



ABO GENETIC SYSTEM, SEXUALLY TRANSMITTED INFECTIONS AND ANDROGEN-ASSOCIATED DERMATOSES

Simeon Radev¹, Ilko Bakardzhiev², Diliانا Panova³, and Vladimir Michev⁴

¹Clinic of Dermatology, Naval Hospital, Varna, Bulgaria, ²University Medical College, Varna, Bulgaria,

³Piedmont Hospital, Atlanta, GA, USA, and ⁴Clinic of Dermatology, Military Medical Academy, Sofia, Bulgaria

In the middle of 20th century, it was proved that ABO genetic system was the result of play of selection, including the infectious mortality due to two deadly epidemic collisions in the antiquity and with the main scene being Asian societies. It was discovered that plague tended to kill blood group O while smallpox blood group A carriers. Onwards no link was sought between this evolutionary phenomenon and blood group-related sexually transmitted infections and recurrent androgen-associated dermatoses (such as pityriasis versicolor and acne vulgaris) as well as sexual and fertility activity. Here we Dance Round such possible links. We found that these are expressed more strongly by BG-B carriers. We emphasize the genesis of blood group-related population gene pool equilibrium level and its attributes such as complex defense responses and co-operated immune reactions.

Biomed Rev 2011; 22: 77-80.

Key words: ABO genetic system, cell-mediated immunity, population gene pool, sexually transmitted infections, recurrent androgen-related dermatoses

INTRODUCTION

Apart from significant advances in the study of human sexual potency, many aspects remain to be elucidated. The difficulty is in the fact that such an essential biological function and sociocultural phenomena along with related sexual practices cannot be reduced to a common denominator. In the middle of 20th century, it has been established that blood group B (BG-

B) is a factual marker of Eastern belonging. Blood group B distribution in India preserves approximately equal values: for 1942 – 34.8% (1), for 1966-1970 - 32-42% (2), and for 1997 – 37.4% (3). Values concerning Japan are even more stable: for 1933-1944 21.9-23.1% (1) and for 1966-1970 – 22.2% (2). Here the reasons consist in the emigration processes which do

Received 2 December 2011, revised 18 December 2011, accepted 23 December 2011.

Correspondence: Simeon G. Radev, MD, PhD, Consulting Dermatologist, Clinic of Dermatology, Naval Hospital, 3 Hristo Smirnovski Street, BG-9010 Varna, Bulgaria. E-mail: simoesradeff@yahoo.co.uk

not alter BO polymorphism. Conversely, for the British Isles (4) and Germany (2), BG-B incidence increases on the expense of BG-A because of the intensive post-war immigration

Blood groups and antibody responses towards some sexually transmitted infections and their tropism to some androgen-associated dermatoses

Research on the relationship between blood groups and sexual potency continuously grow in depth and quantity (5). We were inspired to do this investigation when during the one-act mass screening seroepidemiologic testing in 2001 among a total of 536 young, 18-year-old Caucasian navy sailors. They were examined a year before joining the Navy and then every season, i.e. 12 times during their 3-year service as well as every two years after dismissal. Telephone interviews as well as air-mailed or e-mailed questionnaires were used.

The purpose of the study was to establish if some well-known recurrent androgen-associated dermatoses (RAAD) such as *pityriasis versicolor* (RPv) (when starting during navy or permanently, Pm) and *acne vulgaris* (RAv) were expressed more strongly by BG-B carriers. It was established that BG-B carriers were higher generators of elevated antibody titres (> 1/20) against sexually transmitted infections (STI) such as herpes simplex virus-2 (HSV-2) ($p < 0.05$ - $p < 0.001$) and cytomegalovirus (CMV) ($p < 0.001$) (Table 1) than the other BG carriers. The diagnosis of CMV infection was made by a classic hemagglutinin test while that of HSV-2 by additional clinical monitoring concordant in more than 85% of the cases with antibody responses. The possible BG-B-related higher sexual potency was tested during a 10-year long (2000-2009) randomized longitudinal population follow-up study. In order to achieve a higher preciseness we examined under homogenous endogenous and exogenous conditions such as matched gender, age, secondary educational level, nutrition, military stress level, inhabitants in the coastal area as well as

origin from classic population morphs (CPMs) i.e., hereditary villagers (HVs) and hereditary town dwellers (HTDs).

The cause for discriminating the town-village hybrids (TVHs) will be stated later on. The concept that ABO group polymorphism results from the play of the natural selection, i.e., the infectious mortality from plague and smallpox (2-4) goes with our data where BG-B dominates by 3.58 times among HTDs ($n=107$) (in 43%) than among VHs ($n=429$) (in 12%) ($p < 0.001$) being a consequence of the naturally rare disposition of the latter to these fatal infections.

As shown on Table 1, these RAAD gravitated by 1.25-2.16 times and by 1.65-2.0 times ($p < 0.01$ - $p < 0.001$), respectively, more strongly towards BG-B carriers than towards the other BG ones. A fascinating example was the unique strong BG-B carriers' tropism towards double (RPv and RAv) RAAD ($p < 0.05$ - $p < 0.01$). On the other hand, there was no similar correlation concerning the general recurrence rate of dandruff (RDf). This is in accordance with the hypothesis that Df, an abridged version of seborrheic dermatosis, is a particular kind of eczema aggravated by added commensal, *Malassezia* yeast rather than an infection *sui generis* as considered by some authors (6,7).

Concerning the aggregations of RAAD among BG-B carriers such as RPv+RAv, RPv+RDf, and RAv+RDf it is evident that Df is closer to Pv than to Av, however, there is no correlation to the high antibody responses towards HSV2 and CMV. Apparently, epidemiodynamics of Pv directly correlates with BG-B population frequency rate. Our data from illustrate a statistically significant BG-B domination by two times ($p < 0.001$) among Gypsy recruits of proven Indo-Asian origin ($n=325$) (in 32%) than that among Bulgarian ones ($n=850$) (in 16,2%). RPv incidence rate among Gypsy recruits is of 10.4% but among Bulgarian ones is of 6%. More interesting, generalized Pv (in one third of the cases on the face, too) is by 3.6 times more common among Gypsies (in 5.29%) than

Table 1. Relationships between ABO genetic system and sexually transmitted infections (STI) and recurrent androgen-associated dermatoses (RAAD) during navy (DN) and after navy (AN)

Blood group	n	Titres of STI		RAAD			RDf	Co.RAAD and RAAD+RDf		
		HSV2	CMV	DN and AN				DN and AN		
				RPv		RAv		RPv+RAv	RPv+RDf	RAv+RDf
		≥ 1/20	≥ 1/20	Start DN	Pm					
n/%	n/%	n/%	n/%	n/%	n/%	n/%	n/%	n/%		
O	157	21/13.4	26/16.6	30/19.1	0/0	27/17.2	65/41.4	7/4.46	25/15.9	10/6.36
A	217	72/33.2	104/47.9	25/11.5	6/2.76	45/20.7	58/26.7	6/2.76	7/3.22	8/3.68
B	108	52/48.1	63/58.3	26/24.0	6/5.55	37/34.2	13/13.5	19/17.6	14/13.0	0/0
AB	54	0/0	11/20.4	6/11.1	0/0	0/0	14/25.9	0/0	0/0	0/0

among Bulgarians (in 1.44%). A similar Pv dissemination patterns are typical of the Indian population (8.9) dominated by BG-B carriers (1-3). According to the first outpatient's attendance by students in East Africa, Pv is more common among men than among women as well as among Asian people than among individuals from other countries (10). Av sex-related tropism displays similar features (11,12).

Blood group-related population gene pool differentiation level and its attributes: complex immune and defense (health/disease) responses

All of these BG-associated diseases are multifactorial, i.e. a result from a complex interplay of polygenes and multiple environmental triggering factors. They are, however, not inherited in a simple Mendelian fashion and not associated with chromosomal abnormalities at all. Thus they are an area of supreme priority and challenge facing the contemporary medical genetics. Taking into consideration the basic laws of genetics formulated in 20th century (13-15) and seminal insights of Ajalla and Kriger (16), we recognize that there is another evolutionarily strong but missing key player that governs, subordinates and predetermines not only the well-known and suspected pathways of the pathogenesis and epidemiogenesis of the diseases but also the general outcome of health-disease responses' balance. This substantial common denominator represents an epigenetically assigned major phenotype-related population gene pool (PGP) differentiation level. The latter functions either as a harmonious co-adaptive genetic (allele and interlocus) equilibrium, or as a disharmonious interlocus interaction, the so-called epistatic genetic suppression. The first one represents the normal adaptation, i.e. health by itself, and is typical of CPMs and their BG-B carriers. The second one represents the disadaptation, i.e. the pathological states or diseases by themselves are typical of TVHs as an abridged version of Western hybrid societies (WHSs). That is why

WHSs turn into the most powerful generators of chronic, refractory and recurrent diseases, highly pathogenic flora carriers, infections and allergies.

The concrete PGP balance level was assessed through its sounding via some complex defensive traits such as multiple (triple or double) infectious allergic resistance (MIAR) or susceptibility (MIAS) to grippe and grippe-like conditions (GGLC), RTp, and allergy. The individual and population genetic make-up as causative factor possesses a predetermining g role for human defence and immune homeostasis through the interactions between the genes. These interactions are epigenetically (evolutionarily) assigned via concrete type and intensity of the population mating as compatible inbreeding or abnormal urbanogenic interbreeding and intrinsically related to it genetic suppression. It is due to the fact that in ancient times, BG-B carriers suffered a much more severe pressure of the selection (infectious mortality) which enhanced PGP harmonization and resulted in better defense and immune capacity.

Measurements of stability and power of basic and specific cell-mediated immunity (CMI) were accomplished by intradermal testing with Candidin (C), phytohemagglutinin (PHA) and Trichophytin (T). Individuals with positive delayed skin allergy, the so-called CMI towards C and PHA at one and the same time were classified as such with co-operated basic CMI (Co.BCMI) while those with marked Co.CMI and T were considered as such with co-operative specific CMI (Co. SCMI) (Table 2). BG-B carriers who survived the plague and smallpox epidemics responded not only with a higher Co.CMI ($p<0.01$ - $p<0.001$) but also with an implied general biological compensatory reflex, i.e. with accelerated fertility. This higher fertility goes with our data showing that the parents of the examined BG-B carriers have more than two children in 75% of the cases while among those of the other blood groups this occurs only in 40-50% of the cases ($p<0.001$). Hence one

Table 2. Relationships between ABO genetic system, defense polymorphism and co-operated basic cell-mediated immunity (Co.BCMI)

Blood group	n	Co.CMI		Defense polymorphism				PGP differentiation level
				MIAR		MIAS		
		Co.BCMI C+PHA n/%	Co.SCMI C+PHA+T n/%	triple n/%	double n/%	triple n/%	double n/%	general outcome H/D responses' ratio %/%
O	157	82/52.2	26/16.7	13/8.28	82/52.2	31/19.7	31/19.7	60.5/39.5
A	217	108/49.8	10/4.6	97/44.7	42/19.3	18/8.29	60/27.6	64/36
B	108	72/66.6	18/16.6	36/33.3	36/33.3	18/16.6	18/16.6	66.7/33.3
AB	54	23/42.6	0/0	18/33.4	12/22.2	12/22.2	12/22.2	55.6/44.4

might suppose that hyperandrogeny which determined this phenomenon was coded by evolution in the genome of BG-B carriers. There is no doubt that the intense sexual potency and fertility in Eastern societies resulted in a huge population density and, logically, sufficient sex was given the status of a cult (see e.g. Camasutra and other treatises).

CONCLUSION

Altogether, through a 10-year longitudinal monitoring and a multitheoretical scanning of the processes (17) we establish a significant link between BG-B carriers and some STI and implied evolutionary gene-encoded hyperandrogeny and fertility, respectively. This is confirmed by a striking relationship between BG-B and long-known AADs such as Pv and Av. We hold the opinion that ABO group polymorphism is the result from a powerful pressure of the selection by a selective BG-related infectious mortality (18-20) in ancient times. We found a confirmation of the ideas and hypotheses not only in the dramatic domination of BG-B and high sexual activity among Eastern societies but also in publishing data about a strong domination of some AAD among them. The BG-B carriers having passed through the deadly epidemics are, logically, owners of a relatively higher Co.BCMI and Co.SCM homeostasis and PGP-related harmonization due to a systematic tinkering and coining of evolution. Long-term prospective research is needed to better elucidate these essential topics.

ACKNOWLEDGEMENTS

The authors greatly appreciated Mr Zlatko Kerchev's and Dr Dimitar Tomov's help during the preparation of this manuscript.

REFERENCES

- Salzano FM. Blood groups and leprosy. *J Med Genet* 1967; 4: 102-106.
- Popvasilev I, Bliznakov H. *Blood Group Systems in Man*. Medicina i Fizkultura, Sofia. 1980; 21-23, 48-63 (in Bulgarian).
- Nanu A, Thapliyal RM. Blood group gene frequency in a selected North Indian population. *Indian J Med Res* 1997; 106: 242-246.
- Mitchell RJ. Variation in blood group frequencies in a single population: The Isle of Man. *Hum Biol* 1980; 52: 494-506.
- Ellis L, Ficek C, Das S. Eye color, hair color, blood type, and Rhesus factor: exploring possible genetic links to sexual orientation. *Arch Sex Behav* 2008; 37: 145-149.
- Schwartz RJ. Treatment of seborrheic dermatitis of the scalp. *J Cosmet Dermatol* 2007; 6: 18-22.
- Piérard-Franchimont C, Xhaufflaire-Uhoda E, Piérard GE. Revisiting dandruff. *Int J Cosmet Sci* 2006; 28: 311-318.
- Chakravarti MR, Verma BK, Hanurav TV, Vogel F. Relation between smallpox and the ABO blood groups in a rural population of West Bengal. *Humangenetik* 1966; 2: 78-80.
- Kamalam A, Thambiah AS. A study of 3981 cases of mycoses in the tropics. *Sabouraudia* 1976; 14: 129-148.
- Arya OP, Benett FJ. The epidemiology and prevention of skin disease in university students in East Africa. *Dermatol Int* 1968; 7: 196-203.
- Chetty GN, Kamalam A, Thambiah AS. Pityriasis versicolor. A study of 200 cases in a tropical Skin Clinic. *Mykosen* 1979; 22: 234-246.
- Vanbreuseghem R. Diagnostic et prevalence du Pityriasis versicolor en Afrique. *Bull Soc Franc Mycol Méd* 1973; 2: 165-166.
- Dobzhansky T. Evolution and environment. In: S. Tax, editor. *Evolution of Life*. University of Chicago Press, Chicago, IL. 1960.
- Haldane JBS. Population genetics. *New Biol* 1955; 18: 34-51.
- Mayr E. *Populations, Species, and Evolution*. The Belknap Press of Harvard University Press, Cambridge, MA. 1970; pp 48-66, 83-95, 102-205.
- Ajalla FJ, Kriger JA, editors. *Modern Genetics*. 2nd ed. C. Benjamin Commings Publishing Comp. Inc., Menlo Park, CA. 1984; pp 645-675, 751-791, 851-880.
- Radeff S, Mitscheff V, Balabanoff V, Miteff G. *Oberflächliche Hautaffektionen -Epidemiogenetische, valeomediznische und therapeutische Aspekte*. Varna, 2005.
- Vogel F, Chakravarti MR. ABO blood groups and smallpox in a rural population in the West Bengal and Bihar (India). *Humangenetik* 1966; 3: 166-180.
- Vogel F, Krüger J. Statistische Beziehungen zwischen den ABO-Blutgruppen und Krankheiten mit Ausnahme der Infektionskrankheiten. *Blut* 1968; 16: 351-376.
- Shuster S, Blatchford N. Seborrheic dermatitis and dandruff - a fungal disease. *Roy Soc Med Serv* 1988; 132: 1-54.