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The prevalence of secretor status and co-expression of lewis antigen in voluntary blood donors

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ABSTRACT

Background: Blood group substances are present in soluble form in a majority of individuals in secretion such as saliva and body fluids. Secretor status refers to the presence (SeSe and Sese) or absence (sese) of secretor gene which secrete ABH soluble substances. Secretor status can be used to resolve ABO discrepancies of people whose blood group cannot be identified by routine blood grouping and it can also help in identifying patients who may be a high risk group for getting certain diseases. **Aims and Objectives:** Our aim and objectives of the study is to find out the Prevalence of Secretor Status and Co-expression of Lewis Antigens among the Voluntary Blood Donors. **Materials and Methods:** This study was conducted in sixty volunteers and the method used to determine the secretor status was hemagglutination inhibition method. Their blood was used to detect the type of Lewis (Le) antigen since the type of Lewis antigen correlated with the secretor status of the individual. **Results:** Among the sixty subjects tested, forty five of them were found to be secretors and fifteen of them were Non-secretors. The number of Lewis (a+b-) individuals were twelve, Lewis (a-b+) were thirty nine and Lewis (a-b-) were nine. **Conclusion:** The prevalence of secretors was 75% and non-secretors were 25% respectively. We found 65 % of the volunteers were found to be Le (a-b+) positive, 20% were Le (a+b-) and the remaining 15% were Le (a-b-) which correlated with the ABH antigen secretor status.

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INTRODUCTION

Blood group substances are present in soluble form in a majority of individuals in secretion such as saliva and body fluids. Secretor status refers to the presence (SeSe and Sese) or absence (sese) of secretor gene which secrete ABH antigen soluble substances.[1] The biosynthesis of H antigen found on red blood cells and that found in secretions involves two different α, 1, 2 – fucosyl transferase (FUT) enzymes encoded by two closely linked genes on chromosome 19, FUT-1 and FUT-2. These genes are also referred to as the H gene and Se (secretor) gene. Both encode enzymes that add on H specific fucose sugar to a precursor oligosaccharide core structure but they act on different precursor substances. The terminal carbohydrate sequences of the ABH antigens in saliva and plasma are identical to those on red cells. But the backbone or the framework carbohydrate structures are different.

H antigen is synthesized in RBCs when the H (FUT 1) gene encoded fucosyl transferase attaches a fucose via an α 1, 2 linkage to the terminal galactose of type 2 precursor chains. H antigen in secretions is synthesized when the Se gene encoded fucosyl transferase attaches a fucose via an α 1, 2 linkage to the terminal galactose of type 1 precursor chains in secretory tissues.[2]

The ability to secrete ABH antigens is genetically inherited, approximately 80% are secretors and 20% are non-secretors.[3] This percentage of secretor and non secretor

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varies according to the race and ethnicity. This trait is inherited as a single locus gene in simple mendelian fashion. The secretor gene is dominant and non secretor is recessive. People having the secretor gene are called Secretors and those who do not have are called Non-secretors.[3] In secretor individuals of the appropriate ABO group, ABH antigens are detected in the secretions of the goblet cells and mucous glands of the digestive (saliva, gastric juice, bile, meconium), genitourinary (spermatic fluid, vaginal secretion, ovarian cyst fluid, urine) and respiratory tracts as well as in other secretions (milk, sweat, tears and amniotic fluid).[4]

The ABO, Lewis (Le) and Secretor/Non secretor system are considered an entity in which all antigens are chemically related but in which independent system of genes determine the phenotype.[5] The Lewis gene (FUT 3) resides on Chromosome 19 and is distantly linked to H and Se loci. Lewis antigens, unlike antigens of all other blood group system are not intrinsic to the RBC but are synthesized in intestinal epithelial cells. Lewis antigens circulate in plasma while bound to glycosphingolipids-and are passively adsorbed onto RBCs. The presence of the particular type of Lewis antigen approximately indicates the secretor status of the individual. Individuals who type as Le (a-b+) are secretors, Le (a+b-) are nonsecretors and Le (a-b-) individuals could be either secretors or nonsecretors. Lewis system is associated with disease such as peptic ulcers and bladder cancer. Le^b is associated with Helicobacter Pylori infection. The incidence of Lewis blood group phenotype in Asian population is 72% for Le^b, 22% for Le^a, 6% for Le (a-b-) and 3 % for Le (a+b+) individuals.[6] S Akhter et al in his study of blood donors in Dhaka, Bangaldesh reported 19 % were Le (a+b-), 53 % were Le(a-b+), 26% were Le (a-b-) and 2 % were Le (a+b+).[6]

Sara et al in her results demonstrated that the secretor status plays an intrinsic role in resistance to H.pylori infection and suggested that the fucosylated secretor ABH antigens constitute interactive members of human and primate mucosal innate immune system.Non secretion of blood group antigen influences the pathogenic sequel of urinary tract infection.[7]

Camps et al in his study of 1000 alcoholic patients reported that the percentage of non secretors were 32.7%. The frequencies of non secretors varied in different part of British Isles where the study was undertaken. He noted there were a general pattern of increase in group-A non secretors and Le^a individuals among the alcoholic patients.[8]

Dayaprasad et al in his study of non secretor status – a predisposing factor for vaginal candidiasis had reported that the prevalence of vaginal candidiasis was significantly higher in non secretor group. In his control group, the percentage of secretors and non secretors were 77% and 23% respectively.[9]

Genotyping is regarded as the gold standard method in secretor and Lewis blood grouping because it is able to identify minor errors which can occur in agglutination-based phenotyping.[10] Our aim and objective of our study was to find out the Prevalence of Secretor Status and Co-expression of Lewis Antigens among the Voluntary Blood Donors

AIM AND OBJECTIVES

To find out the Prevalence of Secretor Status and Co-expression of Lewis Antigens among the Voluntary Blood Donors

MATERIALS AND METHODS

This study was conducted in the Department of transfusion Medicine, The Tamilnadu Dr.M.G.R Medical University. A total number of 60 voluntary blood donors donated their blood and participated in this study. Ethical clearance was obtained and informed written consent was obtained from the volunteers who donated blood in the study. The blood groups of the subjects were determined using conventional tube technique. Their blood was tested for the Lewis antigen by using Lewis antisera (Dia Clon Anti- Le^a and Anti- Le^b from Bio rad).

The method used to determine the secretor status was hemagglutination inhibition method. The secretors have water soluble blood group substances which are readily detected in very minute quantities because they have the property of reacting with their corresponding antibodies and thereby neutralizing or inhibiting the capacity of antibody to agglutinate erythrocytes possessing antigen. The reaction provides a means of finding the relative activity or potency of these water soluble blood group substances.

Five ml of saliva was collected from all voluntary blood donors and it was placed in a boiling water bath to inactivate the salivary enzymes. Antisera are added to the saliva and incubated for 10 minutes at room temperature. To that appropriate pooled cells are added and incubated for 45 minutes at 37°C. Agglutination of RBC indicates that the person is a non-secretor and its absence indicates the person is a secretor[11] (Table 1).

Lewis antigen was determined from the blood of all volunteers using the Lewis antisera Le (a+b-) and Le (a-b+).

Table 1: Determination of secretor status by hemagglutination inhibition method

Diluted antisera+ Supernatant saliva	Incubate for 8 to 10 min.	Addition of appropriate indicator red cells	Incubate for 30 to 60 minutes	Centrifuge	Agglutination	Nonsecretor
					No Agglutination	Secretor

Table 2: Co-expression of lewis antigen in secretors and non secretors

	Lewis (a+b-)	Lewis (a-b+)	Lewis (a-b-)	Total
Secretors	-	39	6	45
Nonsecretors	12	-	3	15
Total	12	39	9	60

Table 3: ABO blood group & secretor status of the participants

Blood Group	Secretor	Non secretor	Total
A	9	4	13
B	16	5	21
O	17	6	23
AB	3	0	3
Total	45	15	60

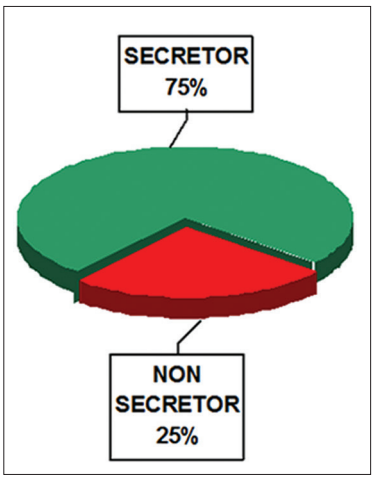

Figure 1: Secretor status

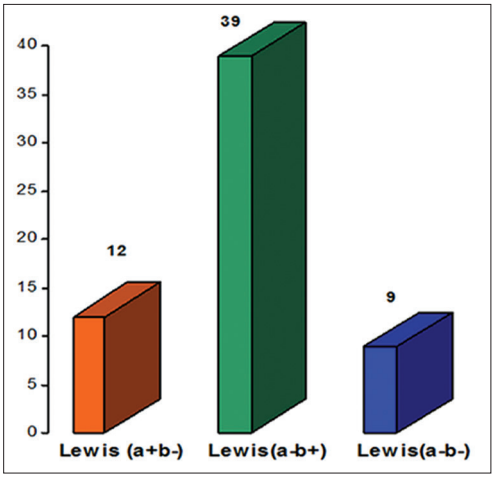

Figure 2: Frequencies of lewis antigens

RESULTS

Among the 60 subjects tested, 45 of them were found to be secretors and 15 of them were Non-secretors. The number of Lewis (a+b-) individuals were 12, Lewis (a-b+) were 39 and Lewis (a-b-) were 9.

DISCUSSION

In our study the prevalence of secretors was 75% and non-secretors were 25% respectively (Figure 1). The percentage of secretors and non-secretors varies with the racial and ethnic population. In Caucasians about 20% of people are non-secretors while our study showed 25 % of our volunteers are non-secretors.[12]

Agarwal in his study of ABH secretor status in North India found out that 64% were secretors, 21% were non secretors and 14% were aberrant secretors. Individuals who have substance H in their saliva or those who have substance H but not A and B substances are called as aberrant secretors.[13]

Luiz et al control group were 76.1% secretors and 23.9% nonsecretors in his study of patients infected or uninfected by the H.pylori bacillus.[14]

The blood sample of the 60 persons was also tested for the presence of Lewis antigen. In our study, 39 were found to be Le (a-b+) positive, 12 were Le (a+b-) positive and the remaining 9 were Lewis (a-b-) (Figure 2). The 39 Le (a-b+) were secretors and the 12 Le (a+b-) were nonsecretors. Six of them were secretors and three of them were nonsecretors in the remaining nine Le (a-b-) (Table 2). S Akhter et al in his study of forty two individuals reported 19 % were Le (a+b-), 53 % were Le (a-b+), 26 % were Le (a-b-) and 2 % were Le (a+b+).[15] Our study revealed A group volunteers had more percentage of non-secretors (31%) when compared to B (24%) and O groups (26%) (Table 3).

Tereza et al reported higher percentage of Lewis (a-b-) in black population and suggested that the reason could be due to nongenuine Lewis phenotype (Lewis antigen absent on red cells but present on salivary secretions). She also attributed this high frequency of Lewis (a-b-) phenotype due to decreased concentration of circulating Lewis antigen

occurring during diseases caused by parasites, infection or other pathologic conditions.[16] The limitations of the study was the relatively low sample size which cannot be used as an assessment of prevalence of secretor status in the local population.

CONCLUSION

The prevalence of secretors was 75% and non-secretors were 25% respectively. In our study, 65 % of the volunteers were found to be Le (a-b+) positive, 20% were Le (a+b-) and the remaining 15% were Le (a-b-) which correlated with the prevalence of ABH antigen secretor status. Secretor status of the individual can be used to resolve ABO discrepancies which may occur in certain conditions like Leukemia, Hodgkin disease and Acquired B. It also helps in finding out rare subgroups of A and B, where normal haemaggulitination method fails. The haemaggulitination inhibition method of finding out the secretor status is cost effective and can be done in any laboratory centre. Co-expression of Lewis antigens on red blood cells plays a contributory role in assessing ABH secretor status of the individual. However, for the doubtful cases of ABH Secretors or Lewis antigens expression, the only unambiguous option available is the secretor gene analysis.

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Authors Contribution:
RRB - Concept and design of the study, collected data,reviewed the literature, manuscript preparation and critical revision of the manuscript; **PA -** Manuscript preparation and critical revision of the manuscript.

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