The significance of the 2.2 percent increase in b is interpreted as follows. The tetrahedral sheets of the kaolinite structure normally are constrained by their attachment to the Al–O,OH octahedral sheets by rotations of the tetrahedra through angles of about 11.3° (*8*) so that the sheet symmetry is not hexagonal but is more nearly ditrigonal. This is illustrated in Fig. 1, where larger rotations (20°) are shown in order to clarify the contraction of the sheet structure by these rotations. When the octahedral sheets are disrupted by dehydroxylation, the constraints on the tetrahedral sheets are removed, and the highly charged Si ions will repel each other to the maximum limit set by the Si–O bond length, 1.62 Å. The maximum relaxation of the tetrahedral sheet corresponds to the hexagonal arrangement shown in Fig. 1, and from the geometry of the tetrahedron it follows that $b = 4\sqrt{2}$ (Si–O) $= 4\sqrt{2}$ (1.62) = 9.15 Å. This agrees exactly with the measured parameter for metakaolin.

The present results provide no direct confirmation for the Al–O chain arrangement in metakaolin proposed by Pampuch, but indirectly they indicate a similar conclusion. In attempting to fit an Al–O sheet structure with Al in fourfold coordination, to the original a and b parameters of the kaolinite structure, Brindley and Nakahira (*1*) found that the Al–O tetrahedra must be very highly distorted. Indeed, if an Al–O sheet structure is maintained after dehydroxylation, some reduction in the a and b parameters is to be expected and certainly not an expansion. The considerable expansion now observed points strongly toward a disruption of the octahedral sheet structure, and to this extent the chain arrangement suggested by Pampuch is supported.

G. W. Brindley
D. L. Gibbon
Department of Geochemistry and Mineralogy, Pennsylvania State University, University Park 16802

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Genetics of Diego Blood Groups in Guatemalan Indians: Use of Antiserums to Diego a and Diego b Antigens

Abstract. *Red blood cells of 255 inhabitants of San Antonio Palopó, an Indian community on the eastern shore of Lake Atitlán, Guatemala, have been typed with antiserum to Diego a (Di^a) and the newly discovered antiserum to Di^b. Individuals with erythrocyte antigenic types Di(a+b−, Di(a+b+), and Di(a−b+) have been found, but the type Di(a−b−) has not been encountered. Population frequencies of antigenic types and family studies support the hypothesis that the erythrocyte antigens, Di^a and Di^b, are controlled by two codominant alleles at a single autosomal locus.*

The discovery of the antiserum to Diego b (Di^b) antigen (*1*) provides additional information on the genetics of the Diego blood group system, a polymorphism in American Indians and some Asian populations, and a monomorphism in Caucasians and Negroes (*2*). Initially, a single erythrocyte antigen, Diego a (Di^a), was detected by antiserum to Di^a (*3*), and subsequently it was found that the frequency of the phenotype Di(a+) varied from 0 to 0.5 in American Indians (*4*). Genetic analysis of data from families studied with the antiserum to Di^a has shown that the polymorphism consists of two (or more) alleles at a single autosomal locus, one of which, Di^a, is dominant and controls the antigen, Di^a (*3, 5*). We have been studying the distribution and the inheritance of the erythrocyte antigens Di^a and Di^b in Mayan Indian descendents in Guatemala, and we present here the results of our initial genetic analysis. To our knowledge, these are the first genetic studies with both antiserum to Di^a and antiserum to Di^b.

The antiserum to Di^b was discovered in two Di(a+) Mexican Indian women with blood transfusion compatibility problems (*1*). Their serums, which did not react with their own red blood cells, reacted consistently with Di(a−) and Di(a+) erythrocytes selected at random with respect to other antigenic types. Red blood cells from the parents and siblings of one of the women were Di(a+) in each case, and, except for cells from one sibling, they reacted with the newly discovered serums.

Table 1. Frequency distributions of Diego erythrocyte antigenic types determined from reactions with antiserums to Di^a and Di^b antigens.

Antigens	Distribution		
	Males	Females	Total
Di(a+b−)	1	2	3
Di(a+b+)	15	39	54
Di(a−b+)	70	128	198
Di(a−b−)	0	0	0
	86	169	255

Test for association of antigen types with sex: $\chi^2_{(1)} = 1.050^*$; $P > .30$

* Types Di(a+b−) and Di(a+b+) were pooled because of the small numbers of individuals of the former type.

The antiserum to Di^a was provided by Dr. M. Layrisse, and the antiserum to Di^b was made available to us by Hyland Laboratories. Both antiserums require antiserum to human globulin for the detection of the respective antigens (*6*). Blood was collected in ACD (citric acid, trisodium citrate, dextrose) solution and immediately refrigerated until processed. A volume of packed red blood cells was suspended in two volumes of 40-percent buffered glycerol (*7*) and then stored at −20°C. No more than 1 week (and usually less time) elapsed between obtaining a blood specimen and suspending the erythrocytes in glycerol. All samples were typed for erythrocyte antigens within 4 months from initial collection. In this laboratory, frozen stored erythrocytes show agglutination reactions with antiserums to Di^a or to Di^b identical to those of fresh red blood cells. The individuals studied in this investigation are Cakchiquel-speaking, Mayan Indian inhabitants of San Antonio Palopó, an Indian community on the eastern shore of Lake Atitlán, Guatemala. This population shows high frequencies of blood types O, M, and MN, and a very low frequency of type rh (cde). Previous studies in two other lakeshore communities (*8*) revealed frequencies of 0.116 and 0.200 of the Di(a+) phenotype.

The sample studied was made up of 255 Indian inhabitants of San Antonio Palopó. The sample contained complete "nuclear" families (father, mother, and one or more offspring), fragments of nuclear families, and individuals of

Table 2. Frequencies of Diego erythrocyte antigenic types and of presumed genotypes and alleles controlling these types.

Antigens	Presumed genotype	Expected frequency	Total sample		Parents of nuclear families	
			Obs.	Exp.	Obs.	Exp.
Di(a+b−)	Di^a/Di^a	p^2	3	3.5	1	0.5
Di(a+b+)	Di^a/Di^b	$2pq$	54	52.9	9	9.9
Di(a−b+)	Di^b/Di^b	q^2	198	198.6	46	45.5
		$\chi^2_{(1)}$	0.096		0.587	
		P	.80 to .70		.50 to .40	
Frequency of allele Di^a, p			0.1176		0.0982	
Frequency of allele Di^b, q			0.8824		0.9018	
		Standard error	0.0143		0.0281	

Table 3. Parental mating types and distribution of Diego erythrocyte antigenic types among offspring. Figures in parentheses are those expected if the antigens are controlled by two alleles at a single autosomal locus.

Mating types		Matings (No.)	Offspring (No.)		
Erythrocyte antigenic types	Presumed genotypes		Erythrocyte antigenic type		
			Di(a+b−)	Di(a+b+)	Di(a−b+)
Di(a−b+) × Di(a−b+)	$Di^b/Di^b \times Di^b/Di^b$	19			44(44)
Di(a+b+) × Di(a−b+)	$Di^a/Di^b \times Di^b/Di^b$	7		13(11.5)	10(11.5)
Di(a+b−) × Di(a−b+)	$Di^a/Di^a \times Di^b/Di^b$	1		2(2)	
Di(a+b+) × Di(a+b+)	$Di^a/Di^b \times Di^a/Di^b$	1			1(0.25)

other varying relationships; two (and occasionally three) generations were included in the sample. Individuals were classified with respect to the following Diego erythrocyte antigenic types: Di(a+b−), Di(a−b+), Di(a+b+), and Di(a−b−) (Table 1). Of the 255 individuals studied, 198 (0.776) were Di(a−b+) and only three individuals possessed the Di(a+b−) type. No individual was found whose erythrocytes failed to react with at least one of the antiserums, that is, the type Di(a−b−) was not encountered.

The data indicate no association of the erythrocyte antigenic types with sex. The distribution of the antigenic types did not differ significantly between males and females in the sample (Table 1). These findings extend and support previous pedigree analyses (*3*) which demonstrated that Di^a was not linked to the X chromosome. If Di^a and Di^b are controlled by alleles, our data suggest that these are autosomal alleles.

If the antigens Di^a and Di^b are each controlled by an allele (Di^a and Di^b respectively) at the same autosomal locus, the frequency distribution in the population of erythrocyte antigenic types can be predicted by the Hardy-Weinberg law, which formally requires that mating is at random, that natural selection is not acting on the genotypes, and that generations in the sample do not overlap. Although the last criterion was not met in our sample, we estimated allele frequencies by counting genes (the maximum likelihood estimates), and derived the expected phenotype and genotype frequencies. The observed and expected frequencies are presented in Table 2, and the predicted values fit the observed data. Similar results were obtained when the data from parents of nuclear families were used to estimate allele and expected genotype frequencies (Table 2). Thus, the population data support the hypothesis that two codominant alleles at a single autosomal locus control these erythrocyte antigens.

Segregation of erythrocyte antigenic types was studied in the 28 complete nuclear families included in the population sample (Table 3). Of the six parental mating types expected when there are two codominant alleles at a single autosomal locus, we found four. The most frequent mating type encountered was that between two individuals each of phenotype Di(a−b+). Seven backcrosses, each with a parent of phenotype Di(a−b+), were next most frequently found. One cross between Di(a−b+) and Di(a+b−) parents and one intercross were also present. No exceptional segregants issued from any of these matings. The phenotypes of the offspring from these matings were those expected from the proposed genetic hypothesis.

Some of the backcross matings were informative for studying segregation of the Diego locus with other loci. No evidence was found for close linkage of the Diego locus to the Rh, MNSs, Duffy, or haptoglobin loci.

We conclude that the antigens Di^a and Di^b are controlled, respectively by separate alleles, Di^a and Di^b, at a single autosomal locus (or at closely linked loci). The genetic data presented here confirm the conclusions from serologic data (*1*) that antiserum to Di^b detects an antigen controlled by an allele of Di^a. Additional intercrosses and backcrosses are required to strengthen these conclusions. We could not adduce, from family or population data, evidence for a third allele at the *Di* locus, but more data must be examined for this possibility. The offspring of additional Di(a+b−) × Di(a−b+), Di(a+b−) × Di(a+b−), and Di(a−b+) × Di(a−b+) incrosses should be observed for evidence of segregation, and progeny of presumed backcrosses should be examined for evidence of segregation in the presumed homozygote parent. More individuals should be studied to find the erythrocyte antigenic type, Di(a−b−).

HOWARD M. CANN
BETTY VAN WEST
CLIFFORD R. BARNETT
Department of Pediatrics and Human Development, Stanford University School of Medicine, Palo Alto, California 94304

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