tions [17], while in our sample of the North region the frequencies were21.2 % and0.13 % respectively.

The frequency of the same blood groups in diseased patient that resident in medicine department of the central hospital of the city (Table 2) was that of blood group O (50.70%) followed by blood group A and B (18.22% and 20.00% respectively) and the lowest of blood group AB (11.08%). Among the Rhesus phenotype, the majority (87.01%) are Rhesus positive. The frequency of coexisting ABO/Rhesus phenotypes were calculated and the highest was that of O+ (43.69%) followed by A+ (15.80%) and B+ (18.09%) and AB+(9.42%). The blood groups O- , A-, B- and AB- occurred at lower frequency of 7.01%, 2.42%, 1.91% and 1.66% respectively.

Table 2: Prevalence of the phenotype of ABO and Rh alleles in diseased population

Phenotype		Nov.2010		Dec.2010		Overall	
		Freq.	%	Freq.	%	Freq.	%
O	Rh+	135	38.24	208	48.15	343	43.69
	Rh-	20	5.67	35	8.10	55	7.01
Total O phenotype%							50.70%
A	Rh+	66	18.70	58	13.43	124	15.80
	Rh-	7	1.98	12	2.78	19	2.42
Total A phenotype%							18.22%
B	Rh+	78	22.10	64	14.81	142	18.09
	Rh-	7	1.98	8	1.85	15	1.91
Total B phenotype%							20.00%
AB	Rh+	32	9.06	42	9.72	74	9.42
	Rh-	8	2.27	5	1.16	13	1.66
Total AB phenotype%							11.08%
Total		353	100%	432	100%	785	100%

In the comparison the results of our study between ABO frequencies between healthy and diseased individuals we saw high deference in blood group type O frequency which it was 67.95% in healthy group in comparison with 50.70% with high significance result (P > 0.01). This unexpected result (*at least for us*)include also the other types of blood group A, B, and AB. In addition to this we observed an inverted percent for blood group A and B. In normal population normally the frequency of type A exceed type B all over the world but in this study we saw increasing type B frequency against type A (P > 0.01).

Also there is a significant difference (P > 0.05) in overall blood Rhesus positive between healthy individual 78.2% and diseased population 87.01% .In regions that are highly endemic for *Plasmodium falciparum* malaria , it is well recognized that a range of red blood cell polymorphisms associated with resistance to severe disease have undergone positive selection[18]. Moreover , as mentioned before, the blood group O proved to be protective factor against sever malaria[19, 20].However, since in the Brazilian Amazon region malaria predominates in Mesoendemic condition with wide variation in transmission, malaria endemicity could be viewed as a selective pressure for maintenance of the observed frequencies of genotypes of the ABO system , which could be very interesting as the focus of a new investigation, including analysis of the genotypes in severe and non- severs malaria patients, as well as in individuals living in nonendemic areas.

In the comparison of the same blood group frequency in thalassemia individuals (Table 3), which it was that of blood group O (35.38%) followed by blood group A (31.60) , B (26.42)% and the blood group AB (6.60%). Among the Rhesus phenotype, the majority (89.62%) are Rhesus positive. The frequency of coexisting ABO/Rhesus phenotypes were calculated and the highest was that of O+ (33.49%) followed by A+ (27.36%) and B+ (23.11%) and AB+(5.66%). The blood groups O-A-, B- and AB- occurred at lower frequency of 1.89%, 4.25%, 3.30% and 0.94% respectively.

Table 3: Prevalence of the phenotype of ABO and Rh alleles in thalassemic patients

Phenotype		Thalassemia		Total type frequency	Blood type %
		Freq.	%		
O	Rh+	71	33.49	75	35.38
	Rh-	4	1.89		
A	Rh+	58	27.36	67	31.60
	Rh-	9	4.25		
B	Rh+	49	23.11	56	26.42
	Rh-	7	3.30		
AB	Rh+	12	5.66	14	6.60
	Rh-	2	.94		
Total		212	100%		100%

In comparison the result of ABO blood types between normal and thalassemic (*A* genetic disease marked by failure to produce a functional mRNA for one of the two major adult hemoglobin proteins, α-globin or β-globin.) patients we saw a dramatic drop in frequency of blood group type O in thalassemic individuals (35.38%) in

comparison with the frequency of the same blood group in normal population (56.41%), (P. > 0.01). And we saw an increase frequencies of type A and B (31.60 % and 26.42 %) in thalassemic person in comparison with the same blood group frequencies in healthy individuals (15.38% and 10.26%) respectively, with high significance results, (P. > 0.01). In comparison overall blood Rhesus positive (Rh+) frequency (Table 4) between both healthy individual (78.2%) and thalassemic individuals (89.62), there was a significant difference (P > 0.05) between them.

Table 4: Overall blood Rhesus positive (Rh+) frequency betweendifferent groups of population

Sample type(No.)	Number of Rh+	Number of Rh-	Rh+%
Normal individuals (78)	61	17	78.2%
Diseased patient (785)	683	102	87.01%
Thalassemic patient (212)	190	22	89.62

This data may give an indicator that individuals with blood group type O may have a genetic resistance against thalassemia.

Also there are a significant deviation in frequencies among blood types in diseased population especially type O which recorded a high decrease in frequency in comparison with healthy people, also the same opinion has been observed for A and B blood group.

Iraqi people “in general” have less blood group type O than Hujazi or Kuwati people, and because of this type of blood group have more resistance survival phenomena (have more phenotype frequency than the expected genotype one).When the expected change gene frequency in 1 generation is calculated [21]**,** allowing selection to work against the dominant phenotypes A and B, or favors the recessive and q to be =0.9 and s=0.1 , the resultant value is about 0.008. This small change, in the presence of other systematic and dispersive processes, is too weak to be noticed, or to produce a drastic shift in the present gene frequencies. On these bases it is expected that the persistency of the polymorphic state and the 3 other alleles (A, B and O) will remain in the population for many generations.

References

[1] Athreya B, Coriell LL. Relation Of Blood Groups To Infection, I. A Survey And Review Of Data Suggesting Possible Relationship Between Malaria And Blood Groups American journal of epidemiology 1967; 86: 292-304.
[2] Cserti CM, Dzik WH. The ABO blood group system and Plasmodium falciparum malaria.Blood 2007; 110: 2250-8.
[3] Yamamoto F-i, McNeill PD, Hakomori S-i. Genomic organization of human histo-blood group ABO genes.Glycobiology 1995; 5: 51-8.
[4] Olsson ML, Chester MA. Frequent occurrence of a variant O1 gene at the blood group ABO locus.Voxsanguinis 1996; 70: 26-30.
[5] Chester MA, Olsson ML. The ABO blood group gene: a locus of considerable genetic diversity.Transfusion medicine reviews 2001; 15: 177-200.
[6] Yamamoto F. Review: ABO blood group system--ABH oligosaccharide antigens, anti-A and anti-B, A and B glycosyltransferases, and ABO genes. Immunohematology/American Red Cross 2003; 20: 3-22.
[7] Loscertales M, Owens S, O'Donnell J, Bunn J, BoschâCapblanch X, Brabin BJ. ABO blood group phenotypes and Plasmodium falciparum malaria: unlocking a pivotal mechanism. Advances in parasitology 2007; 65: 1-50.
[8] LellB, May Jr, Schmidt-Ott RJ, et al. The role of red blood cell polymorphisms in resistance and susceptibility to malaria.Clinical Infectious Diseases 1999; 28: 794-9.
[9] Al-Ezzy AIA, Al-Samarrae S. The Genetic Association between Baldness, Coronary Heart Diseases and ABO Blood Group System in Men in Baqubah City-Diyala Province in Iraq.Journal of Biology, Agriculture and Healthcare 2015; 5: 174-7.
[10] Camargo EP, Alves F, da Silva LHP. Symptomless Plasmodium vivax infections in native Amazonians.The Lancet 1999; 353: 1415-6.
[11] Steel RGDT, J.H. .Principles and Procedures of Statistics New York: McGraw Hill 1980.
[12] Bahaj AA. ABO and Rhesus groups distribution in Hadhramut population. Hadhramut for studies and researches 2003.
[13] Zago M, Costa F, Tone L, Bottura C. Hereditary hemoglobin disorders in a Brazilian population. Human heredity 1983; 33: 125-9.
[14] Franco RF, Sim¡´es BP, Guerreiro JF, Santos SE, Zago MA. Molecular bases of the ABO blood groups of Indians from the Brazilian Amazon region. Voxsanguinis 1994; 67: 299-301.
[15] Olsson ML, Santos SE, Guerreiro JF, Zago MA, Chester MA. Heterogeneity of the O alleles at the blood group ABO locus in Amerindians.Voxsanguinis 1998; 74: 46-50.
[16] Batista Dos Santos SE, Rodrigues J, Ribeiro Dos Santos A, Zago M. Differential contribution of indigenous men and women to the formation of an urban population in the Amazon region as revealed by mtDNA and Yâ€ـdNA. American journal of physical anthropology 1999; 109: 175-80.
[17] Mattos LC, Sanchez FbE, Cintra JR, Salles AB, Bonini-DomingosCuR, Moreira HW. Genotipagem do locus ABO (9q34. 1) emdoadores de sangue da regionoroeste do Estado de S¡£o Paulo. Rev Bras HematolHemoter 2001; 23: 15-22.
[18] MinâOo G, Gros P. Erythrocyte variants and the nature of their malaria protective effect. Cellular microbiology 2005; 7: 753-63.
[19] Barragan A, Kremsner PG, Wahlgren M, Carlson J. Blood Group A Antigen Is a CoreceptorinPlasmodium falciparum Rosetting. Infection and Immunity 2000; 68: 2971-5.

[20] Chung WY, Gardiner DL, Hyland C, Gatton M, Kemp DJ, Trenholme KR. Enhanced invasion of blood group A1 erythrocytes by Plasmodium falciparum. Molecular and biochemical parasitology 2005; 144: 128-30.
[21] Falconer DS.Introduction to Quantiatative Genetics.. Edinburgh, Scotland.: Oliver and Boyd, 1960.