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ABO Gene and Fecundity

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Abstract

ABO B allele is the lowest frequency allele at ABO gene so possible causes of its rarity including that of de-selection of phenotypes of ABO B including lower fecundity is addressed. Although fecundity is only one aspect of selection though an important one, other phenotypes of ABO B may play a role in its frequency in human populations. If ABO B evidences lower fecundity, higher longevity could be a linked phenotype since research supports lower fecundity having a trade-off of higher longevity.

A phenotype of lower fecundity and higher longevity could be caused by an allele of a gene in linkage disequilibrium with ABO B. Research on dopamine beta hydroxylase (DBH) and its linkage with ABO supports this. Linkage disequilibrium of ABO B gene with dopamine beta hydroxylase (DBH) low activity gene having higher longevity would be an example of balancing selection supporting ABO B's survival albeit at a lower frequency than the other ABO alleles.

In ob-gyn patients, ABO blood types as a proxy for ABO alleles were compared as to number of pregnancies. The survey was of patients from one physician's practice in the southeast USA Patients having more than three children were studied since that is above the average fecundity in the USA and thus focuses on higher fecundity to compare as to ABO blood group.

The distribution in this population of ABO blood groups was ABO O .44, ABO A .42, ABO B .09, ABO AB .05. Thirteen patients had both known ABO blood type and were of greater than three parity. ABO O type was present in five of these patients, ABO A in seven patients and ABO AB in one patient.

Though the study is quite limited by the small number of subjects, the lower fecundity of ABO B was suggested and raised the question of what counterbalancing linked genetic mechanisms like DBH low activity may be supporting survival of ABO B allele. The answer to this question increases knowledge of what phenotypes ABO gene and genes in linkage disequilibrium to it codes for.

Keywords: ABO gene; DBH gene; fecundity; longevity; Pleiotropy

Introduction

The ABO alleles are traced to 20 million years ago with the fish to amphibian evolution. ABO A is thought to have been the only ABO allele in the first homo sapiens. The second allele to appear is thought to have been ABO B, emerging some 3.5 million years ago. ABO B allele has been thought to have been de-selected from populations and to have reappeared several times. ABO O first appeared some 2 million years ago. [1,2]

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Current world population frequencies are ABO O .47, ABO A .21, ABO B .09 and ABO AB .03. Whether this is a result of neutral or random evolution or a result of selection is debated, but given the time frame and the scarcity of the fossil record, a consensus is lacking.

Based on current aboriginal Australian populations, 50,000 years ago ABO frequencies of migrants from Africa to Australia were ABO A .60 and ABO O .40. So from that migration from Africa no ABO B gene can be inferred to have been present in Africa at that time. ABO frequencies in later migrations from Africa can not be as clearly deduced given the scarcity of any modern day aboriginal populations dating from the time of African ancestors migrating to Asian and Middle Eastern and further European populations. However modern day population ABO frequencies in Indian subcontinent are fairly equally distributed among all the ABO alleles. Middle Eastern populations are polymorphic for the ABO blood groups as are Asian and to a lesser extent European populations. The frequencies of ABO alleles in those populations have been thought caused by malaria as a vector de-selecting ABO A. [3]

The time of the origin and/or proliferation of the ABO B allele in the Indian subcontinent and in China as well as in areas as diverse as the Baltic shores and in eastern Europe may be much later than 50,000 ya as a result of Mongolian expansions in various time periods as documented by pre-historical archeological studies and in historical accounts.

Mongolian populations have highest frequency of ABO B allele in the world at .40-.45. It is not known whether a mutation occurred in Mongolia to explain this high frequency, but migrations from Africa to Asia some 40,000 years ago could have included ABO B allele. It is not known what the frequency of ABO B in Africa would have been at that time, but current frequencies in Asia would suggest ABO B to have had frequency of .25-.30 in Africa at that time. Current ABO B frequency in Africa is .20. So selection, founder effects as well as random effects seem to have acted to produce these differing ABO frequencies in various timeframes. Given the lowering of ABO B frequency to .09 in modern times compared to higher ABO B frequencies in historical human migration periods ABO B allele could be trending toward another extinction period. This lowering of ABO B allele frequency could suggest selective disadvantages of ABO B allele. ABO incompatibility is the most frequent genetic cause of fetal loss from a single gene and thus a force of selection. The most frequent allele at the ABO locus and the most frequent ABO allele producing fetal loss is ABO O since mothers of ABO O type have natural antibodies against ABO A and ABO B and ABO AB fetuses with a result of loss of 3% of such conceptions. [4]

Other selective factors are at work that have produced modern day frequencies of ABO O .47 , ABO A .21, ABO B .09, ABOAB .03. Negative frequency dependent selection relates to gut bacterial or parasitic effects favors rare alleles thus ABO A and B while balancing selection by viral vectors via natural antibodies against A and/or B antigens carried by different viral vectors advantages ABO O [5-7]

Another factor de-selecting ABO B could be low fecundity from a lower propencity to interact intimately with other people. Genetic cause of this could be linkage disequilibrium of ABO B with DBH low activity. The major allele at the most studied gene for DBH activity, rs1611115C, codes for high activity variant and is the ancestral allele. The minor allele codes for low activity and like ABO B in linkage appears to be deselected in human populations. Extreme phenotypes associated with low activity DBH include ADHD, autism, schizophrenia all of which are associated with low fecundity. So this type of lower fecundity based on lower interpersonal intimate interaction as a phenotype of low activity DBH appears to be also a phenotype of ABO B and thus a possible contributor to the low frequency of ABO B allele in human populations. [8-16]

Materials and Methods

To approach this question, patients in an ob-gyn office were assessed for ABO blood type and possible association with higher parity. The prediction was that having ABO B blood type by virtue of linkage disequilibrium with DBH low activity and thus lower propensity to interact intimately to form families with others would have lower parity.

Parity greater than 3 was assessed as to ABO blood type. Parity greater than three was queried to assess higher fecundity since this number exceeds average fecundity in modern USA population. Rate of patients without any partners using donor sperm was very low so still allowed using higher number of pregnancies as a possible measure of propensity toward higher intimate interaction with others. Reproductive rights of individuals to choose are high in this population and this data was collected prior to the Supreme Court overturning Roe vs. Wade decision and the enactment of more restrictions on reproductive rights in this state.

A second query was for whether the patient had a twin or higher order multiple pregnancy. This query was assessed to approach aspects of fecundity less related to higher propensity to interact intimately with others and would be predicted to be less related to low activity DBH or to ABO blood type. That is, patients having multiples could have only 1 or 2 pregnancies but 4 or more children.

Results

The distribution in this population of ABO blood groups was ABO O .44, ABO A .42, ABO B .09, ABO AB .05

Thirteen patients had ABO blood type listed and were of greater than three parity. ABO O, A, and AB had 5 and 7 and 1 such high fecundity states respectively.

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Five patients had multiples. ABO O had 3 such pregnancies, and ABO A had one, and ABO B had one. So no relationship to ABO blood groups to having multiples in pregnancy was noted as predicted.

Discussion

This finding of ABO B blood group having a lower fecundity could be based on a phenotype of lower propensity to interact intimately with others because of ABO B linkage disequilibrium with low activity DBH. ABO B in linkage disequilibrium with DBH low activity allele coding for high dopamine to epinephrine ratio would predict less propensity to interact with other people. Other possible extreme phenotypes of low activity DBH such as autism and schizophrenia mirror this state of behavior. "Negative symptoms" in schizophrenia and perhaps autism relate to dopamine effects in subcortical areas of the brain and are consonant with the low activity DBH phenotype of low intimate interaction with others putatively causing low fecundity.

While the current world frequency distributions of the ABO blood groups relate to evolutionary events that are not completely elucidated, more study of fecundity could highlight this phenotype's role in ABO frequency distributions.

If ABO B allele is associated with a lower propensity for intimate behavior with other people and thus a lower fecundity, this allele may be on tract to remain at stable though low frequency or on the trend of disappearing at least for a time from some modern populations. If on tract to remain at stable low frequency, there must be compensatory mechanisms to offset low fecundity such as is seen with ABO's linkage disequilibrium with DBH whereby low frequency DBH allele linked with ABO B may cause both lower fecundity and a counterbalancing higher longevity. Lower fecundity as a trade- off that increases longevity has been supported by research on the theory that somatic linked genes may be less physiologically supported than germ-line related genes over the long-haul of the lifespan. [17]

Since ABO blood type of the individual consists of two alleles where A and B are dominant and O is recessive thus making for inaccuracy in which gene in any given ABO blood type is affecting any given phenotype, studies of ABO alleles and longevity would be more relevant. However studies of ABO blood type and longevity have been done but without consensus reached in the research literature regarding longevity and ABO blood type. In a Japanese study a modern population of fairly equal ABO blood type frequencies and homogeneous ethnicity, ABO B blood type is of highest longevity. (18) and in linkage with ABO B, low activity DBH also has phenotypes promoting longevity including lower hypertension and lower cancer risks. So DBH low activity appears to be in linkage with ABO B and to associate with both lower fecundity and higher longevity. Conversely lower longevity could be a correlate of higher activity DBH and ABO group A and ABO O via risks of increased intimate interactions with other people promoting such exposures as infectious illness and interpersonal violence as well as accidental death. [19-25]

Since high activity DBH would evidence higher norepinephrine to dopamine ratio, the higher adrenergic effects can be seen to promote more activity if not more aggressiveness, more risk-taking. (26) Of course humans are social beings and thus outgoing, active, enthusiastic and energetic but a higher level of these traits which are associated more with ABO O and ABO A as well as high activity DBH could open more avenues of shorter life because of extreme levels of activity and more interaction with others. [27-33]

Biochemical basis for this putative DBH low activity effect on low fecundity and on higher longevity would be high dopamine: norepinephrine ratio. Addressing avoidance type behaviors such as those seen in individuals with autism and schizophrenia as a guide, dopamine activity in subcortical brain and in the frontal brain causing autism and schizophrenia relate to low activity DBH and associated avoidance of normal instinctual behaviors such as childbearing and even enjoying company of others for conversation and companionship.

Conclusion

Many genetic linkages and ethnic factors and stratification of populations may explain some of the associations research has found with ABO gene including thrombosis and cancer, infectious diseases, and behavior. The possibility of ABO B gene linked with both lower fecundity and higher longevity via linkage disequilibrium with DBH low activity, could be easily tested in large populations controlled for ethnicity and other cultural factors, and this small pilot study suggests that lower fecundity could be related to ABO B blood type.

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