRhizome.

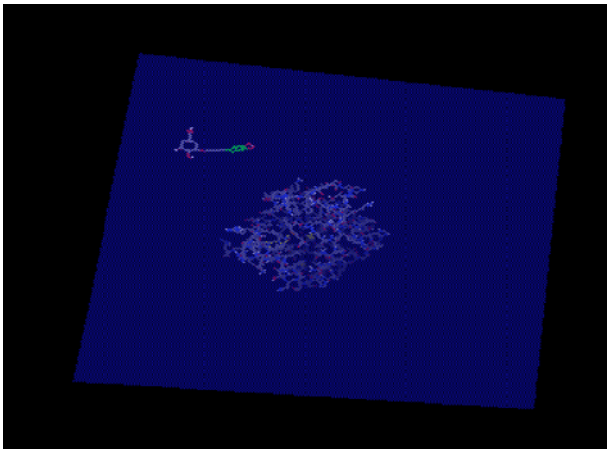

Fig. 3. Preparation of grid box of hemoglobin alpha chain with epiberberine.

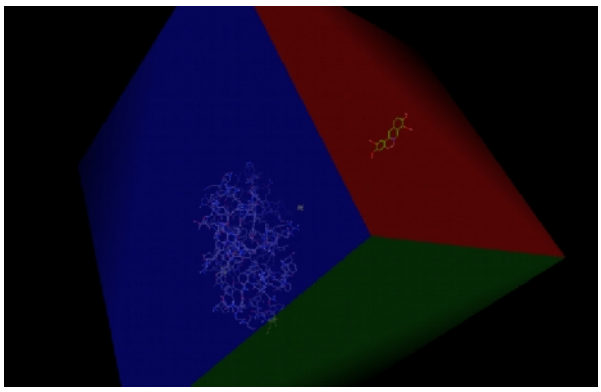

Fig. 4. Preparation of grid box of hemoglobin beta chain berberine.

(d) Generating maps and grid data files: during this step, we'd like to save lots of our grid as grid.gpf
(e) Generating a docking parameter file: during this step, we'd like to arrange the docking parameter file that bundles the knowledge that's required by AutoDock as docked.dpf.
(f) Running AutoDock: during this step, we've got created a full bunch of various files and were able to score the protein-ligand complex.
(g) Generating the results: we are able to analyze our docking information by opening us .dlg file. At the very bottom, we will see the estimated free energy of binding together with the opposite component that's included within the calculation of the previous.

RESULTS AND DISCUSSION

We prepared our target proteins, i.e., Hemoglobin alpha chain, and Hemoglobin beta chains. The binding pattern of the complex structure of both Hemoglobin alpha

chain and Hemoglobin beta chain was explored by means of LigPlot+ program(https://www.ebi.ac.uk/thornton-srv/software/LigPlus/download.html).

We have used Indicaxanthin as the ligand. In both the structures, hydrogen bond is present. The binding packets are essential for ligand binding. Here, 13 binding packets are available for hemoglobin alpha chain and 15 binding packets are available for hemoglobin beta chain. We have found that His46A chain and His88A chain are one of the best residue chains for hemoglobin alpha chain. His93D chain is one the best residue chains for hemoglobin beta chain (Figs. 5 and 6).

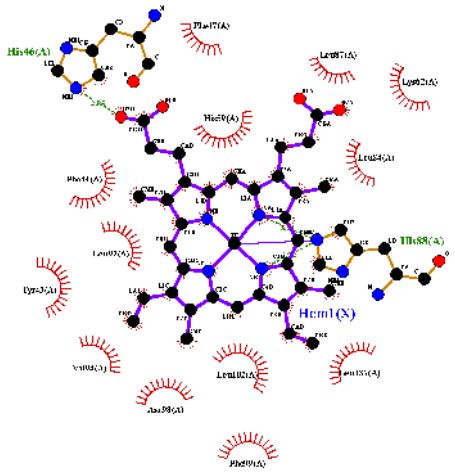

Fig. 5. Interaction pattern of hemoglobin alpha chain with Indicaxanthin as ligand.

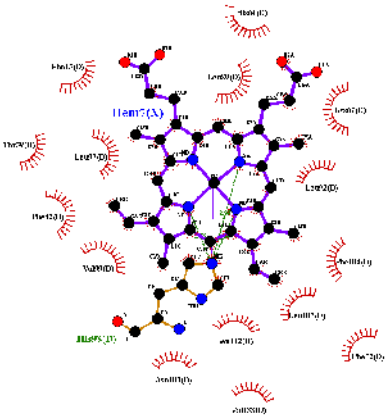

Fig. 6. Interaction pattern of hemoglobin beta chain with Indicaxanthin as ligand.

The binding energy parameter is used to determine which ligand has a stable complex interaction with protein. The more negative value or lower binding affinity, the more stable ligand-receptor is achieved (Tables 1, 2, 3 and 4). Greater intermolecular force between the ligand and its receptor results in High-affinity ligand binding and less intermolecular force between the ligand and its receptor results in low-affinity ligand binding. The most negative free energy change and minimum inhibition constant is the desirable conformation of binding. A better docking score corresponds to Low Binding Energy. Here, the hit molecule of alpha hemoglobin is Deferasirox with binding energy of -6.87 Kcal/mol. The Mean Binding Energy is -6.84 Kcal/mol. The estimated Inhibition Constant, Ki is 9.17 uM. The hit molecule for beta hemoglobin is Indicaxanthin. Indicaxanthin has a binding energy of -5.13 Kcal/mol in Hemoglobin beta chain. The estimated Inhibition Constant, Ki is 173.47 uM. The Mean Binding Energy is -4.69 Kcal/mol.

Among the bioactives derived from Coptidis Rhizome, the hit molecule of alpha hemoglobin is Chilenin. Chilenin has a binding energy of -6.33 Kcal/mol in the hemoglobin alpha chain. The estimated Inhibition Constant, Ki is 22.75 uM. The Mean Binding Energy is -6.11 Kcal/mol. The hit molecule beta hemoglobin is Berberine. Berberinehas a binding energy of -6.00 Kcal/mol in the hemoglobin beta chain. The estimated Inhibition Constant, Ki is 40.16 uM. The Mean Binding Energy is -5.98 Kcal/mol.

Very few works have been previously done for the treatment of thalassemia with the help of bioactive compounds from Coptidis Rhizome. One research work revealed for patients with chronic cytopenic hematological conditions that berberine is a safe bioactive compound without any toxicity and electrolyte imbalance when administered with the herbal concoction of Coptidis Rhizome for 1252 days. Based on traditional dosage and indication, this herb is safe for oral concoction (Linn *et al.*, 2012). Comparing this work, we have also found out that berberine is the best hit molecule for beta hemoglobin and can be an effective treatment option for thalassemia.Chilenin has demonstrated cytotoxic activity against human tumor cell lines with IC50 values in the micromolar range (Piko *et al.*, 2009). This activity of Chilenin can be used as a cytological tool affecting hematological disorders.

Table 1: Binding Energy of Bioactives from Coptidisrhizome With Hemoglobin *Alpha Chain.*

Sr. No.	Bioactives	Binding Energy (Kcal/mol)
1	Berberine	-5.8
2	Coptisine	-6.31
3	Epiberberine	-6.05
4	Columbamine	-5.52
5	Ferulic Acid	-6.23
6	Jatrorrhizine	-5.23
7	Palmatine	-5.61
8	Rhamnetin	-4.29
9	Vanillic Acid	-4.52
10	Wogonin	-5.3
11	Wooregenin	-3.87
12	Chilenin	-6.33

Table 2. Binding energy of bioactives from coptidisrhizome with hemoglobin *beta chain.*

Sr. No.	Bioactives	Binding Energy(Kcal/mol)
1.	Berberine	-6.0
2.	Coptisine	-5.49
3.	Epiberberine	-5.8
4.	Apocyanol	-2.06
5.	Ferulic Acid	-4.1
6.	Jatrorrhizine	-5.4
7.	Palmatine	-5.32
8.	Rhamnetin	-3.99
9.	Vanillic Acid	-0.65
10.	Wogonin	-4.91
11.	Wooregenin	-2.72
12.	Chilenin	-4.42

Table 3: Binding energy of available drugs with hemoglobin *alpha chain.*

Sr. No.	Available Drugs	Binding Energy(Kcal/mol)
1.	Deferasirox	-6.87
2.	Deferiprone	-3.93
3.	Desferrioxamine	-1.35
4.	Hydroxyurea	-3.36
5.	Indicaxanthin	-6.08

Table 4: Binding energy of available drugs with hemoglobin *beta chain.*

Sr. No.	available drugs	binding energy(kcal/mol)
1.	deferasirox	-4.72
2.	deferiprone	-3.34
3.	desferrioxamine	0.44
4.	hydroxyurea	-2.82
5.	indicaxanthin	-5.13

CONCLUSION

Concluding, we can say that the results of this study appear to support the usage of Coptidis Rhizome for the treatment of thalassemia and its related conditions. Coptidis Rhizome has tremendous potential as anti-atherosclerotic, anti-obesity, maintaining lipid effect, and anti-hepatic steatosis effect. Among the bioactive compounds, Chilenin and Berberine was determined to be best for treatment of thalassemia compared to the already available drugs available. Since, thalassemia is a huge burden in many developing countries such as India, this traditional remedy can be exploited to develop a wide range of drugs which in turn will help reduce the global prevalence of thalassemia. In some cases, Coptidis Rhizome has been termed as safe for oral concoction based on traditional dosage and indication. This option can be cost effective solution particularly in developing countries which can be a huge future prospect. Thus, effective, safe and affordable treatment options can be given to thalassemia patients by using this approach.

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Conflict of interest. There is no conflict of interest to declare. Both authors have seen and approved the manuscript being submitted.

REFERENCES

Belmont, A., & Kwiatkowski, J. L. (2017). Deferiprone for the treatment of transfusional iron overload in thalassemia. *Expert review of hematology*, *10*(6), 493-503.

Cappellini, M. D., Viprakasit, V., Taher, A. T., Georgiev, P., Kuo, K. H., Coates, T., ... & Piga, A. (2020). A phase 3 trial of luspatercept in patients with transfusion-dependent β-thalassemia. *New England Journal of Medicine*, *382*(13), 1219-1231.

Colah, R., Italia, K., & Gorakshakar, A. (2017). Burden of thalassemia in India: the road map for control. *Pediatric Hematology Oncology Journal*, *2*(4), 79-84.

Dehury, S., Purohit, P., Patel, S., Meher, S., Kullu, B. K., Sahoo, L. K., & Patel, D. K. (2015). Low and fixed dose of hydroxyurea is effective and safe in patients with HbSβ + thalassemia with IVS1· 5 (G→ C) mutation. *Pediatric blood & cancer*, *62*(6): 1017-1023.

Fan, W., Fan, L., Peng, C., Zhang, Q., Wang, L., Li, L., & Wu, C. (2019). Traditional uses, botany, phytochemistry, pharmacology, pharmacokinetics and toxicology of Xanthium strumarium L.: A review. *Molecules*, *24*(2), 359.

Fisher, S. A., Brunskill, S. J., Doree, C., Gooding, S., Chowdhury, O., & Roberts, D. J. (2013). Desferrioxamine mesylate for managing transfusional iron overload in people with transfusion-dependent thalassaemia. *Cochrane Database of Systematic Reviews*, (8).

Herbert, L., Muncie, J. R., & Campbell, J. S. (2009). Alpha and beta thalassemia. *Am Fam Physician*, *80*(4), 339-44.

Ou-Yang, H., Hua, L., Mo, Q. H., & Xu, X. M. (2004). Rapid, accurate genotyping of the common− α4. 2 thalassaemia deletion based on the use of denaturing HPLC. *Journal of clinical pathology*, *57*(2), 159-163.

Iqbal, A., Ansari, S. H., Parveen, S., Khan, I. A., Siddiqui, A. J., & Musharraf, S. G. (2018). Hydroxyurea treated β-thalassemia children demonstrate a shift in metabolism towards healthy pattern. *Scientific reports*, *8*(1), 1-9.

Kontoghiorghe, C. N., & Kontoghiorghes, G. J. (2016). Efficacy and safety of iron-chelation therapy with deferoxamine, deferiprone, and deferasirox for the treatment of iron-loaded patients with non-transfusion-dependent thalassemia syndromes. *Drug design, development and therapy*, *10*, 465.

Linn, Y. C., Lu, J., Lim, L. C., Sun, H., Sun, J., Zhou, Y., & Ng, H. S. (2012). Berberine-induced haemolysis revisited: safety of Rhizoma coptidis and cortex phellodendri in chronic haematological diseases. *Phytotherapy Research*, *26*(5), 682-686.

Meng, F. C., Wu, Z. F., Yin, Z. Q., Lin, L. G., Wang, R., & Zhang, Q. W. (2018). Coptidis rhizoma and its main bioactive components: recent advances in chemical investigation, quality evaluation and pharmacological activity. *Chinese Medicine*, *13*(1), 1-18.

Morris, G. M., & Lim-Wilby, M. (2008). Molecular docking. In *Molecular modeling of proteins* (pp. 365-382). Humana Press.

Pagadala, N. S., Syed, K., & Tuszynski, J. (2017). Software for molecular docking: a review. *Biophysical reviews*, *9*(2), 91-102.

Piko, B., & Leonard, M. (2009). Studies directed toward the total synthesis of chilenine.

Ravangard, R., Mirzaei, Z., Keshavarz, K., Haghpanah, S., & Karimi, M. (2018). Blood transfusion versus hydroxyurea in beta-thalassemia in Iran: a cost-effectiveness study. *Hematology*, *23*(7), 417-422.

Ricchi, P., & Marsella, M. (2015). Profile of deferasirox for the treatment of patients with non-transfusion-dependent thalassemia syndromes. *Drug design, development and therapy*, *9*, 6475.

Tesoriere, L., Allegra, M., Butera, D., Gentile, C., & Livrea, M. A. (2006). Cytoprotective effects of the antioxidant phytochemical indicaxanthin in β-thalassemia red blood cells. *Free radical research*, *40*(7), 753-761.

Thein, S. L. (2013). The molecular basis of β-thalassemia. *Cold Spring Harbor perspectives in medicine*, *3*(5), a011700.

Waheed, N., Ali, S., & Butt, M. A. (2014). Comparison of deferiprone and deferrioxamine for the treatment of transfusional iron overload in children with beta thalassemia major. *Journal of Ayub Medical College Abbottabad*, *26*(3), 297-300.

Wong, L. P., George, E., & Tan, J. A. M. A. (2011). Public perceptions and attitudes toward thalassaemia: Influencing factors in a multi-racial population. *BMC Public Health*, *11*(1), 1-9.

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