Genetic Structure of Mennonite Populations of Kansas and Nebraska

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Abstract We describe the gene frequency distributions for 29 different blood group, serum, and erythrocytic proteins for three Mennonite communities from Kansas and Nebraska and compare their gene frequencies with those of Amish, Hutterite, and Mennonite populations using the topological method of Harpending and Jenkins (1973). Subdivision of these communities into congregations reveals that the "fission-fusion" model best characterizes the relationship between the genetic patterns and historical events. These Mennonite populations, although reproductively isolated at the turn of this century, are presently entering the mainstream of US rural culture.

During the 1950s and 1960s geneticists, anthropologists, and physicians often focused on the genetics of human isolates. Small, geographically or socially defined populations that were reproductively isolated and highly inbred were being studied throughout the world (Goldschmidt 1963). The primary purposes of those investigations were to document the effect of genetic drift and to understand the mode of transmission of rare genetic diseases. This interest gave impetus to the study of such human populations as the Habbanite Jews, the Samaritans, Tristan da Cunha, Parma Valley, and St. Bartholemew. During this time the genetics of various Anabaptist groups, such as the Amish, Dunkers, and Hutterites, was investigated. These early genetic isolate studies yielded a bounty of information on the genetics of rare mutant genes, for example, the Ellis–van Creveld syndrome, limb-girdle muscular dystrophy, hemophilia B, and congenital deafness (McKusick et al. 1964). The documentation of genetic drift was less dramatic, even though D.F. Roberts's study of Tristan da Cunha succeeded in demonstrating the effects of unique

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historical events and stochastic processes on small populations (Roberts 1968).

Although the Amish and Hutterites were intensively studied, the Mennonites were by and large ignored by the early geneticists and anthropologists. With the exception of Allen and Redekop's (1967) prospectus for future research among the Old Colony Mennonites of Mexico and a publication of gene frequencies for 51 Mennonites encountered by Brown et al. (1974) in the Gran Chaco region of Paraguay, little is known about the genetics of the Mennonites.

The genetic structure of the Mennonite populations of Kansas and Nebraska is one portion of a long-term research program initiated by the University of Kansas during the mid-1970s on the genetics of differential biological aging. To date, these populations have been described demographically (Stevenson et al. 1989; Lin and Crawford 1983), anthropometrically (Devor et al. 1986a,b; Sirijaraya and Crawford 1989), physiologically (Devor and Crawford 1984a,b; Koertvelyessy et al. 1982), and genetically (Crawford and Rogers 1982; Rogers 1987).

The primary purposes of this article are (1) to describe the gene frequency distributions for various blood markers in the Mennonite populations of Kansas and Nebraska, (2) to examine the genetic structure of these populations and its relationship to the ethnohistory of the Anabaptists, and (3) to determine how systematic pressures, such as migration, affect the genetic structure of the Mennonites.

Populations

The Mennonite communities of Kansas and Nebraska trace their origins to sixteenth-century Europe. These diverse groups, constituting the Anabaptist or left wing of the Protestant Reformation, hold in common beliefs in the separation of church and state, adult baptism, and pacifism. Different Anabaptist denominations came to be identified in Europe by their prominent local leaders. For example, the followers of Jacob Hutter are known as Hutterites, the followers of Jacob Amann became the Amish, and the followers of Mennon Simons call themselves the Mennonites.

Persecution of the Anabaptists in Western Europe, particularly in the Netherlands and Austria, forced them to settle in the underdeveloped agricultural regions of Eastern Europe. In 1669 fifteen Mennonite refugee couples settled in the swampy lowlands south of Danzig in West Prussia and thus founded the Przechowka Church. The congregation increased

in number and maintained meticulous records. In 1821 all but seven families of this congregation emigrated to Russia and settled in the Ukraine near the Molotschna River. This congregation adopted the name Alexanderwohl, in honor of the Russian czar.

Economic conditions, changes in Russian governmental policies, and possibly internal strife induced the Alexanderwohl Mennonites to emigrate to the United States in 1874. Virtually the entire congregation emigrated, followed by many of their relatives and descendants, who continued the exodus until the Soviet Revolution ended most of this flow. On their arrival in the United States, competition between railroad agents for land sales and additional strife within the group resulted in the original Alexanderwohl community splitting into three major divisions. One group settled west of Lincoln, Nebraska, in today's Henderson. The other two groups settled in Kansas, 40 miles north of Wichita.

As these congregations prospered and grew in size, offshoots or subdivisions formed. Today there are ten congregations derived from or related to the original Przechowka Church. In 1874 the Kansas group was subdivided into two communities, Hoffnungsau and Alexanderwohl. Shortly after the turn of this century (1909), Tabor split off the Alexanderwohl congregation. In 1920 the Goessel church separated from the Alexanderwohl congregation. In addition, the Nebraska Mennonite Bethesda congregation also underwent a fission from the Evangelical Mennonites.

For purposes of comparison, we include a more conservative Mennonite group from Meridian, Kansas, in this study. This community, known locally as the Holdemans of the Church of God in Christ Menno, is a heterogeneous population founded by John Holdeman in 1859 in Ohio. The membership is largely Pennsylvania German and Dutch (Ostroger Mennonites who migrated in 1875 from Volhynia in central Poland) mixed with the Kleine Gemeinde Mennonites, who migrated to Manitoba from southern Russia in 1874.

Methods

We collected 1251 blood specimens from three communities of Kansas and Nebraska (Table 1), packed them in ice, and shipped them by air to the Minneapolis War Memorial Blood Bank for analysis. Although the sample size for Meridian appears to be small, it represents 54% of the total community and almost the entire adult population. The Goessel sample represents 47% of the entire Alexanderwohl church membership. The Henderson sample includes more than 50% of the town residents.

Table 1. **Size of Sample Used for Blood Analyses**

Population	*Sample Size (N)*
Goessel	616
Meridian	87
Henderson	549
Total	1252

We performed red cell typings for the following systems: ABO (anti-A, A_1, B), Rhesus (anti-C, c, D, E, e), MNS (anti-M, N, S, s), Kell (anti-K, k), (Kp^a, Kp^b), Kidd (anti-Jk^a, Jk^b), Duffy (anti-Fy^a, Fy^b), P (anti-P_1), Vel (anti-V+), Lutheran (anti-Lu^a, Lu^b), and several low-frequency antigens, including Mt^a, Fr^a, and Bu^a. We performed the red cell typings on microtiter (U) plates using 2% suspensions of washed erythrocytes and a modification of the method of Crawford et al. (1970). We diluted antisera to obtain optimal reactivity and incubated typings for 1 hr at room temperature (20°C). Before reading, we spun the typing plates for 2 min at 500 rpm. We also carried out tests that required an antiglobulin phase. For these tests we used microtiter plates, washing them three times with saline and rinsing in a Coombs rinser; we spun the plates before recording the results.

We phenotyped serum proteins either electrophoretically or through isoelectric focusing (IEF). Transferrin (Tf), haptoglobin (Hp), and ceruloplasmin (Cp) phenotypes were identified from acrylamide slabs with amido black stain (Polesky et al. 1975). The group-specific component (Gc) and the properdin factor (Bf) systems were simultaneously phenotyped using the agarose electrophoretic method described by Dykes and Polesky (1980). We employed the isoelectric focusing method of Dykes et al. (1981) for phenotyping Gc1.

We obtained stroma-free hemolysates for determining erythrocytic enzyme phenotypes by washing cells three times in saline, diluting 1:1 in distilled water, and centrifuging at high speed. Specimens were stored at −70°C until tested.

We phenotyped eight red cell protein markers. Adenylate kinase (AK), 6-phosphogluconate dehydrogenase (6-PGD), and acid phosphatase (AcP) were simultaneously electrophoresed on a single horizontal starch gel (Dykes and Polesky 1976). The esterase D (EsD), isocitrate dehydrogenase (ICD), and malate dehydrogenase (MDH) phenotypes were identified on a starch gel using a citrate-phosphate buffer system, pH = 5.9, of Karp and Sutton (1967). Locus 1 of the enzyme phosphoglucomutase (PGM) was phenotyped using the original technique of Spencer

et al. (1964). In addition, glyoxalase (GLo) was phenotyped using the methods of Harris and Hopkinson (1976).

Allelic frequencies for the blood group systems are maximum likelihood estimates computed by a modified MAXLIK program of Reed and Schull (1968). These gene frequencies are not corrected for the biological relationships that are observed among the individuals included in the sample.

Analytical Methods

Population structure of the Anabaptist groups is represented by the method described by Harpending and Jenkins (1973). Sample allelic frequencies are converted to a relationship matrix R of dimension ($L \times L$), where L is the number of sample groups. The i,jth element of R is

$$r_{ij} = \frac{1}{k} \sum^{k} \frac{(P_i - P_L)(P_i - P_L)}{P_L(1 - P_L)}, \tag{1}$$

where k is the number of alleles. The diagonal elements of R, r_{ii}, describe the overall deviation of the allelic frequencies of the array (Harpending and Jenkins 1973; Workman et al. 1976). The weighted mean of the diagonal of the relationship matrix (R_{ST}), that is, the mean genetic heterogeneity of all populations, is equivalent to Wright's F_{ST}.

Relative genetic relationships among the Anabaptist groups are graphically represented by a least-squares approximation of the R matrix. Reduced-space eigenvectorial representations provide two-dimensional "genetic maps" of allelic frequency distributions. The eigenvector axes of the maps are scaled by the root of the corresponding eigenvalues and thus equalize the scale of projection (Lalouel 1973).

The relative contributions of systematic versus nonsystematic pressures on the microdifferentiation of subdivided populations are explored through the method of Harpending and Ward (1982). This method is based on the assumption that under uniform systematic pressure heterozygosity should decrease in proportion to the increasing genetic distance from the centroid of the array. Thus a uniform negative slope should result from the regression of heterozygosity on a relative genetic distance. This relationship is represented by a two-dimensional plot whose ordinate is genetic distance (r_{ii}) and whose abscissa is the mean per locus heterozygosity (H_0):

$$\bar{H}_0 = 1 - (\sum_i p_i^2 / I), \tag{2}$$

where P_i is the frequency of the ith allele and I is the number of loci.

To compare the genetic relationship of the various Mennonite congregations with a historically derived dendogram, we performed a cluster analysis on the gene frequency data. We used a BMDP2M computer program (Dixon 1985), which is based on the Euclidean distance between two populations (j and k) and is defined as

$$d_{jk} = \left[\sum_i (x_{ij} - x_{ik})^2\right]^{1/2}, \tag{3}$$

where x_{ij} is the value of the ith variable in the jth population. The algorithm employed by this cluster program uses the distance between centroid clusters as a criterion for amalgamating clusters. Eight blood group systems and 25 alleles were utilized in this cluster analysis.

Results

Tables 2 and 3 summarize the phenotypic counts and gene frequencies for blood groups, serum proteins, and erythrocytic proteins in three Mennonite communities.

Of the 31 blood systems tested by the Minneapolis Blood Bank (29 systems reported in this publication), 19 loci exhibit variant forms at an incidence of 1% or more. We call these "polymorphic" (Table 4). We tested a number of rare familial variants, such as Froese (Fr^a), Scianna 2 (Bu^a), Lw, Gregory, Wright, and Miltenberger, for variation in these Mennonite populations. Although these antigens are rare in the general US population, the presence of some of them, namely Fr^a and Bu^a, has been reported in Mennonite groups (Lewis et al. 1978). We did not detect any Fr^{a+} individuals in this study, although 3 out of 547 individuals of O blood group were Bu^a positive. Although the Lutheran blood group system is usually polymorphic in northern Europe, Lu^a ranges from 1% to 6%; it is monomorphic in the three Mennonite communities.

On the basis of the blood group frequencies, the Goessel and Henderson populations show genetic similarity and both groups differ slightly from the Meridian population. These gene frequency patterns reflect the ethnohistory of these communities, with Goessel and Henderson having separated reproductively in 1874. The Meridian Mennonites are a recent mixture of Pennsylvania Germans, Swiss, and Dutch. Because of the small size of the founding population, the gene frequencies consistently fall outside the ranges observed in Germany, Switzerland, and the Netherlands. For example, in the MNS system both Goessel and Meridian Mennonites differ from the gene frequencies observed in Western

Table 2. **Phenotypes and Gene Frequencies for Blood Group Antigens**

System and Phenotypes	*Goessel*		*Meridian*		*Henderson*	
	Counts	*Gene Frequency*	*Counts*	*Gene Frequency*	*Counts*	*Gene Frequency*
ABO						
A_1	201	$A_1 = 0.207$	25	$A_1 = 0.170$	195	$A_1 = 0.215$
A_2	74	$A_2 = 0.089$	3	$A_2 = 0.022$	46	$A_2 = 0.064$
B	83	B = 0.104	9	B = 0.061	78	B = 0.096
A_1B	32	O = 0.600	1	O = 0.747	14	O = 0.625
A_2B	7		0		8	
O	219		48		208	
MNSs						
MS	21		8		29	
MSs	75		11		82	
MSs	63	MS = 0.209	3	MS = 0.351	37	MS = 0.236
MNS	15	Ms = 0.316	1	Ms = 0.23	19	Ms = 0.270
MNSs	139	NS = 0.031	34	NS = 0.032	119	NS = 0.068
MNs	175	Ns = 0.444	19	Ns = 0.404	120	Ns = 0.426
NS	0		1		3	
NSs	10		1		33	
Ns	116		8		104	
Rhesus						
CDe	98		15		103	
CcDEe	78	CDe = 0.403	14	CDe = 0.372	69	CDe = 0.428
CcDe	223	cDE = 0.151	20	cDE = 0.238	205	cDE = 0.145
cDE	16	cDe = 0.019	0	cDe = 0.000	10	cDe = 0.054
cDEe	79	Cde = 0.002	5	Cde = 0.000	68	Cde = 0.012
cDe	11	cdE = 0.002	17	cdE = 0.000	21	cdE = 0.002
Ccde	1	cde = 0.423	0	cde = 0.390	4	cde = 0.359
cdEe	1		0		1	
cde	109		0		66	
Duffy						
Fy a+b−	115	$Fy^a = 0.456$	23	$Fy^a = 0.500$	131	$Fy^a = 0.499$
a+b+	311	$Fy^b = 0.544$	39	$Fy^b = 0.500$	284	$Fy^b = 0.501$
a−b+	150		23		135	
Kidd						
Jka+b−	166	$Jk^a = 0.521$	20	$Jk^a = 0.477$	65	$Jk^a = 0.433$
a+b+	276	$Jk^b = 0.479$	42	$Jk^b = 0.523$	337	$Jk^b = 0.567$
a−b+	134		24		147	
P_1						
+	460	$P_1 = 0.495$	70	$P_1 = 0.555$	439	$P_1 = 0.554$
−	156	$P_2 = 0.505$	16	$P_2 = 0.445$	110	$P_2 = 0.446$
Kell						
KK	4	K = 0.066	1	K = 0.076	4	K = 0.093
Kk	71	k = 0.934	11	k = 0.924	94	k = 0.907
kk	525		74		449	
Kpa+b−	2	$Kp^a = 0.042$	0	$Kp^a = 0.020$	0	$Kp^a = 0.065$
a+b+	11	$Kp^b = 0.958$	2	$Kp^b = 0.980$	27	$Kp^b = 0.935$
a−b+	197		47		197	

(Table 2 cont.)

System and Phenotypes	Goessel Counts	Goessel Gene Frequency	Meridian Counts	Meridian Gene Frequency	Henderson Counts	Henderson Gene Frequency
Lewis						
Le a+b−	73	Le(a+b−) = 0.126	11	Le(a+b−) = 0.129	102	Le(a+b−) = 0.187
a+b+	0	Le(a−b+) = 0.766	0	Le(a−b+) = 0.742	0	Le(a−b+) = 0.754
a−b+	446	Le(a−b−) = 0.108	63	Le(a−b−) = 0.129	412	Le(a−b−) = 0.059
a−b−	63		11		32	

Europe, whereas Henderson Mennonites conform more closely to the observed European pattern (Mourant et al. 1976). These data suggest that the founding Mennonites do not represent a random sample of either Germany or the Netherlands but constitute a unique genetic amalgam.

There is little comparative information on the blood genetics of Mennonite populations. Brown et al. (1974) described the blood group frequencies of 51 Mennonite settlers who were encountered by the researchers in the Gran Chaco region of Paraguay. This Chaco colony emigrated from Canada in 1926. There is considerable genetic difference between this small Paraguayan sample and the Mennonites of Kansas and Nebraska. Specifically, the Chaco Mennonites exhibit high frequencies of O blood group (79%), Ms (42%), and CDe (59%) and low frequencies of B (0), MS (16%), and cde (23%). The differences between these Mennonite groups are probably a result of a combination of factors, namely, the genetic composition of the founders and the small size of the sample from Paraguay.

Most of the serum protein gene frequencies observed in Mennonite populations fall within or just outside the published European gene frequency ranges. For example, in European populations the group-specific component (also termed the vitamin D binding component or DBP) varies for the Gc^2 allele between 20% and 30% (Constans et al. 1985). The frequency of this allele in the three Mennonite communities is slightly elevated and beyond the published European ranges. Although the Gc^{1S} allele has a frequency of 55–60% in Europe, the Mennonite populations range between 54% and 57%. A sample of Mennonites (combined Goessel and Meridian communities) has been described for Gc and PGM isoelectric focusing subtypes (Dykes et al. 1983). As expected, these reported frequencies are intermediate between values for each of the two communities.

One interesting finding in one of the Mennonite communities is the presence of the Gc^{1A1} gene. This gene is rarely observed in European populations unless there is a history of African or Australian aborigine admixture. Although Gc^{1A1} was first detected in Australian aborigines and was initially named Gc^{Ab}, its highest incidence is in central Africa

Table 3. **Phenotypes and Gene Frequencies for Serum and Red Blood Cell Proteins**

Systems and Phenotypes	*Goessel*		*Meridian*		*Henderson*	
	Counts	*Gene Frequency*	*Counts*	*Gene Frequency*	*Counts*	*Gene Frequency*
Haptoglobin						
Hp 1-1	80	$Hp^1 = 0.353$	3	$Hp^1 = 0.235$	58	$Hp^1 = 0.345$
2-1	278	$Hp^2 = 0.642$	34	$Hp^2 = 0.765$	260	$Hp^2 = 0.650$
2-2	259	$Hp^{Carl} = 0.005$	48	$Hp^{Carl} = 0.000$	224	$Hp^{Carl} = 0.005$
2-Carl	6		0		4	
Ceruloplasmin						
CpAB	4	$Cp^A = 0.003$	0	$Cp^A = 0\ 001$	10	$Cp^A = 0.009$
BB	585	$Cp^B = 0.997$	86	$Cp^B = 1.000$	536	$Cp^B = 0.990$
BC	0	$Cp^C = 0.000$	0	$Cp^C = 0.000$	1	$Cp^C = 0.001$
Bf						
F	22		6		36	
FS	153	F = 0.167	22	F = 0.224	188	F = 0.247
S	391	S = 0.813	40	S = 0.724	274	S = 0.705
FF1	1	SO.7 = 0.006	0	SO.7 = 0.019	4	SO.7 = 0.024
FSO.7	2	$F_1 = 0.014$	0	$F_1 = 0.033$	7	$F_1 = 0.024$
F_1S	16		5		21	
SSO.7	6		3		16	
SO.7	0		0		1	
SO.7F1	0		0		1	
Gc						
1-1	284	$Gc^1 = 0.678$	38	$Gc^1 = 0.698$	232	$Gc^1 = 0.665$
2-1	245	$Gc^2 = 0.322$	32	$Gc^2 = 0.302$	265	$Gc^2 = 0.334$
2-2	70	$Gc^{Ab} = 0.000$	6	$Gc^{Ab} = 0.00$	50	$Gc^{Ab} = 0.001$
2-AB	0		0		1	
Gc: (isoelectric focus)						
1S	176		28		40	
1S1F	67	1S = 0.565	9	1S = 0.572	196	1S = 0.535
1S2	177	1F = 0.116	30	1F = 0.121	211	1F = 0.130
1F	12	2 = 0.319	3	2 = 0.307	4	2 = 0.334
1F2	43	1A1 = 0.00 0	5	1A1 = 0.000	47	1A1 = 0.001
2	58		8		49	
1A1(Ab)	0		0		1	
ADA						
1-1	570	$ADA^1 = 0.968$	77	$ADA^1 = 0.895$	498	$ADA^1 = 0.958$
2-1	39	$ADA^2 = 0.032$	9	$ADA^2 = 0.105$	44	$ADA^2 = 0.042$
2-2	0		0		1	
Adenylate Kinase						
AK1-1	557	$AK^1 = 0.954$	86	$AK^1 = 1.000$	512	$AK^1 = 0.971$
2-2	57	$AK^2 = 0.046$	0	$AK^2 = 0.000$	32	$AK^2 = 0.029$

(Table 3 cont.)

Systems and Phenotypes	*Goessel*		*Meridian*		*Henderson*	
	Counts	*Gene Frequency*	*Counts*	*Gene Frequency*	*Counts*	*Gene Frequency*
6-PGD						
AA	605	$Pd^A = 0.993$	86	$Pd^A = 1.000$	539	$Pd^A = 0.995$
AC	9	$Pd^C = 0.007$	0	$Pd^C = 0.000$	1	$Pd^B = 0.001$
AB	0		0		5	$Pd^C = 0.004$
AcP						
A	94		14		40	
AB	254	$acP^A = 0.377$	40	$acP^A = 0.401$	214	A = 0.291
B	214	$acP^B = 0.594$	31	$acP^B = 0.593$	213	B = 0.639
BC	25	$acP^C = 0.029$	0	$acP^C = 0.006$	53	C = 0.070
AC	7		1		21	
C	1		0		1	
Esterase D						
1–1	509	$EsD^1 = 0.911$	58	$EsD^1 = 0.808$	433	$EsD^1 = 0.901$
2–1	93	$EsD^2 = 0.089$	23	$EsD^2 = 0.192$	96	$EsD^2 = 0.099$
2–2	8		5		5	
PGM_1						
1–1	428	$PGM_1{}^1 = 0.848$	61	$PGM_1{}^1 = 0.843$		$PGM^1 = 0.815$
2–1	156	$PGM_1{}^2 = 0.152$	23	$PGM_1{}^2 = 0.157$		$PGM^2 = 0.184$
2–2	13	$PGM^r = 0.000$	2	$PGM^r = 0.000$		$PGM^r = 0.001+$
1–r	0				1	
PGM_i						
1+1–	315	$1^+ = 0.764$	64	$1^+ = 0.807$	246	$1^+ = 0.704$
1–	76	$1^- = 0.085$	3	$1^- = 0.026$	85	$1^- = 0.112$
1+2+	1	$2^+ = 0.141$	0	$2^+ = 0.146$	5	$2^+ = 0.173$
1+2–	136	$2^- = 0.010$	21	$2^- = 0.021$	153	$2^- = 0.010$
1–2+	1	1r = 0.001	3		8	1r = 0.001
1–2–	11		2		14	
2+	1		0		1	
2+2–	9		2		13	
1r	1		1		1	
GLO						
1–1	25	$GLO^1 = 0.456$		NT	21	$GLO^1 = 0.353$
2–1	80	$GLO^2 = 0.564$			64	$GLO^2 = 0.647$
2–2	44				65	
ICD						
1–1	610	$ICD^1 = 1.000$		NT	527	$ICD^1 = 0.998$
2–1	0	$ICD^2 = 0.000$			2	$ICD^2 = 0.002$
MDH						
1–1	610	$MDH^1 = 1.000$		NT	529	$MDH^1 = 1.000$

Table 4. **Summary of Polymorphic and Monomorphic Loci among at Least One of Three Populations Reported in This Study**

Polymorphic Loci	*Monomorphic Loci*
ABO	Lutheran
Rhesus	Ceruloplasmin
MNS	6-Phosphogluconate dehydrogenase
Kidd	Isocitrate dehydrogenase
Duffy	Malate dehydrogenase
P	Gregory
Kell	Froese (Fr^a)
Lewis	Scianna 2(Bu^a)
Gm*	Lw
Glyoxalase I	Vel
Km*	Wright
Haptoglobin	Miltenberger
Properdin Factor	
Group specific component	
Adenosine deaminase	
Adenylate kinase	
Acid phosphatase	
Esterase D	
Phosphoglucomutase	

* Reported in another publication.

(Constans et al. 1985). Considering the presence of an African marker PGM_1^r gene in the same person with the Gc^{1A1}, it is highly likely that both these genes were introduced through American black gene flow into the population.

The frequency of the Hp^1 allele in the three Mennonite communities is lower than its incidence in Germany, Switzerland, or the Netherlands. In particular, Hp^1 occurs in Meridian at 23.5% versus the 36–40% in those regions of Europe from which the Mennonites originated.

A rare haptoglobin variant, Carlberg, was detected in two of the Mennonite communities. Hp Carlberg (Hp Ca) was originally described by Galatius-Jensen (1958). This phenotype resembles a mixture of Hp 2–2 and Hp 2–1 in variable proportions, prompting Sutton (1965) to suggest that this variant may be the result of genetic mosaicism. More recently, the Hp Ca phenotype has been explained on the basis of a possible mutation of the $Hp^1\alpha$ chain followed by a decrease in the synthesis of the polypeptide (Bowman and Kurosky 1982).

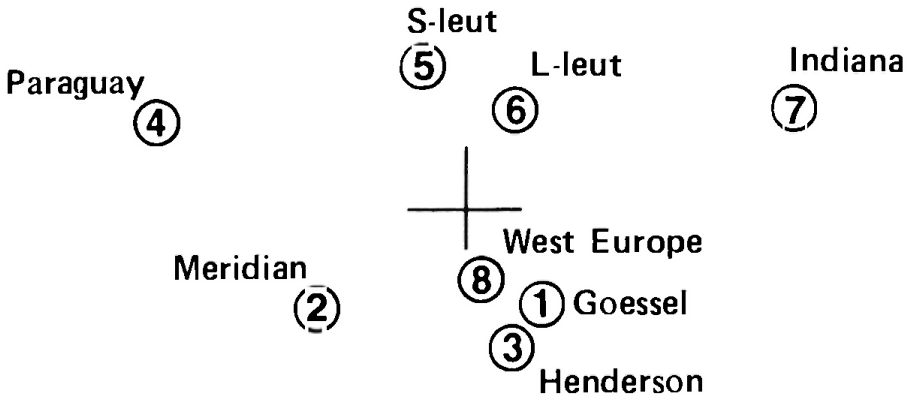

Figure 1. Least-squares reduction genetic map of the eight Anabaptist populations based on allelic frequencies for ten alleles and three loci. Three Kansas and Nebraska Mennonite communities are represented by Meridian, Goessel, and Henderson. Gene frequencies for the other Anabaptist groups were obtained from Juberg et al. (1971), Brown et al. (1974), and Steinberg et al. (1967).

Population Structure

Figure 1 shows the population structure in the reduced-space genetic map of the three Kansas and Nebraska Mennonite communities compared to other Anabaptist groups and Western Europe (represented by mean gene frequencies from the regions of origin of the Mennonites). This plot is based on ten alleles and three loci. The number of loci used in this analysis was necessitated by the availability of data on other Anabaptist groups. Samples are arrayed along the first two scaled eigenvectors, which together account for 71% of the total variation in the sample (eigenvector 1, 51%; eigenvector 2, 20%). The addition of the third eigenvector in a pseudo-three-dimensional representation accounts for 83% of the total variation. The genetic map in Figure 1 indicates little genetic divergence among the various Anabaptist groups, with the cluster populations in close proximity to the centroid of the distribution. Of the three Mennonite communities studied in Kansas and Nebraska, the Meridian population differs from the other two groups. This genetic difference is a result of their ethnohistory and genetic foundation, with Goessel and Henderson being a single community until 1874. Meridian had few founders. Goessel and Henderson individuals, who are General Conference Mennonites (the most mainstream of the Mennonites), have the closest genetic affinity to the composite Western Europe population. The Paraguay Mennonites differ from the other groups probably because of their small sample size ($N = 51$) and may represent a fission of the larger Canadian gene pool along familial lines.

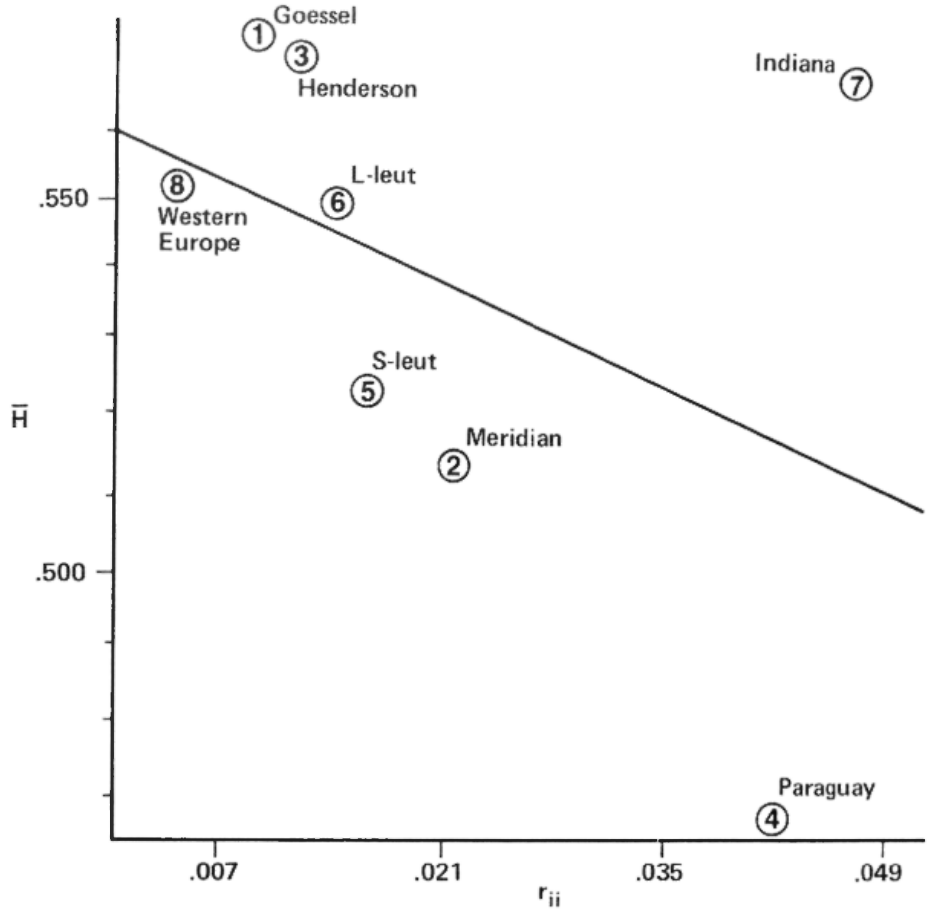

Figure 2. Plot of mean per locus heterozygosity H (H_0 is used interchangeably) against distance from the centroid r_{ii} of the relationship matrix for eight Anabaptist communities. The R matrix is based on ten blood group alleles and three loci.

Figure 2 indicates the relationship between mean per locus heterozygosity (H_0) and the relative distance from the centroid of the allelic distribution (r_{ii}) for the seven Anabaptist groups and a composite Western Europe group. This comparison reflects the ethnohistory and origins of the Anabaptist groups. The Paraguayan Mennonites exhibit low heterozygosity and high r_{ii}, suggesting that this population is experiencing the action of nonsystematic pressures because of its geographic reproductive isolation and small size. By contrast, the Indiana Amish have high heterozygosity and high r_{ii} and, like the Paraguay Mennonites, are located far from expectation, as demonstrated by their distance from the theoretical regression line. This unusually high level of r_{ii} and H_0 among the Amish is probably the result of a highly heterogeneous founding population acted on by nonsystematic pressures. However, the Goessel and Henderson populations exhibit relatively high levels of heterozygosity and low r_{ii}, suggesting elevated migration rates and exogamy.

Wright's F_{ST} statistics have been widely used to measure the genetic microdifferentiation of subdivided populations. In comparison with other studies of F_{ST}, the Anabaptist populations exhibit a low F_{ST} level of 0.002. This value indicates that these Anabaptist groups can be considered genetically homogeneous (based on 10 alleles) and have experienced little genetic differentiation. In contrast, subdivided circumpolar populations attain F_{ST} values of 0.12, and the major geographic entities of *Homo sapiens* exhibit values up to 0.15 (Crawford and Enciso 1982).

Figure 3 contains a least-squares reduction genetic map of the three Mennonite communities subdivided by congregations. We reduced 44 alleles and 15 genetic loci into 2 eigenvectors. The first two scaled eigenvectors account for almost 62% of the gene frequency variance in the array. The first eigenvector (e_1) has almost twice the discriminating power and accounts for 41.5% of the variance, versus 21.5% for e_2. The first scaled eigenvector separates Kansan from Nebraskan Mennonites. The Goessel community is subdivided into three congregations, namely, the Alexanderwohl, Tabor, and Goessel churches. Goessel and Tabor split from the Alexanderwohl church in 1909 and 1920, respectively. In Figure 4 the Alexanderwohl church is the most proximal to the centroid of the array, whereas the Goessel and Tabor churches have been separated from their parental groups by the frequencies of the P^- and esterase D^1 alleles.

The bases for the distribution of the groups along the second axis ($e_2^{1/2}$) shown in Figure 4 is less obvious. The reduced-space representation of the alleles used to compute the genetic map shows that Gc^{1F} and cde separate the populations along the second axis.

Figure 5 is a plot of the mean per locus heterozygosity and r_{ii} for the various congregations. This mean of H and r_{ii} is based on 44 alleles instead of the 10 shown in Figure 2. Alexanderwohl, Meridian, and, to a lesser extent, Goessel and Tabor remain closest to the theoretical regression line (the predicted relationship between the two variables). The Henderson Mennonite congregation exhibits the highest heterozygosity level, whereas the Bethesda and the Evangelical congregations have the lowest H. In this plot the heterozygosity levels of the congregations are reduced to some extent because of the addition of a large number of diallelic loci.

Major differences are observed in a comparison of two Mennonite congregation dendograms, one a historical reconstruction of population fission and fusion (Figure 6) and the other based on the cluster analysis of gene frequencies (Figure 7). Genetically, the Tabor population is separate from the other Mennonite communities, even though historically it split off only two generations ago. The Evangelical church in Hender-

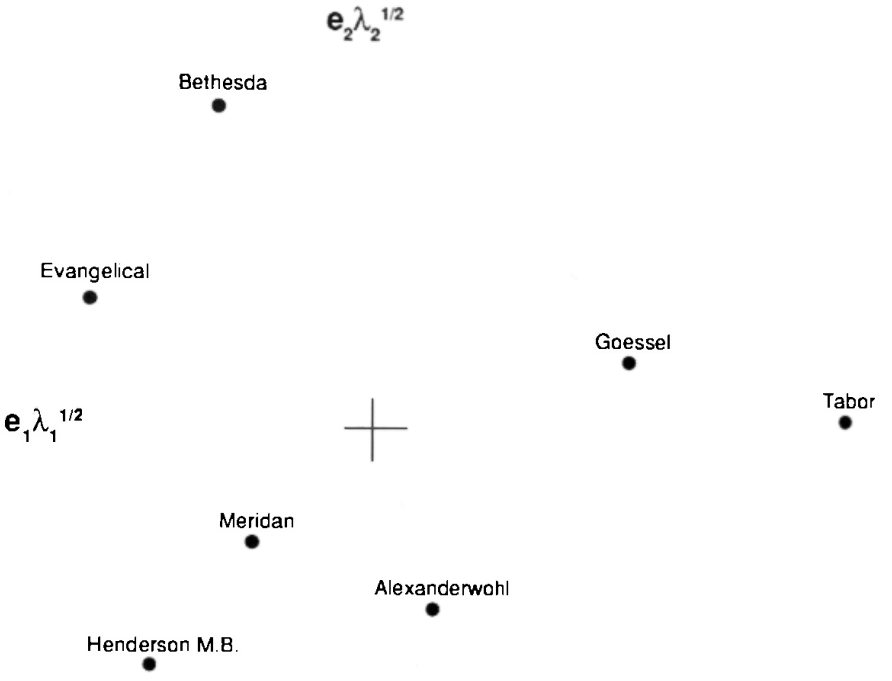

Figure 3. Least-squares reduction genetic map ot the three Mennonite communities subdivided into seven congregations. Frequencies from 44 alleles and 15 genetic loci were used to construct the R matrix.

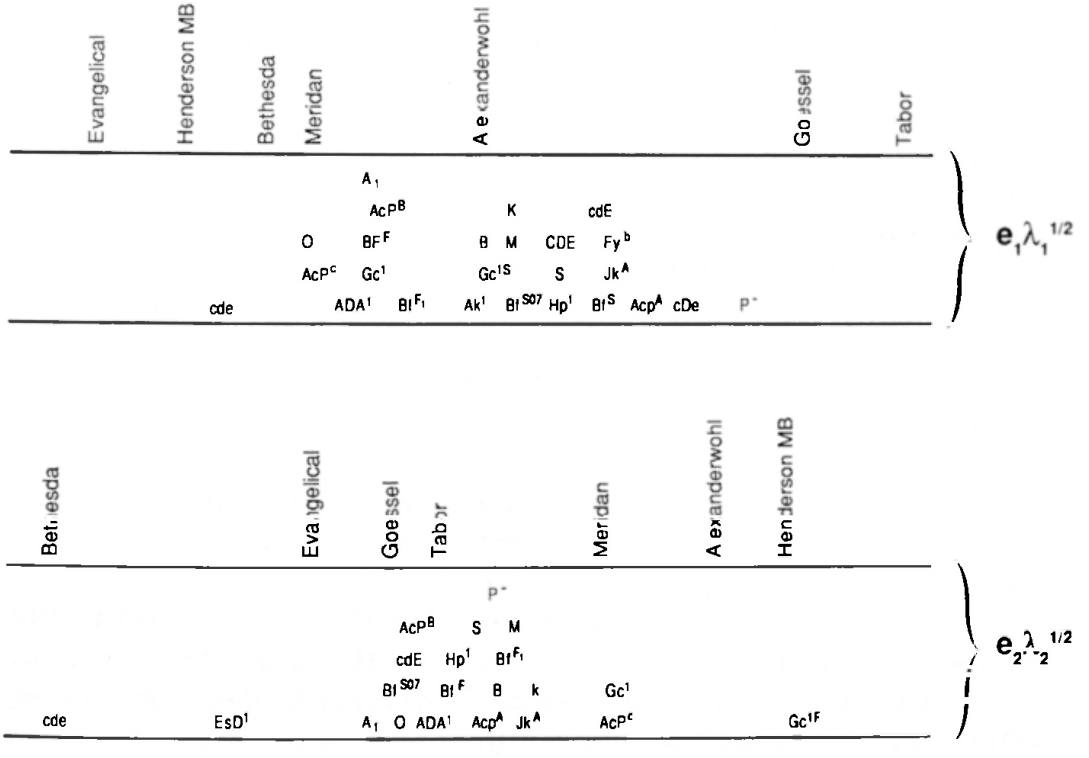

Figure 4. Dispersion of the seven Mennonite congregations and alleles along eigenvectors associated with the two largest eigenvectors of R and A matrices.

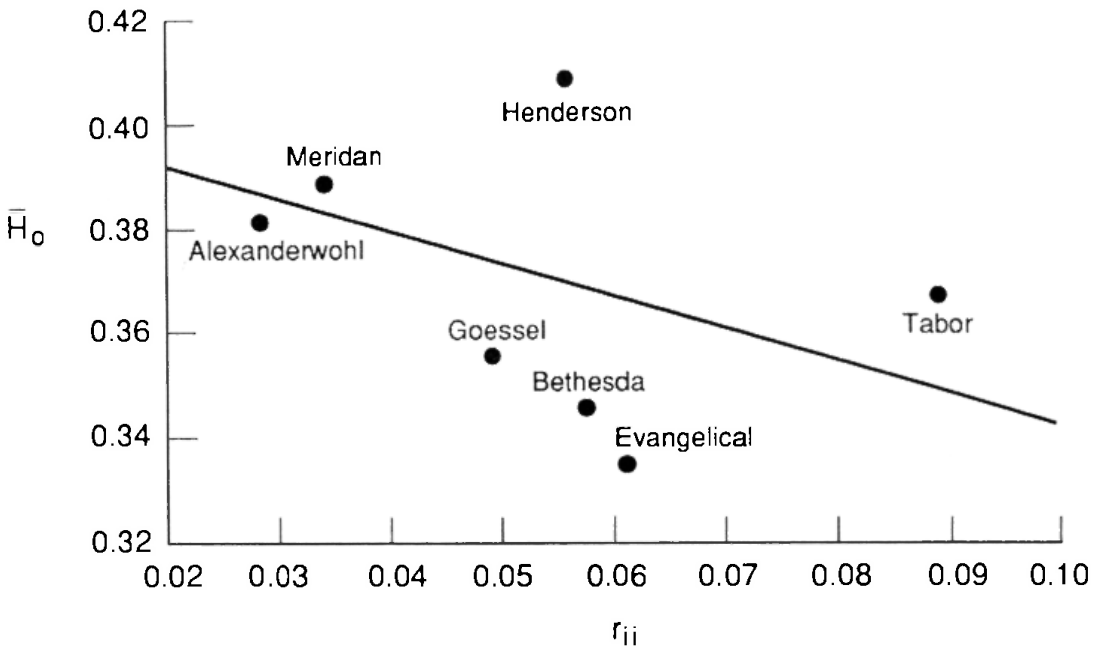

Figure 5. Plot of mean per locus heterozygosity (H_0) against distance from the centroid r_{ii} of the R matrix for seven Mennonite congregations. Frequencies from 44 alleles and 15 loci were used.

son, Nebraska, and the Goessel congregation came off the genetic tree next, even though historically their origins must be traced to Russia. These findings support the hypothesis that the fission of the Mennonite gene pools occurred along familial lines and does not represent a random subset of the population. The Bethesda and Alexanderwohl communities, both remnants of the original division of the Alexanderwohl congregation in 1874, cluster together genetically. Yet, despite their heterogeneous European origins, both Meridian and the Henderson Mennonite congregations show closer genetic affinities than do the offshoot congregations.

Discussion

The population history of the Mennonites can be characterized in terms of a fission-fusion model, which is schematically represented in Figure 6. The founders of the various Mennonite groups came from the Netherlands, Switzerland, northern Germany, Moravia, Alsace, and Tirol. The earliest groups were founded by a small number of individuals coming from different regions of Europe.

The Przechowka Church in West Prussia, which eventually gave rise to the Alexanderwohl congregation, was founded by 15 refugee couples from the Netherlands and Switzerland. This congregation, through high

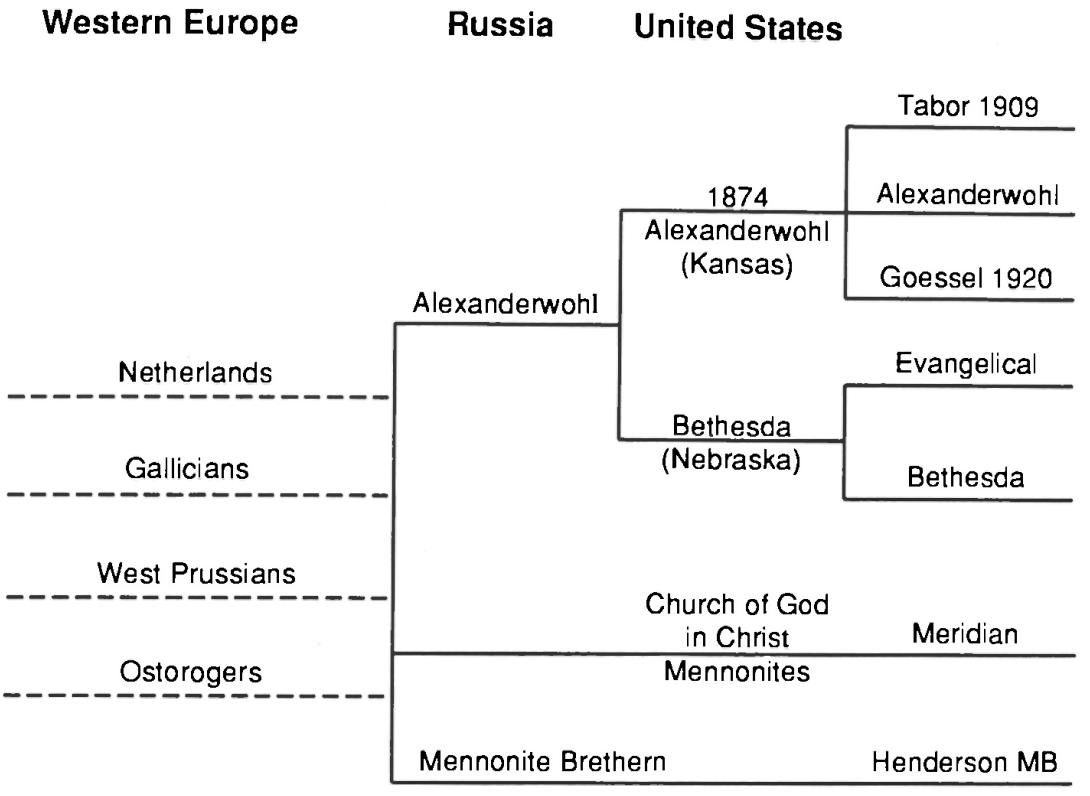

Figure 6. The ethnohistory of the seven Mennonite congregations summarized by a dendogram. The dotted lines indicate that various combinations of Dutch and German populations contributed to the formation of the Nebraskan and Kansan Mennonites. This dendogram is based on an unpublished flow diagram constructed by John Janzen.

fertility and the addition of other migrants from Prussia and Moravia, increased in number and in 1821 relocated in the Ukraine. The community was renamed Alexanderwohl, and when the Russian government revoked the military exemptions for Mennonites, the entire community emigrated to the United States in 1874. On arrival, Alexanderwohl underwent the first fission, with one group settling in Nebraska and founding the town of Henderson and the second group settling in Kansas. This Kansas subdivision of the Alexanderwohl community settled in two communities, Hoffnungsau and Goessel. Shortly after the turn of the twentieth century, in 1909, Tabor split from the Alexanderwohl congregation (located in the town of Goessel) and established the Goessel church in 1920. In addition, the Nebraska Mennonite Bethesda congregation also underwent a fission, with the separation of the Evangelical Mennonites.

A similar fission and fusion of populations has been described by Olsen (1987) for the Hutterites of North America. Olsen characterized the formation of Hutterite colonies on the basis of historic and demographic

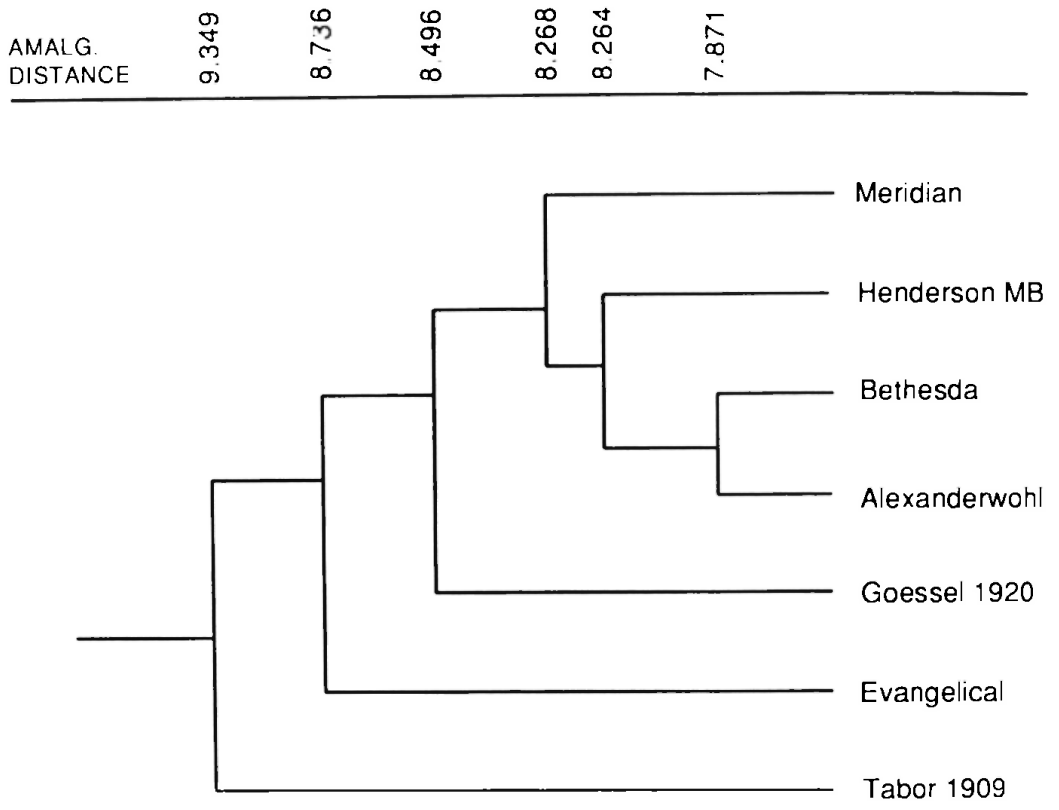

Figure 7. A dendogram based on the cluster analysis of the frequencies of 8 blood group systems and 25 alleles. This clustering method is based on the distance between centroids as a criterion for amalgamation.

evidence for 1878–1970 and likened their structure in many respects to the Yanomamo of Venezuela. Although the Mennonites do not explicitly follow a policy of splitting off daughter colonies because of high fertility, this population subdivision does occur de facto. Most of the General Conference Mennonite splits appear to be due to personality conflicts, doctrinal disagreements, or both. Although economic considerations are not voiced, they should not be ignored as one of the motivating factors for population fission.

This fission-fusion process is reflected in the genetic structure of the Mennonites. For example, the contemporary Alexanderwohl congregation remains proximal to the center of the allelic array, whereas its offshoots, Tabor and Goessel, show marked genetic difference. Given the short time since the fission of these populations (2–3 generations), it is unlikely that the Tabor and Goessel Mennonites would experience this magnitude of genetic microdifferentiation. The two more likely explanations for this genetic uniqueness are population sampling and subdivision by families. Sampling may play a major role, because the Tabor and

Goessel samples are the smallest of all the congregations studied. It is also likely that the fission process involving Tabor, Goessel, and Alexanderwohl took place along familial lines, producing a nonrandom division of the gene pool. In support of this, Rogers (1984) demonstrated through the use of surname analysis that the initial fissioning of the Mennonite immigrant group was nonrandom. There were 63 surnames among the 191 families that immigrated; however, only 3 of these surnames (5%) are found in all three settlements.

The Meridian community (Church of God in Christ Menno) is a heterogeneous population founded in 1859. Although this group is small and highly conservative with regard to endogamy, it has experienced a low level of inbreeding (Sirijaraya 1983). As a result of the genetic heterogeneity of its founders, the Meridian congregation remains close to the regression line in the r_{ii} versus $\bar{H}_0$ comparison. Meridian displays a relatively high heterozygosity but a low distance from the centroid of the allelic frequency distribution (see Figure 5).

The Kansas Mennonite populations also experienced various degrees of reproductive isolation and inbreeding throughout their history. According to Rogers (1984), their inbreeding coefficient (F) varied from 2% in 1800–1819 to 0.5% in 1860–1874. At present, the General Conference Mennonite communities of Kansas and Nebraska cannot be characterized as genetic isolates because a high proportion of marriages are with non-Mennonites. During the last 20–25 years, approximately 46% of all the marraiges in Hoffnungsau, Kansas, were contracted with non-Mennonites (Kay 1978). However, from 1874–1915 the community was 99% endogamous. Since World War I the reproductive isolation has rapidly broken down with the congregation members becoming more integrated into mainstream American life. Before 1936 more than 95% of the marriages were Mennonite endogamous. Marital partners were Mennonite but from a different village or town. By 1957 Mennonite endogamy occurred in 72.3% of the marriages and 27.7% were exogamous (Kay 1978).

The blood genetics support the conclusion that the General Conference Mennonites described in this study are no longer members of genetic isolates and are culturally approaching the mainstream farming communities. The high levels of heterozygosity reflect considerable systematic pressure in the form of migration. The mean per locus heterozygosity varies from 33% to 41%, based on 29 blood systems. In addition, the presence of several African marker genes suggests gene flow from the American black gene pool into the Mennonite groups.

Contemporary Kansas and Nebraska General Conference Mennonite genetic structure can best be explained on the basis of episodes of

fission and fusion, followed by a rapid breakdown of reproductive isolation. Thus these Mennonite populations can no longer be characterized as highly inbred genetic isolates.

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