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A qualitative study of Group 1 blood serum (Jansky)

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A QUALITATIVE STUDY

of

GROUP 1 BLOOD SERUM(JANSKY)

by

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Introduction

The success of blood transfusions in cases requiring such procedure has been demonstrated beyond doubt to the medical profession. It is true that post-transfusion reactions sometimes occur, but it is thought that these disturbances are due to improper classification or faulty technique. More knowledge of blood groups will lessen these possibilities and permit, perhaps, a greater use of transfusions than has heretofore been possible.

Since the discovery of different blood groups in man by Landsteiner much experimental work has been done. Landsteiner thought, at first, he was dealing with pathogenic blood, an opinion which he speedily changed upon further research. And so, another scientific discovery was made useful to man. Probably no other discovery since 1900 has been more important or far-reaching than this observation, for transfusions were not blind attempts anymore and they became another method of saving lives in which sickness or accidents had caused a loss of blood. Surgery advanced now that it was known that skin grafts must be made from individuals of the same group.

The work did not stop here, however. Epstein and Ottenberg (5), then von Dungern and Hirschfeld (37)

adduced evidence showing that these blood groups were inherited according to the laws of Mendelian inheritance. Since these groups followed definite laws of inheritance they could be used in the field of forensic medicine to show parentage or illegitimacy. Unfortunately, these tests could not be used in all cases because of the small number of combinations possible from only four groups.

Further research has demonstrated that all groups do not fit in with the classification as they should, and these have been shown to be sub-groups. Most of this work has been done on the cells of the different groups, very little attention being paid to the serum of the respective types.

The serum of all normal Type 1 (Jansky) individuals contains agglutinins Alpha and Beta. The object of this problem was to demonstrate either positively or negatively the absence of Alpha or Beta agglutinin or both in Type 1 serum. The problem was therefore entitled: A Qualitative Study of Group 1 Serum (Jansky). There is another classification of blood groups by Moss which is the direct opposite of Jansky's, and to avoid confusion the so-called International system of classification will be used, in which Group 1 becomes Group O, Group 2 becomes Group A, Group 3 becomes Group B and Group 4 becomes Group AB.

As some of the blood samples were secured from people with Tuberculosis, separate datum was kept on the blood of these groupings so that the relationship of Tuberculosis to Blood Groups could be noted, if any. The purpose was not to show that certain blood groups and disease were linked, but that one or another of the blood group factors might tend to affect indirectly some morphologic, physiologic or pathologic condition, thus causing an abnormal distribution of the groups in the particular condition affected.

Historical Review

In the year 1896 Gruber and Durham (7) reported on the specificity and reactions of agglutination. Work had been done before this date but theirs was the more thorough and exhausting and to these men goes the honor of discovery. It was noted by some observers that when bacteria were mixed with immune serum they lost their motility, if they were motile, and then clumped together and precipitated to the bottom of the tube. Gruber and Durham extended these observations and it was found later that the cells of the body would go through the same reaction.

Ehrlich (41), the famous German scientist and Immunologist, attempted to bring agglutination into line with his "receptor" theory. He interpreted the process

of agglutination as a chemical union of agglutinin and bacteria or cell (agglutinogen). The agglutinin he regarded as consisting of two atom complexes, one of the"haptophore", having affinity for the bacterial or cell protein, and concerned with the union, the other the"ergophore" or "zymophore", by means of which the actual agglutination could be brought about when the union had taken place.

Bordet (1), working with the same phenomenon, disagreed with Ehrlich and formulated the "two-phase" theory. He noticed that agglutination failed to take place in the absence of electrolytes, and reasoned that there was an adsorption of agglutinin by the agglutinin-ogen (cell), which in some way altered the charge of the mass, and which, when electrolytes were added, neutral-ized the charge, with consequent agglutination.

The theory, with some modifications, is the accepted reaction of today. Eagle (4) has shown that in the case of cellular antigen the antibody is present as an invisible film of specifically adsorbed protein, while in the precipitation reaction it may constitute the bulk of the material formed. In either case, what was once a hydrophilic globulin has become water-insoluble or denatured, upon combination with the antigen. Colloidal chemistry is familiar with these reactions and this property of adsorbed proteins affecting an

otherwise stable colloidal suspension is not peculiar to immunity. In immune reactions this denaturation of the proteins, that is, of the antibody globulin, is due to the fact that its specificity is determined by hydrophilic groups. When these combine with the antigen, hydrophobic groups necessarily face the water phase, thus determining the surface properties of the antigen-antibody complex. But when normal serum is adsorbed, since there are no groups with a specific affinity to antigen, the molecules naturally orient themselves at the interface so that the hydrophilic groups face the water, and the adsorbed protein acts as a protective film away from its isoelectric point. In the words of the author, three things determine agglutination;

> " The hydrophilic antigen is covered, with (1) a film of immune globulin, denatured by its combination with antigen. In the absence of electrolytes the charge due to the ionization of the protein suffices to prevent aggregation. Minute concentrations of (2) electrolytes, however, depress this surface charge below the critical value necessary for stability. The resulting aggregation is therefore primarily of the immune globulin surfaces, and only incidentally of the associated antigen."

Schroder (33) has shown that isoagglutination is always accompanied by a decrease in the electrical charge of the erythrocytes and a decrease in amount of globulin, especially euglobulin.

The discovery of agglutination in human blood groups has been attributed to various men, Furuhata

claiming that Japanese and Chinese books of the 17th century had dealt with this particular phase. Levine doubted this and requested Furuhata's reference. Furuhata gave, as his reference, a book edited by H. Breitenstein, entitled "Die Gerichtliche Medizin der Chinese," by Wang-in-Hoai, Leipsig, 1908. Wiener (39) has given the reference and translation and, though somewhat long, it is unique in so-called immunological studies and follows:

> "If the skeleton of a father or mother is found in a different locality, and the son or daughter desires to recognize it, the public officer must have the son or daughter prick himself or herself with a needle so that a drop of blood is allowed to fall on the bones. If it is the parent's skeleton, the blood will penetrate the bones."
> Note:
> "There is still another blood test: Two persons stick themselves and let a drop of blood fall into some water. If these individuals are actually father and child.... the bloods will flow together. If the bones were washed with salt water, the blood would not penetrate the bones, even if a true relationship between father and son.... should exist. This is a trick that wicked men use and one should therefore be careful to watch out for it.
> If true children and true brothers, who, perhaps have lived apart from childhood wish to recognize each other, it is very difficult to differentiate between true and false relationship; the order should therefore be given that they stick themselves and let a drop of blood fall into an earthern vessel; if they are true brothers or sisters the drops of blood will form a cake.
> A grandchild can recognize its grandfather by a blood test; since the husband and wife have not the same parents, how then shall the test be made upon husband and wife? If one say that in that case the blood penetrates the bones, how about the case where a woman adopts, suckles and raises a new-born child? This child absorbs the blood of the mother from birth; should not the blood of such child more easily

penetrate the bones of its foster-mother? Nevertheless, this does not happen (also not with man and wife).
In the blood test with water, one must take into account the following: If the vessel is large and contains much water, the drops of blood are far-apart and cannot come together. Or: if in the blood test the drops do not fall into the water simultaneously, a difference in temperature will exist, so that the blood likewise cannot come together."

The introduction of isoagglutination in human blood has been recognized for a little more than thirty years. The earliest mention of this subject was in a communication made by Samuel Shattock (34) to the London Pathological Society in 1899. His paper shows that he was studying rouleaux formation (pseudoagglutination) and not isohemagglutination.

The credit for this discovery is usually given to Landsteiner (18) but his first reference to isoagglutination in human blood occured on March 3, 1900, almost a year later. This contribution occupied eight lines and appeared as a footnote on page 361 of his article on a totally different theme.

"Das serum gesunder Menschen wirkt nicht nur auf tierische Blutkorperchen agglutinierend, sondern ofters auch auf menschliche, von andern Individuen stammende. Es bleibt zu entscheiden, obdiese Erscheinung durch upsprungliche individuelle Verschiedenheiten oder durch die erfolgte Einwirkung von Schadigungen etwa bakterieller Natur bedingt ist. Thatsachlich fand ich das erwahnte Verhalten bei Blut, das von Schwerkranken herruhrte, besonders ausgepragt. Es konnte diese Erscheinung mit dem von Maragliano geschilderten Losungvermogen des serums fur Blutkorperchen bei verschiedenen Krankheiten zusammenhangen." (1x.

Kongr. f. inn. Med.,1892.)

Following these publications a number of authors wrote upon the subject but their work was mostly concerned with the diagnostic value in certain diseases.

Landsteiner's (19) real work appeared in 1901 when he showed that isoagglutination was independent of health and that it was not a random reaction but followed certain definite laws. He showed that the blood of 22 persons investigated by him fell into, in most cases, three distinct groups which he called A, B and C. The serum of members of Group A agglutinated the red cells of Group B and no others. The serum of members of Group B agglutinated the cells of Group A and no others. The serum of Group C agglutinated the cells of both A and B. To explain these reactions it was necessary to assume the presence of at least two agglutinins, one present in the serum A, the other present in serum B, and both present in serum C.

In the following year Decastello and Sturli (3) further advanced our knowledge of this subject. In a series of 155 persons whose blood they examined, four were encountered which formed an exception to Landsteiner's rule of three isoagglutinable groups. The serum from none of these four persons was capable of agglutinating the red cells of a member of Groups A, B or C, but their red cells were agglutinated by the serum

of each of the three groups, and thus a fourth group was plainly established. They also proved that isoagglutination was not a manifestation of disease but normal and showed that there were two agglutinins, one in serum A, the other in serum B, and both together in serum C.

Five years later (1907) Jansky (17) reported the existence of four isoagglutinable groups, three of them with characteristics corresponding to those by Landsteiner and the fourth in all respects like that formed by the four exceptions of Decastello, but he made no reference to them.

In the same year, Hektoen (11) classified blood into groups ignoring the fourth because of its rarity.

In 1909 Moss (24), also overlooking the real nature of Decastello's findings, and unaware of the work of Jansky, worked out the existence of the groups. While his paper was in the hands of the printer he came across Jansky's work and added a footnote to that effect.

During the eleven years that followed Shattock's work a considerable amount of information accumulated concerning isoagglutination. In addition to the separation into four blood groups it was definitely proven that this phenomenon was independent of disease. From examination of the blood of infants it was shown that the grouping was often not established at birth; that

the red cells, as a rule, acquired agglutinogen before agglutinin appeared in the serum; and that both agglutinogen and agglutinin appeared before the child was six months old. Moss (40) then showed that Types A and B sera could be used for grouping.

The four groups were thus established and described by Landsteiner and his pupils in 1902. Jansky introduced a numbering of the groups, and Moss, not knowing of Jansky's work, did the same but reversed Jansky's Groups O and AB. Von Dungern and Hirschfeld, therefore, to avoid confusion suggested the naming of the groups by the letters O,A,B and AB according to the antigenic substances of the respective cells. This method of nomenclature will be used throughout the paper. The agglutinins which correspond to the antigens are called by the Greek letters Alpha and Beta. The table shows the relationship of the three classifications.

Nomenclature	Groups			
Jansky	1	2	3	4
Moss	4	2	3	1
International	O	A	B	AB
Cells (agglutinogen)	O	A	B	AB
Serum (agglutinins)	ab	b	a	O

During the numerous transfusions which followed this work, cases were encountered which did not react as one of the four groups would be expected to do. Group O had been called the "Universal Donor" because it could be transfused to any of the four types. It is true that the

serum of Type O would agglutinate the cells of Types A, B, and AB, but the serum of the donor is diluted so rapidly by the serum of the recepient that no actual or very little agglutination takes place. The point which is stressed in transfusions is that the cells of the donor must not be agglutinated by the serum of the recepient and for this reason Type AB is called a "Universal Recepient", since the serum does not contain any agglutinins and transfusions may be given from any group to this Type AB.

However, some transfusions give symptoms of mild shock, even when types are apparently the same, and it was thought that these types were not true to classification. Now hospitals do not only type the blood, but match its corpuscles against the serum of donor and recepient (36).

From time to time various anomalous groups have been reported. Jansky (17) found 19 cases whose blood cells reacted like Group B, but were not all of the same type. Ottenberg (26) found that of all his tests there was only one which did not fit in--" red blood cells of a Group A were not agglutinated by the presence of a clear Group O serum." This Group O blood agglutinated Group B cells but failed to agglutinate those of the four members of Group A against which it was tested.

Meleney, Stearns, Fortuine and Ferry (22) say that a Group B is occasionally met with whose serum has no agglutinin for Group A. Schutze in 13 Group B blood samples found five whose cells were feebly agglutinated, and nine whose cells were strongly and equally agglutinated by the serum from a member of Group O.

Hooker and Anderson (13) described the production of group-specific heteroagglutinins by inoculation of rabbits with human blood from the four groups, and to their surprise they secured a serum of even greater group-specificity with the cells of Group O, which are supposed to contain no agglutinogens, than those of the other three groups. Ottenberg and Reuben (27) reported a case in which the donors blood contained an abnormal agglutinin active against the patients cells (Group B) and Group O cells.

Clara Nigg (25), working with Group O cells, found one which was agglutinated by the serum of a number of Type B people, despite the fact that Type O is characterized by the lack of agglutinogen in the cells. Nowhere throughout the literature could any reference be found to direct work on Group O serum.

Landsteiner believed that the four groups of blood were dependent on two sets of hereditary factors which were allelomorphic. The allelomorphic genes A and B represented the agglutinogens present in the cells while

the agglutinins alpha and beta represented the allelomorphs of the serum.

With the work of Epstein and Ottenberg (5) the inheritance of these blood groups was first shown. Von Dungern and Hirschfeld (37) studied 72 families and concluded that their inheritance was consistent with the Mendelian formula. Later work of Hirschfeld (10) changed the theory of inheritance. Instead of there being two sets of allelomorphs there are three, the two dominants A and B, and the recessives R. All work to date has justified this viewpoint. In medico-legal cases it is possible sometimes to demonstrate the paternity or non-paternity of children by computations of the laws of inheritance.

In 1927 Landsteiner and Levine (16) found further individual differences in human red cells, and demonstrated the existence of three additional agglutinogens, M, N and P, by means of rabbit immune serum. These agglutinogens differ from A and B in so far as there are no natural corresponding agglutinins existing in human blood, so that these agglutinogens can only be detected by means of immune agglutinins prepared by injecting human blood into animals.

In 1928 Landsteiner and Levine (20) demonstrated that the agglutinogens M and N were inherited as Mendelian dominants, and also showed that they were

inherited independently of A and B agglutinogens. These authors further proved that M and N were not inherited independently of each other, but that the inheritance of M and N may depend upon a pair of single allelomorphic genes, M and N. Such a theory would permit the existence of only three different genotypes: MM, MN, NN, corresponding to the phenotypes M plus N negative, M plus N plus, N plus M negative. In other words, a blood lacking both M and N could not exist. As a matter of fact, in an examination of several thousands of samples neither Landsteiner nor Levine (21) found an M negative N negative blood. So now by means of the two different sets of Agglutinogens it is possible by means of blood tests to detect one third of all illegitimate children.

Experimenters have reported changes in blood groups due to disease. Ferrero (6) has shown that blood groups must be considered as constant factors throughout life, "unmodified by physical, chemical or humoral factors and that, contrary to Politzer and Rapisardi, who hold to the non-agglutinability of mother's corpuscles by infant's serum, the latter behaves according to its group characteristics." In the six sets of twins he examined, the blood groups were identical. Blood groups do not change even after death.

Experimental.1.

Before the problem was started it was decided to secure 1000 samples of Type O serum. To do this it was necessary first to type individuals who were willing to give a sample of blood. Since about 45% of the population are Type O, it was assumed that approximately 3000 people must be typed to secure this number, but after securing Type O samples from some it was impossible to bleed them from the arm, or they refused to be bled; consequently, the number of people typed before securing 1000 Type O's was 4850.

The typing sera were taken from various Type A and Type B people. The sera of the Type A donors were pooled to make the serum of uniform strength, and the same was done with Type B serum. As great numbers were typed during short intervals, it was necessary to keep large supplies of typing sera on hand.

It has been shown by numerous investigators that sera may give false results from three causes; 1. Hemagglutinins being decomposed by bacteria; 2. Development of bacteria giving rise to new products causing pseudo agglutination; 3. Contaminated sera containing agglutinative bacteria. To guard against these errors the blood was drawn as aseptically as possible and placed in sterile tubes containing sterile glass beads.

The blood was then shaken to defibrinate, centrifuged and the sterile serum was placed in autoclaved ampules, care being taken not to contaminate. To further insure sterility, the method of Rosenthal (32) in preserving serum was used. To each c.c. of sterile serum of Group A was added .01 c.c. of a 1% aqueous solution of neutral acriflavine and .01 c.c. of a .5% aqueous solution of basic fuschin. To each c.c. of Group B serum was added .02 c.c. of a 1% aqueous solution of brilliant green. Thus not only was the serum preserved from decomposition or contamination but the different colors served in preventing errors in reading of wrong sera. By this method Type A serum was colored red and Type B green.

Blood for typing was taken from the ear of finger by a small lancet, enough being taken to color slightly 3 c.c. of a .9% saline solution. As the blood was typed within an hour no precautions were taken to keep the blood sterile or aseptic. A drop of Type A serum and Type B serum respectively was placed on either end of a slide with a few drops of the blood-saline suspension being placed in each drop of serum. After mixing thoroughly the slides were placed in petri dishes to avoid evaporation and concentration of the salts. The serum of A would clump both B and AB cells; whereas, the serum of B would clump A and AB cells. If both the

sera clumped the blood cells it was Type AB; if neither clumped, it was Group O. If A serum clumped the cells and B serum did not it was Type B, and if B serum clumped the cells but A did not it was Group A. The results were read macroscopically and all Type O's which were slow in showing that peculiar suspension color, which indicates that no agglutination will take place, were discarded. The time alloted for agglutination was 15 minutes.

All Type O people, who consented, were then bled from the median cubital vein and three to five c.c. of blood withdrawn. The blood was defibrinated by whipping and centrifuged for 15 minutes or until a clear, straw-colored serum was obtained. Each Type O blood was then retyped by taking some of the centrifuged cells and making a 3% cell-saline suspension. This was placed in a small tube to which an equal amount of saline was added and also an equal amount of one part A serum and one part isotonic salt solution. The operation was repeated, substituting Type B for Type A serum (40). A small amount of these mixtures was taken from time to time and examined on a slide to determine if agglutination had taken place. A control of known Types A and B cells was run each time.

Fresh blood was then secured from Type A and Type B individuals by means of a syringe and the bloods

centrifuged. The serum was preserved for further typing and the cells were suspended in 10 c.c. of physiological salt solution. They were shaken for a few minutes and centrifuged, care again being taken not to shake too hard or wash to often, so that the agglutinogens might not be extracted from the cells (2).

The same set up was then used as in typing with the exception that a drop of the Type O serum was placed on either end of the slide. To the one drop of Type O serum was placed a 2% isotonic salt suspension of Type A cells, to the other was placed a 2% suspension of Type B cells. Normal Type O serum contains agglutinins A and B, and if this Type O serum was normal agglutination would be expected on both ends of the slide, since agglutinogens A and B were present. In the event that Type O serum lacked A or B agglutinins, agglutination would occur only on that sample on the slide in which both factors were present. If the serum lacked both agglutinins, agglutination would take place on neither end of the slide.

In the 1000 Type O samples which were examined, agglutination took place on both ends of the slide, showing that all Group O serum examined was normal, that is, contained alpha and beta agglutinins.

Recapitulating briefly, Type O serum should contain agglutinins alpha and beta. If to a drop of Type O serum

is added a suspension of Type A cells, agglutination should take place for the corresponding agglutinogen and agglutinin are present. The same reaction would be expected using Group B cells, and did, actually, occur. The results were therefore negative in that no agglutinin deficient Group O serum was found; positive in that this experimental work shows, in 1000 cases, no evidence of sub-groups in Type O serum.

The people donating serum were of mixed nationalities, of the European type. It was impossible, on account of this mixture of races, to show differences in blood groups among the different nationalities.

The percentage of Group O individuals was slightly higher than most investigators give (3.14%). Group B was about average, while Groups A and AB were slightly less than the average percentage given, being about 1.5% less. The table below shows the differences between Snyder's average (20,000 cases) and the average of the people typed here (4850 cases).

Group	O	A	B	AB
Percentage (here)	48.14	39.34	10.11	2.41
Percentage (Snyder)	45.00	41.	10.00	4.00
Percentage difference	3.14+	1.66-	.11+	1.5-

Experimental, 11.

The collecting of pathogenic blood was done at the State Sanitarium for Tuberculosis, located at Galen, Montana. The same procedure in typing was carried out

here as was done with normal blood.

Shortly after the discovery of the four blood groups, many investigators attempted to link specific blood groups with certain diseases for, as Proescher and Arkush (28) state,

> "When we attempt to determine the causes for the existence of immunity or susceptibility in certain individuals, when we try to find a practical explanation for the biological varities in mankind, we naturally look for aid to the principles of evolution and racial adaptation. If this is justified it becomes desireable to recognize the specific types and to do this we resort to the use of various indices. Thus we attempt to relate biological reactions on the one hand, with anthropological measurements, chemical reactions, blood groups, complexion etc., on the other hand. It is evident that the index of the greatest value will be that which most accurately and conveniently separates into practicable groups the members of the human race. This index is a reflection of nature. The results of Hirszfold's and others indicate that blood groups may, for some purpose at least, fulfill the requirements quite well."

With this idea in mind an attempt was made to prove or disprove the theory that certain groups were more susceptible to Tuberculosis than others. There was no attempt made to show a hereditary factor with complete linkage , but, as explained in the introduction, an attempt was made to demonstrate that certain groups through some physical or chemical change would make the individual more susceptible to certain diseases.

Hirszfeld (12) has made most important studies

concerning the relation of blood groups to diseases. In diptheria, for example, they have shown that immunity for this diseases is not connected with a certain blood group, but inherited with that blood group. This is doubtful, however, for if this is true it means that there is a partial or complete linkage of some hereditary factor with the blood group factor. In this case, although the original mutation would have been linked with but one of the blood group factors, subsequent crossing over would in time distribute the factor proportionately among the four groups.

Other investigators have tried to connect malignant tumors and mental diseases but with no results (15).

Froescher (26) working with 1525 cases of insanity found for Group O a percentage of 53.8; for Group A a percentage of 28.2; for Group B a percentage of 16.5; and for Group AB a percentage of 1.5. He compared these against normal groups which were asffollows: Group O 40%; Group A 43%; Group B 12.3%; Group AB 1.5%.

Such figures would indicate qualitative changes, but at the same time do not convey the facts, because the change should be considered in proportion to the original value. Thus, the shift in Group O distribution of 13.8% is not more than, but exactly the same as the Group B change of only 4.2%. Further results showed,

> "that the chances for normal individuals of blood groups O, A, B and AB to develop a psychosis is in the ratio 4:2 :: 4:1 respectively. This we take to mean that the chances or liability for normal persons of blood Groups O and B is equal and twice as great as for Members of Group AB."

Returning to the evolutionary theory--all people universal donors, at one time, and then mutations--it would seem that the crudest and least highly developed of nervous systems might belong to Group O members. The more simple the less susceptible to injury. In other words Group O should be less susceptible, and Froescher found this to be the case.

Hermanns and Kronberg (8) found no relation of blood groups to disease, with one exception. In hyperthyroidsim they find a high incidence of Group O, in goiter an increased frequency of Group A.

Hirschfeld (9) could not confirm the report of Weitzner in which he claims that individuals of Group AB are more susceptible to cancer than Group O. In 150 cases studied the distribution was equal.

Thomsen found that among 1151 individuals from 65 to 102 years of age his results did not confirm the hypothesis that certain diseases, such as cancer, tuberculosis etc., attack by preference individuals of certain blood groups, which is in direct contradiction to the results of this work. It must be said, however, that his results are not final, for may diseases

are not fatal. His results were: Group O 44.1%; Group A 43%; Group B 8.3%; Group AB 4.5%. From these figures he concluded that blood groups have no connection with disease. Some investigators give the normal percentage of Type B's as 8%, others place Type B's as high as 16% while an average of all current estimations would place Type B at about 13%. In other words, since the general norm has not yet been established absolutely for all the types, Thomsen cannot be too sure that 8% is the normal expectation of Type B individuals, and there is valid grounds on which to state that according to his figures the percentage of Type B individuals has decreased, and consequently, Type B shows susceptibility.

Kaczansky (14) in comparing the Dick and Schick tests to blood groups found: "that the percentage of immune individuals in different blood groups is practically the same."

Mirovesco (23) found the following:

	Group			
	O	A	B	AB
Measles (64)	4	35	8	17
Pertussis (14)	1	6	0	8
Scarlet Fever (35)	5	14	13	16
Typhoid (36)	6	17	18	5

Some authors have compared schizophrenia and manic depressions by means of blood groups, notably Raphael (30), with no specific relationship being observable.

So investigators have shown with some success the relationship of blood groups to disease. Raphael and Searle (30) in a study of groups and tuberculosis found no correlation, their percentages of difference between the normal and pathogenic blood being too small to draw any conclusion.

At Galen 100 people were typed with the following results:

Tuberculosis	Group O	Group A	Group B	Group AB
Galen	50%	29%	18%	3%
California(Raphael)	44%	40.2%	13.5%	2.3%
Non-tubercular				
European Average	39.2%	43.4%	12.8%	4.68%
Average Amer. Worker	44.2%	38.7%	11.3%	5.8%
Montana Average	48.14%	39.34%	10.11%	2.41%

Here would seem a tendency for Type B to be more susceptible than the other three types. It will be noticed in the above chart that the Montana normal average for Group B is 10.11%, while the Galen average for the same Group was 18% or a difference of 7.89%. Likewise,it can be seen that Group A is 10% lower at Galen than the Montana average, which indicates some immunity to tuberculosis, while Group B shows susceptibility.

One fact was overlooked until the final check up was made: namely, many of the patients at Galen were suffering from Silicosis or what is commonly called miner's consumption, a cutting of the lung tissue by abrasive material. Some had no doubt become easy prey

to tuberculosis after having silicosis, which would not be a normal infection, and which nullifies the results which were obtained in this work.

There were 35 cases of tuberculosis at Galen in which silicosis could be definitely ruled out, and the results were surprising. It was found that Type B gave a definite and more pronounced tendency to tuberculosis than in the first results. There was an increase of 13.8% in Group B over the average normal Group B, indicating susceptibility. There was a decrease of 13. 8 % in Type A, showing an immunity or decrease in susceptibility. Groups O and AB did not show any differences from the normal average of these groups.

	Group O	Group A	Group B	Group AB
Tuberculosis	47.23%	25%	25%	2.77%
Non-tubercular	44.2%	38.7%	11.3%	5.8%
% difference	3.03% +	13.7% -	13.7% +	3.03% -

In the percentage difference, when the normal average of the Group was less than the average of the individuals suffering from tuberculosis the percentages were marked positive or plus, and when the opposite was true they were marked negative.

A great number of the patients at Galen wre of Irish descent, so it might be said that the reason for such a high percentage of Type B's was because of this racial fact, that is, a greater percentage of the Irish are Type B than the normal. To ascertain the truth of

this, 45 individuals whose paretnts were Irish on both sides of the family were typed with the following results: Group O 53.3%; Group A 31.1%; Group B 15.5%; with no Type AB's. It is seen that the Irish race approximate the normal average within 3% in Type B and therefore the increase of 13.7% of Type B's cannot be explained by saying a large percentage of individuals would change the normal average of Type B even if taken from one the same race.

From experimental evidence, then, the results indicate that, irrespective of racial inheritance, that while the Group B individual shows a distinct tendency to tuberculosis, the Group A individual shows a distinct resistance.

Summary and Conclusions

1. Experimental work shows that of 1000 Type O blood samples none were found in which the serum lacked alpha, beta or both agglutinins.
2. The average of the normal blood closely approximates the average percentage of the American worker.
3. Results of work at Galen indicates that Type B is more susceptible to tuberculosis than the other three groups and that Type A shows a partial immunity.
4. These susceptibilities are inherited with the group regardless of racial susceptibility.

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