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The Influence of Blood Groups on Diseases Pattern Globally: A Review of Literature

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Authors' contributions

This work was carried out in collaboration among all authors. Author JIA conceived the study, performed literature search, wrote initial manuscript draft. Author DCO proof edited the initial manuscript. All authors read and approved the final manuscript.

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ABSTRACT

The present survey reports the influence of blood groups on disease pattern in worldwide basis. Genetic factors have been associated with vulnerability or resistance to certain disease. For example, individuals who are heterozygous to haemoglobin S and haemoglobin E are resistant to infection with *Plasmodium falciparum.* The manifestation of metabolic syndrome, a sickness characterized by multiple risk factors like obesity, hypertension and glucose intolerance is also influenced by genetic and environmental factors. Several studies have established a relationship between ABO blood type and incidences of *Plasmodium falciparum* infection. This review brought to the fore the relationship between blood groups and susceptibility or resistance to disease. There is need for more studies to be carried out to unravel the exact mechanism how blood groups affect disease. It is recommended that individuals of various blood groups should know the different disease that they are prone to and avoid the predisposing factors to such diseases.

Keywords: Blood group; disease pattern; haemoglobin; microaerophilic bacteria.

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1. INTRODUCTION

Genetic factors have been associated with vulnerability or resistance to certain disease. For example, individuals who are heterozygous to haemoglobin S and haemoglobin E are resistant to infection with *Plasmodium falciparum* [1]. Also children with blood group AB have been reported to be more susceptible to helminthic infections like hookworm, *S. mansoni* and *Ascaris lumbricoides* than other blood groups. The reason for this, according to Haseeb et al. [2] is that the parasite contains polysaccharide that resembles substances in blood group A and that result in inability of immune system to recognize the parasite as foreign and launch appropriate immune response.

Blood group O accounts for 65% of blood type globally and 65.5% among Nigerians studied [3]. Reports also have it that during the outbreak of gastroenteritis caused by Escherichia coli O_{157} strain in Scotland in 1996 people with O blood group were more susceptible [4]. There are reports of greater susceptibility to influenza virus by secretors of ABH substances while non-secretors are known to be resistant to UTI caused by *Escherichia coli* [5]. The susceptibility or resistance to the diseases is the nature of antigen on their red cells. Those with certain carbohydrate antigen are vulnerable or resistant to some disease while those without the antigen are vulnerable or resistant to others. Reasons given for these differences in susceptibility are convincing for some cases while for some, more studies need to be carried out to unravel the mechanisms behind resistance or vulnerability.

The manifestation of metabolic syndrome, a sickness characterized by multiple risk factors like obesity, hypertension and glucose intolerance is also influenced by genetic and environmental factors. For instance, while most Africans affected by the disease manifest with stroke, most Asians are known to come down with heart attack [6]. Dementia and cognitive impairment is also known to be common among people with blood group AB, B and A than group O [7]. Polymorphism and mutation in the glucose 6 phosphate dehydrogenase gene has given rise to protection against falciparum malaria in India and Mediterranean region. Vulnerability or resistance to disease has been advanced as the reason for the differences in the distribution of blood group worldwide. A study relates ABO blood group to incidences of tuberculosis [8]. The differences in the geographical distribution of blood groups may be consequences of epidemic that had occurred in the past [9]. The low prevalence of group O and the high prevalence of group B in the Ganges Delta of Bangladesh are attributed to the selective pressure from cholera [10]. Most cholera pandemic is known to have emanated from this region.

This review brought to the fore the relationship between blood groups and susceptibility or resistance to some selected diseases.

2. ABO BLOOD GROUP AND *Plasmodium falciparum* INFECTION

The ABO is the most important of about 36 blood groups in human blood transfusion studies [11]. There are two antigens associated with the blood group and individuals are classified phenotypically based on the presence or absence of these antigens. ABO is classified into four phenotypes; group A, group B, group AB and group O.

The gene that controls the expression of ABO group is located on the long arm of chromosome 9 and codes for glycosyltransferase, an enzyme that modifies the carbohydrate content of the RBC. The gene has three allelic forms: I, i^A and i^B. Antigen A is formed when glycosyltransferase transfers N-acetyl - galactosamine to H-substance, while the attachment of D-galactose to H-substance results in the formation of B antigen. H allele does not contain a functional enzyme hence it continue to express H-substance in O blood group [12]. ABO blood group is also associated with two antibodies; anti A and anti B all of which are IgM antibodies formed in the first year of life as a result of sensitization to environmental substances such as food bacteria and viruses.

Several studies have established a relationship between ABO blood type and incidences of *Plasmodium falciparum* infection. In a case control study of 567 Malian children, group individuals presented with less severe malaria than other groups [13]. In their findings Tewodros and colleagues [14] concluded that the chance of getting malaria infection is 2.5 times more in non-group O. The study indicated that individuals of group A, B and group AB are more susceptible to P falciparum infection compared to individuals of blood group O. These findings were further supported by that of Pathirana and colleagues [15] who after examining 243 cases of severe malaria agreed that group A, B and AB are more

susceptible to complicated malaria than group O. Panda et al. [16] singled out group B as being more susceptible to malaria than others. However Degarege et al. [17] found no significant association between ABO blood group and severe malaria infection. Perhaps their findings could have been influenced by the settings and the season in which the study was conducted.

Several mechanisms have been suggested to be responsible for increased susceptibility to malaria parasite infection by non-group O. This include: affinity of plasmodium species, common ABO antigen with *Plasmodium falciparum*, impairment of merozoite penetration of RBCs as well as cyto-adherence, endothelial activation and rosetting [18]. Having blood group O does not protect against initial infection by the parasite but only reduce disease severity [13]. Blood group A has a higher chance of rosette formation because it contains several glycosylated adhesion molecules such as intracellular adhesion molecule1, complement receptor1 [19], heparin sulphate like glycosamine glycan, platelet glycoprotein CD 36 [20], high level of von Willebrand factor [21], low arginine and nitrate levels, presence of cellular micro particles and nature of sugar molecules in blood group A [22]. Rosetting allows the blood to clump together, allowing the parasite to spread rapidly through the blood. The process of clumping makes use of ABO antigen which blood group O lacks, hence they have less ability to form rosette when infected with *plasmodium falciparum*. The reason for rosette formation is not very clear, it was initially believed to be a device for the parasite to infect other cells, but it is now understood to be a “cloaking device” that hides the infected cell from immune system [13].

3. ABO BLOOD GROUP AND GASTRIC CANCER

Gastric cancer is the fourth most common malignancy in the world and second leading cause of cancer death worldwide [23]. About one million people are newly diagnosed with the disease yearly with 700,000 deaths every year [24]. Gastric cancer is known to be caused by a combination of environmental and genetic factors. One of such environmental factors linked to gastric cancer is helicobacter pylori infection. However the fact that only small proportions of patients with helicobacter pylori infection develop gastric cancer indicates possible genetic factor involvement [25].

The pioneering work on the relationship between ABO blood group and gastric cancer was carried out by Aird et al. [26] who related 3,632 cancer patients with their blood groups. The study showed 20% increase in gastric cancer incidences among individuals of blood group A compared to group O. In 1961 a combined analysis of gastric cancer cases in fifteen study locations in USA Europe and Australia reported a significant positive association between blood group A and risk of gastric cancer [27]. However forty six years after the observation by Aird and colleagues, over one hundred and fifty studies in different locations agreed that gastric cancer relative incidence ratio among A and O is 1.2 [28]. In a cohort study of 18,244 Chinese blood donors in 1986 who were followed up for 26 years, 560 of them developed gastric cancer. The result showed that group A was associated with higher incidences of gastric cancer, while group A, AB and O were associated with reduced incidences of all cancers [29].

Also a study by Edgren and colleagues [30] concluded that blood group A individuals were more prone to gastric cancer than group O. Patients with blood group AB and B did not show significant difference in gastric cancer risk than group O.

In 2010 a large population study among Swedish and Danish blood donors who were followed up for 35 years a more prevalence of gastric cancer in group A than group O. Wang et al. [31] also collaborated these findings and added that gastric cancer cases is more in those group A patients who are also infected with Helicobacter pylori. Rizato et al. [32] concluded that infection with H pylori can be a factor in the progression to gastric cancer. The exact mechanism in which ABO blood group affect incidences of gastric cancer is not well known, but authors speculate the nature of antigen on the red cell as being responsible.

4. ABO BLOOD GROUP AND HELICOBACTER PYLORI INFECTION

Helicobacter pylori are microaerophilic bacteria that grow in the digestive tract and have the tendency to attack the stomach lining. It infects the stomach of 60% of the world adult population. In most cases the infection is asymptomatic while in others it can lead to ulcer and gastritis [33]. While investigating cases of 120 patients who passed through endoscopy. Mattos and colleague [34] discovered a higher

degree of infection among group O than other blood groups. These findings were collaborated nine years later by Jaff [35] who concluded that ABO, age and gender influences the rate of infection by the organism with group O being more susceptible than other ABO blood groups. The author also concluded that there was no significant difference in the rate of infection between Rh^+ and Rh^- persons and that women and adolescent are more prone to the infection.

In a cohort study Edgren et al. [30] supported the finding that blood group O is associated with peptic ulcer and linked this to Helicobacter pylori infection. This supports views of greater susceptibility of blood group O to infection with H pylori as well conclusion by Alkout et al. [36] who demonstrated that the H antigen represent an important receptor expressed in duodenal mucosal cells to which H pylori adheres. However these findings were at variance with that of Dickey et al. [37] and Aryana et al. [38] who did not find any relationship between ABO blood group, Lewis blood group and secretor to the degree of infection. Rather the differences in their findings were attributed to diversity in the sequence of blood group antigen binding adhesion gene (Baba gene) of H pylori [38].

5. ABO BLOOD GROUP AND INCIDENCES OF PANCREATIC CANCER

Pancreatic cancer arises from abnormal proliferation of cells of the pancreas. Adenocarcinoma is the most common type accounting for 85% of cases. In 2015, 411,600 people died from pancreatic cancer worldwide [39]. Pancreatic cancer is the fifth most common cause of cancer death in the United Kingdom (pancreatic cancer research fund 2015), and the common cause of cancer death in the United States [40]. Pancreatic cancer is one of the most aggressive cancers with mortality rate approaching incidence rate. A correlation has been established between ABO blood group and incidences of pancreatic cancer. After initial controversy, subsequent studies have been consistent in linking ABO blood group to pancreatic cancer.

Aird and colleagues [41] studied 620 patients and established more incidences of pancreatic cancer among group A individuals. Four years later this findings were disputed by Macafee [42]. Thirty years after Aird et al. work, a research carried out in Italy confirms a higher risk of pancreatic cancer among group B individuals [43]. Compared to group O those with group A, B and AB are more likely to develop pancreatic cancer. Seventeen percent of pancreatic cancer cases are attributable to inheriting a non O blood group [44]. A multinational pancreatic consortium supported earlier epidemiological evidence that people with blood group O may have low risk of pancreatic cancer than other groups [45]. There is increased risk with addition of each non O allele as exemplified by increased pancreatic cancer risk among individuals with A_1 group than those with A_2 blood group [46].

A meta-analysis of studies conducted in 89 different locations to assess the relationship between blood groups and cancer reveal increased risk of cancer including pancreatic cancer among group A than O [47]. In a case study of 363 patients and matched control subjects, it was reported that the increased risk of pancreatic cancer among individuals of non O group was even higher if they were sero-positive to Helicobacter pylori [48]. Almost all the studies reviewed failed to ascertain the exact mechanism in which ABO blood group influence the incidences of this disease but most authors call for more studies to be carried out.

6. SECRETOR STATUS AND NOROVIRUS INFECTION

Norovirus, so named after Norwalk Ohio where the outbreak first occurred in 1968, is the most common cause of gastroenteritis [49]. The virus is usually spread by faecal oral route. This may be by food, water or person to person contact. The virus has caused 200,000 deaths globally every year [49]. It most common occur in winter months. Norovirus contains a single RNA genome of 7.7 kb in length surrounded by a 530 long capsid protein which is folded into two major domains: A conserved S domain and more variable P domain. Studies show that the protruding norovirus capsid domain bear antigenic determinant affecting the immuno-logical response and host specificity.

Norovirus is highly contagious and genetically diverse. Not all individuals are susceptible to the same norovirus genotype [50]. The pivotal role of secretor status in determining susceptibility to norovirus was demonstrated by Thorven et al. [51] who compared susceptibility to gastro-enteritis in patients and medical staff involved in hospital outbreak in Sweden. The result showed that only non-secretors were protected from the

infection. Larson et al. [52] further demonstrated lower antibody titre to norovirus in non-secretors. Hutson et al. [53] demonstrated that group O individuals were more likely to be infected with norovirus whereas subjects of B group had decreased risk of infection and symptomatic disease. But Tan and Jiang [54] proposed that the association of ABO blood groups with susceptibility to norovirus infection may be strain specific rather than blood group dependent.

7. HIV INFECTION AND SECRETOR STATUS

Human immunodeficiency virus (HIV) originated from West and central Africa during the late 19th and early 20th century. It was first recognized by CDC in 1981. At the end of 2016, about 36.7 million people were living with HIV globally [55,56] and it resulted in 1 million deaths [55]. In 2016, there were 300,000new cases. Studies have related the degree of infection to ABO type and secretor status. Reduced risk of HIV infection was found in Senegalese commercial sex workers with non-secretors status [57]. Kinberg et al. [58] reported a slow progression of HIV1 in non-secretors. After examining 280 commercial sex workers in Kenya, Chanzu et al. [59] concluded that secretors of ABH substances are at higher risk of HIV infection than non-secretors and went ahead to observe that infection was higher with group A secretors than in other groups and opined that the carbohydrate moities on ABH blood group antigen possibly enhances viral penetration and binding leading to establishment of infection which may be important at the initial stage of viral uptake into cells of female genital tract.

8. ABO BLOOD GROUP AND CARDIOVASCULAR DISEASE

According to world health organization (WHO) cardiovascular disease (CVD) is and still remains the leading cause of death especially in developed countries. There are indications that cases are growing in developing countries. An estimated 17.3 million people died from CVD in 2008 representing 30% of all global deaths [60]. Non-O blood group had nearly threefold higher risk of developing cardiovascular event on follow up compared to O blood group [61]. Galle and Mott [62] found a significantly higher frequency of non- group O in 369 patients with deep vein thrombosis (DVT). To further clarify the interplay between the ABO, von Willebrand factor and factor VIII in the pathogenesis of cardiovascular disease. Koster et al. [63] performed a case control studies of 301 patients with diagnosed episode of venous thrombosis and 301 healthy control. Blood group O was less represented among venous thrombosis patients than in control.

In a longitudinal study of thromboembolism etiology (LITE), Ohira and colleagues [64], concluded that the risk of VTE was significantly higher in non O blood group than in group O. Similar results were obtained in a retrospective case control study in a number of Italian patients with venous thrombosis. Non-O blood group recorded 2.2 times higher cases than group O [65]. The influence of ABO blood group on von willebrand disease has also been reported. Gill et al. [66] concluded that of the 114 patients diagnosed with type 1 von Willebrand disease, 77% were of blood group O, group A had 18%, group B had 4% while group AB had none whereas the frequency of these groups in the general population is different. ABO blood group is known to influence cardiovascular disease and von Willebrand disease through its effect on von Willebrand factor and factor VIII levels. Von Willebrand factor is a glycoprotein that plays a role in blood coagulation. The protein is known to be quantitatively higher in non- group O individuals. The lower level of von Willebrand factor in group O is due to increased clearance of the factor by ADAMTS 13 enzyme [67]. The difference in the level of the factor also explain why individuals of non-O blood group are more prone to CVD while group O individuals have higher chances of developing von Willebrand disease.

9. DUFFY BLOOD GROUP AND *Plasmodium vivax*

The Duffy blood group was discovered in 1950. It was named after a haemophiliac patient who had multiple blood transfusions. A year later Fyb was discovered. The remaining antigen (F3) was discovered twenty years later. The absence of duffy antigen on RBC makes the cell more resistant to infection by *Plasmodium vivax*. To cause sickness the *P. vivax* must enter the human RBC, which it does by binding to the N-terminal extracellular domain of the Duffy glycoprotein through the cysteine rich region of the Duffy binding protein (DBP).

The Duffy antigen is also a receptor for chemokine; interleukin 8 (IL-8) and melanoma growth stimulating activity. IL-8 binds minimally to Duffy negative erythrocyte. Differences in racial distribution of Duffy antigen were discovered in 1954 when it was found that overwhelming majority of blacks are Duffy negative [68], sixty eight percent were African Americans and eighty eight to hundred were African blacks. The Duffy negative phenotype is exceedingly rare in Caucasians [69]. The presence of Duffy null allele could be a selective advantage in the population reducing the rate of infection by *Plasmodium vivax* [70].

Although we retrieved as many articles and literature regarding this issue as possible, there may be other diseases or conditions that may be closely associated with blood typing. We here described some typical or well known conditions, whereby highlighted this issue.

10. CONCLUSION

The rev*iew* brought to the fore the relationship between blood groups and susceptibility or resistance to disease. While mechanisms for the influences are well understood and acceptable for some disease conditions, for some others it is still controversial and not convincing. There is need for more studies to be carried out to unravel the exact mechanism how blood groups affect disease. It is recommended that individuals of various blood groups should know the different disease that they are prone to and avoid the predisposing factors to such diseases.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Chotivanich K, Udomusangpetch R, Pattanapanyasat K. HBE: A balance polymorphism protection against high Parasitaemia and severe *P. falciparum* malaria. Blood. 2002;100:1172-1176.
2. Haseeb MA, Thors C, Linder E, Eveland LK. Schistosomal mansoni: A chemoreceptor through N-acetyl-D-Galactosamine containing receptor in females, insight into increased severity in schistosomiasis in individuals with blood group A. Experimental Parasitology. 2008; 119:67-73.
3. Okoroiwu HU, Okafor IM. Demographic characteristics of blood and blood components transfusion recipients and pattern of blood utilization in a tertiary health institution in southern Nigeria. BMC Hematology. 2018;18:16.
4. Blackwell CC, Dundas S, James VS. Blood group and susceptibility to disease caused by Escherichia coli 0_{157}. Journal of Infectious Disease. 2002;185: 393-396.
5. Sheinfield J, Schaeffer M, Cordon-Cardo C. Association of the Lewis blood group phenotype with recurrent U.T.I in woman. New England Journal of Medicine. 1989; 320:773-777.
6. Zakai NA, Judd SE, Alexander K, McClare LA, Kissela BM, Howard G, Cushman M. ABO blood group and stroke risk: the reason for geographical and racial differences in stroke study. Journal of Thrombosis and Haemostasis. 2014;12: 564-570.
7. Alexander KS, Zakai NA, Gibert S, McClure LA, Wadley V, Unverzagt F, Cushman M. ABO blood type, factor VIII and incidences of cognitive impairment in the REGARDS cohort. Neurology. 2014; 83:1271-1276.
8. Garatty G. Blood group and disease: A historical perspective. Tranfus. Med. Rev. 2000;14:291-301.
9. Mourant AE, Kopec AC, Damaniewska-Sobczak Y. Blood group and disease. Oxford University Press. 1978;105-107.
10. Glass RI, Holmgreen J, Haley CE. Predisposition to cholera of individuals with O blood group. Possible evolutionary significance. American Journal of Epidemiology. 1985;121:791-796.
11. Story JR, Castiho L, Chen Q, Daniels G, Denomme G, Flegel WA, Glasner G, de Hass M. (). International society of blood transfusion working party on red cell immunogenetics and terminology: report of the Seoul and London meeting. ISBT Science Series. 2016;11:118-122.
12. Morgan WTJ, Watkin WM. Genetic and biochemical aspect of human blood group

A, B and H, Le^{-a} and Le^{-b} specificity. British Medical Bulletin. 1969;25:30-34.

13. Rowe JA, Handel IG, Thera MA, Deans AM, Lyke KE, Kone A, Diallo DA, Mould JM. Blood group protects against severe *Plasmodium falciparum* malaria through the mechanism of reduced rosetting. National Academy of Sciences. 2007;104: 17471-17476.
14. Tewodros Z, Abraham D, Berhana E. Association of ABO blood group and *Plasmodium falciparum* malaria in Dore Bafeno area, southern Ethiopia. Asian Pacific Journal of Tropical Medicine. 2011; 11:60045-60047.
15. Parthirana SL, Alles HK, Banadara S, Phone-Kyaw M, Perera MK. ABO blood groups with severe *Plasmodium falciparum* malaria. Annals of Tropical Medicine and Parasitology. 2005;99:119-124.
16. Panda AK, Panda SK, Sahu AN, Triparthy R, Ravindran B, Das BK. Association of ABO blood group with severe falciparum in adult: A case control study and meta analysis. Malaria Journal. 2011;10: 309.
17. Degarege A, Medhin G, Animut A, Legess M, Erko B. Association of ABO blood group and *P. falciparum* malaria related out-comes: A cross-sectional study in Ethiopia. Acta Trop. 2012;123:164-169.
18. Loscertales MP, Owens SO, Donnell J, Bunn J, Bosch-capblanch X, Brabin BJ. ABO blood group phenotype and *plasmodium falciparum* malaria: Unlocking a pivotal mechanism. Advance parasitology. 2007;65:1-50.
19. Lublin DM, Grifith RC, Atkinson JP. Influence of glycosylation on allelic and cell specific Mr variation, receptor processing and ligand binding of the complement C_{3b}/C_{4b} receptor. J. Biol. Chem. 1986;261: 5736-5744.
20. Rout R, Dhangadamajhi G, Mohapatra BN, Kar SK, Ranji M. Genetic diversity ofPfEMP1- DBL1 and its association with severe malaria in hyper endemic state of India. Asian Pacific Journal of Tropical Medicine. 2010;3:505-509.
21. Jenkin PV, O'Donnell JS. ABO blood group determines plasma von Willebrand factor level: A biologic functions after all? Transfusion. 2006;46:1836-1844.
22. Daniels G. The molecular genetic of blood group polymorphism. Transplantation Immunology. 2005;14:143-153.
23. Jemal A, Bray F, Center MM. Global cancer statistic. Cancer Journal. 2011;61: 69-90.
24. Parkin DM, Bray F, Ferlay J, Pisani P. Global cancer statistics. CA Cancer Journal Clin. 2002;55:74-108.
25. Gouzalez CA, Sala N, Capella G. Genetic susceptibility and gastric cancer risk. International Journal of Cancer. 2002;100: 249-260.
26. Aird I, Bentall HH, Robert JAF. The relationship between cancer of the stomach and ABO blood groups. British Medical Journal. 1953;1:799-801.
27. Clarke CA. Blood group and disease. In: Steiberg AG. Editor Progress in Medical Genetic. 1961;1:81-119.
28. Garatty G. Do blood group have biological role? In: Garatty Immunobiology of transfusion medicine. Marcel Dekker Inc. New York. 1994;205-255.
29. Huang JY, Wang R, Gao YT, YUAN JM. ABO blood type and the risk of cancer. Findings from the Shangai cohort study. Plos One. 2017;12:e0184295.
30. Edgren G, Hjalgrim H, Rostgaard K. Risk of gastric cancer and peptic ulcer in relation to ABO blood type: A cohort study. American Journal of Epidemiology. 2010; 172:1280-1285.
31. Wang Z, Liu L, Ji J. ABO blood group system and gastric cancer: A case control study and meta-analysis. International Journal of Molecular Science. 2012;13: 13308-133021.
32. Rizato C, Kato I, Plummer M. Risk of advance cancer precancerous lesions in *Helicobacter pylori* infected subjects is influenced by ABO blood group and Cag A status. International Journal of Cancer. 2013;133:315-322.
33. Colledge H, Cfasso J. *Helicobacter pylori* infection; 2017.
Available:www.health.com
[Accessed June 14, 2019]
34. Mattos LC, Rodrigues CJ, Sanches FE, Ruiz MA, Mureira HW. ABO, Lewis, secretor and non-secretor phenotypes in patients infected or uninfected by the *Helicobacter pylori* bacillus. Sao Paulo Medical Journal. 2002;120:55-58.
35. Jaff MS. Relationship between ABO blood group and *Helicobacter pylori* infection in symptomatic patients. Clinical Experimental Gastroenterology. 2011;4:221-226.
36. Alkout AM, Blackwell CC, Weir DM. Isolation of cell surface component of

Helicobacter pylori that bind sH type 2, Lewis A, Lewis B antigen. Gastro Enterology. 1997;112:1179-1187.
37. Dickey W, Collins JSA, Watson RGP, Sloan JM. Secretor status and *Helicobacter pylori* infection are independent risk factor for gastroduodenal disease. Gut. 1993;3:351-353.
38. Aryana K, Keramati MR, Zakavi SR, Sadeghian MH, Akbari H. Association of H pylori infection with Lewis and ABO blood groups in dyspeptic patients. Nigerian Medical Journal. 2013;54:196-199.
39. Global Burden on Disease; 2015.
40. Hariharan D, Saied A, Kocher HM. Analysis of mortality rates for pancreatic cancer across the world. Hpb. 2008;58:62.
41. Aird I, LEE DR, Roberts IA. ABO blood group and cancer of esophagus, cancer of pancreas and pituitary adenoma. British Medical Journal. 1960;1:1163-1166.
42. Macafee AL. ABO blood group and carcinoma of the pancreas. Ulster Medical Journal. 1964;33:129-131.
43. Vioque J, Walker AM. Pancreatic cancer and ABO type: A study of cases and controls. Medical and Clinical. 1991;96: 761-961.
44. Wolpin BM, Chan AT, Hart GE. ABO blood group and the risk of pancreatic cancer. Journal of National Cancer Institute. 2009; 101:1015-1023.
45. Ammundadotir L, Kraft P, Solomon RZ. Genome wide association study identifies variants in the ABO locus associated with susceptibility to pancreatic cancer. Nature and Genetics. 2009;41:986-990.
46. Wolpin BM, Kraft P, Gross M. Pancreatic cancer risk and ABO blood group alleles. Result from Pancreatic Cancer Consortium. 2010;70:1015-1023.
47. Zhang BL, He N, Huang F, Chen KX. ABO blood group and risk of cancer: Systematic review and meta-analysis. Asian Journal of Cancer Prev. 2014;15:4643-4650.
48. Risch HA, Yu H, Lu L, Kidd MS. ABO blood group, *Helicobacter pylori* sero positivity, the risk of pancreatic cancer: a case control study. Journal of National Cancer Institute. 2010;102:502-505.
49. Centre for Disease Control; 2017.
50. Nordgren J, Kindberg E, Lindgren PE, Maussek A, Svensson L. Norovirus gastroenteritis outbreak with secretor independent susceptibility pattern. Emerging Infectious Disease. 2016;16:81-87.
51. Thorven M, Grahn A, Johansson H, Larson G, Svensson L. Homozygous nonsense mutation in human secretor (FUT2) Gene provides resistance to symptomatic to norovirus infection. Journal of Virology. 2005;79(24):15351-15355.
52. Larson MM, Rydell GE, Grahn A. Antibody prevalence and titre to norovirus correlate to secretor (FUT2), but not with ABO phenotype or Lewis (FUT3) genotype. Journal of Infectious Disease. 2006;194: 1422-1427.
53. Hutson AM, Atmar R, Graham DY. Norwalk virus infection disease associated with ABO blood group. Journal of Infectious Disease. 2002;194:1422-1427.
54. Tan M, Jiang X. The association of histo blood group antigen with susceptibility to norovirus infection may be strain specific rather than group dependent. Journal of Infectious Disease. 2008;198:940-941.
55. World Health Organization (WHO). Global Health Observatory (GHO) data: HIV/AIDS.
Available:www.who.gho/hiv/en
[Accessed June 14, 2019]
56. Okoroiwu HU, Okafor IM, Enosakhare AE, Okpokam DC. Seroprevalence of transfusion-transmissible infections (HBV, HCV, syphilis and HIV) among prospective blood donors in a tertiary health care facility in Calabar, Nigeria: An eleven years evaluation. BMC Public Health. 2018;18: 645.
57. Ali S, Niang MA, Ndoye I. Secretor polymorphism and human immunodeficiency virus infection in Senegalese woman. Journal of Infectious Disease. 2000;18:737-739.
58. Kinberg E, Hedeman B, Brat G. A nonsense mutation in the fucosyl transferase FUT2 gene affects the progression of HIV 1 infection. AIDS. 2000;20:685-689.
59. Chanzu NM, Mwanda W, Oyugi J, Anzak O. Mucosal blood group antigen expression profile and HIV infections. A study among female sex workers in Kenya. Plos One Journal. 2015;10:e0133049.
60. WHO. World Health Organization; 2008.
61. Capuzzo E, Bofanti C, Frantini F, Montors P, Turdo R. The relationship between ABO blood group and cardiovascular disease: Result from cardio risk programme. Annals of Transfusion Medicine. 2016;4:89.
62. Wautrech JC, Galle C, Motte S, Serene JP. The role of ABO blood group I the

incidences of deep vein thrombosis. Thrombosis and Haemostasis. 1998;79: 688-695.
63. Koster T, Blann AD, Briet E. Role of clotting factor VIII and effect of von Willebrand factor on the occurrence of deep vein thrombosis. Lancet. 1995L345: 152-155.
64. Ohira T, Cushman M, Tsai MY. ABO blood group, other risk factors and incidences of venous thrombosis. The longitudinal investigation of thromboembolism etiology. Thrombosis and Haemostasis. 2007;5: 1455-1461.
65. Spiezia L, Campello E, Bon M. ABO blood group and the risk of venous thrombosis in patients with inherited thrombophilia. Blood Transfusion. 2013;11:250-253.
66. Gill JC, Endros-Brooks J, Bauer PJ, Marks WJ, Montgomery RR. The effect of ABO blood group on the diagnosis of von willebrand disease. Blood. 1987;69:1691-1695.
67. Bowen DJ. Influence of ABO blood group on the rate of proteolysis of von Willebrand factor by ADAMTS 13 enzyme. Blood. 2005;106:1988-1991.
68. Horuk R, Chinis CE, Darbonne WC, Dolby TJ. A receptor for malaria parasite *Plasmodium vivax*: The erythrocyte chemokine receptor. Science. 1993;261: 1162-1164.
69. Lewinson W. Medical microbiology and immunology. Examination board review. New York: Lange Medical Books/McGraw Hill. 2004;143:199-203.
70. Cavasini CE, Mattos LC, Couto AA. *Plasmodium vivax* infection among Duffy antigen negative individual from the Brazilian Amazon region: An exception? Society for Tropical Medicine. 2007;101: 1042-1044.

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