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# Pingelap and Mokil Atolls: Historical Genetics

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The contemporary genetic structure of a population reflects hybridization, drift, and selection in previous generations. Inferences of greater or less precision about these phenomena can be made from pedigrees, gene frequencies, historical documents, and legends. Such studies, having as their end the interpretation of contemporary genotype frequencies in terms of past events, constitute historical genetics.

In large populations the deterministic processes of hybridization and selection are ascendant, whereas small numbers of founders and subsequent genetic drift are important in isolates, to which the present paper is restricted. One of the objectives of historical genetics is to determine the contribution of principal founders to the current gene pool. If we define as founders all members of the population in certain previous generations whose parents came from other groups or are unknown, and if genes of a particular allelic class are derived from a small number of founders, then we may be able to identify these founders.

Pingelap and Mokil Atolls in the Eastern Caroline Islands are typical isolates, in which genealogies for the last two centuries are nearly complete [1, 2]. Genetic characters available for study include blood groups, immunoglobulins, red cell isozymes, serum proteins, colorblindness, and achromatopsia. Leprosy will be considered as an additional character, since Pingelap is a focus of leprosy in Micronesia, and leprosy may have a genetic component on which historical genetics can throw some light [3].

## THE FOUNDER EFFECT

On both atolls the present population is descended from a handful of survivors of the famine which followed Typhoon Lengkieki around 1775 [1]. Estimates of population size since 1853 indicate that Pingelapese outnumber Mokilese in the ratio of 2:1 (tables 1 and 2), yet the effective population size determined from pedigrees is substantially the same for the two atolls, 87 for Pingelap and 82 for Mokil [2]. Can this paradox be resolved by the methods of historical genetics?

The census sizes in tables 1 and 2 can be fitted by an exponential regression. Since the mean survivor of Typhoon Lengkieki was estimated to have been born

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TABLE 1

CENSUS SIZE FOR MOKILESE POPULATION (INCLUDING PONAPE)

| Reference | Year of Census | Census Size |
|---|---|---|
| Weckler [4] | 1775 | 30 |
| Hammet [5] | 1853 | 90 |
| Hermann [6] | 1901 | 170 |
| Bentzen [7] | 1948 | 600 |
| *Ponape Almanac* [8] | 1967 | 900 |

TABLE 2

CENSUS SIZE FOR PINGELAPESE POPULATION (INCLUDING PONAPE)

| Reference | Year of Census | Census Size |
|---|---|---|
| Dison Aia, informant | 1775 | 30 |
| Hammet [5] (about 150 men and boys) | 1853 | 450 |
| Agassiz [9] | 1899 | 1,000 |
| Bascom [10] | 1946 | 900 |
| *Ponape Almanac* [8] | 1967 | 1,600 |

in 1761 [2], we set $t =$ year $- 1761$. Then the census sizes are $N = 40e^{.019t}$ for Pingelap and $N = 20e^{.018t}$ for Mokil, suggesting that Pingelap (with an area of 0.676 sq miles) had more survivors of Typhoon Lengkieki than Mokil (with an area of 0.478 sq miles), and that subsequent increase has been at the rate of nearly 2% per year. If population size follows a law $N_0 f(t)$, the harmonic mean as $t$ goes from 0 to $T$ is $N = TN_0/\int_0^T [1/f(t)]\, dt$. For $f(t) = e^{bt}$, $N = TN_0 b/(1 - e^{-bT})$. With $T = 200$ years, the values of $N$ for Pingelap and Mokil are 155 and 74, respectively. The effective size of the two atolls $(87 + 82)$ has been 73% of the census size $(155 + 74)$. This is an unusually large fraction. Children in utero, infants, and the elderly were presumably the first victims of famine, during which both induced abortion and infanticide may have been practiced in premissionary times. Even today unwanted pregnancies are said to be sometimes aborted by trauma. Thus the proportion of adults of reproductive age may increase, and both natural and artificial selection against large families may be more intense in times of famine. Immigration increases the effective size of an isolate [11]. The high ratio of effective to census size following Typhoon Lengkieki is not surprising, but the subequality of the effective sizes of the two atolls requires explanation.

Consider the founder sibships which contained among parents or sibs one or more survivors of Typhoon Lengkieki, and let $\phi_{it}$ denote the mean kinship of the

$i$th founder sibship to the $t$th generation (i.e., the probability that a given gene among the four alleles in the parents of the $i$th founder sibship be identical by descent with a random allele in the $t$th generation). There were 22 such sibships on Pingelap and 10 on Mokil, the corresponding numbers of individuals (44 and 20) being close to the above estimates of census size. Table 3 shows the largest

TABLE 3

CONTRIBUTIONS OF PRINCIPAL FOUNDER SIBSHIPS TO GENE POOL OF $t$TH GENERATION, $4\phi_{it}$

| POPULATION AND FOUNDER SIBSHIP | GENERATION $t$ | | |
|---|---|---|---|
| | 3 | 5 | 7 |
| Pingelap: | | | |
| 1067 | .188 | .185 | .145 |
| 0757 | .090 | .092 | .069 |
| 1121 | .040 | .070 | .079 |
| Mokil: | | | |
| 0001 | .104 | .103 | .131 |
| 0003 | .073 | .063 | .072 |
| 0004 | .063 | .052 | .070 |

values of $4\phi_{it}$, which is the expected contribution of the $i$th founder sibship to the gene pool of the $t$th generation. By this criterion Pingelap had as large a founder effect as Mokil, the principal founder sibship contributing 15% of the gene pool.

Table 4 shows the distribution of sibship size among nonfounders in generations 1 and 2, excluding infertile matings which were not ascertained. By fitting a truncated negative binomial distribution to these data, we can estimate the mean ($K$) and variance ($\sigma^2$) of the complete distribution, and from these an index of variability [12], which was greater for Pingelap. The family of the *nanmwarki* tended to be large, either by polygamy or an unusually fertile spouse. The principal founder sibship 1067 contained two polygamous chiefs, who left several of the survivors of Typhoon Lengkieki. Their successor, Mwanenised, had 10 children. This achievement so impressed his contemporaries that he was given the posthumous name of "backbone of Pingelap." It was then the custom after the death of an important man for an elder to recount a dream in which the dead man appeared and revealed his new name. Mwanenised's ghost explained that his name did not mean strength or wisdom, but that he had made Sou Serawi (his wife's clan) great on Pingelap. This generation of rapid population growth established sibship 1067 as the principal founder sibship, with consequences for the Pingelapese which we shall now examine.

## ACHROMATOPSIA

About 5% of the Pingelapese and a smaller proportion of Mokilese are affected with congenital achromatopsia, accompanied by high myopia [13]. Inheritance is

TABLE 4

SIBSHIP SIZE IN GENERATIONS 1 AND 2, EXCLUDING FOUNDER SIBSHIPS

| POPULATION | NO. NAMED CHILDREN $s$ | | | | | | | | | | TOTAL |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 10 | 12 | 14 | |
| Pingelap | 16 | 13 | 7 | 7 | 6 | 13 | 5 | 4 | 2 | 2 | 75 |
| Mokil | 1 | 0 | 4 | 8 | 5 | 1 | 0 | 0 | 0 | 0 | 19 |

| | FITTING NEGATIVE BINOMIAL DISTRIBUTION | | |
|---|---|---|---|
| | Mean $K$ | Variance $\sigma^2$ | $\frac{\sigma^2}{K} + K - 1$ |
| Pingelap | 3.75 | 12.01 | 5.95 |
| Mokil | 3.91 | 3.91 | 3.91 |

$$f(s) = \binom{z}{s} m^s(1-m)^{z-s}/[1-(1-m)^z]$$

$$K = mz$$

$$\sigma^2 = mz(1-m)$$

$$\frac{\sigma^2}{K} + K - 1 = m(z-1)$$

NOTE.—Each parent entered once, with his half-sibships pooled.

as an autosomal recessive. Inspection of the complex genealogies suggests that sibship 1067 was the carrier founder for all cases on both Pingelap and Mokil. The latter evidence is given in figure 1 as a sibship pedigree, which presents the relevant facts more clearly and preserves anonymity better than the conventional diagram.

To detect carrier founders we scored each parent 8, 4, 2, or 0 for affected, child or parent of an affected, child or parent of a carrier, or not so. The sum of the two parental scores was regressed stepwise on the coefficients of kinship with the 37 Pingelapese founder sibships in generations 1–3. Five sibships had significant coefficients, all positive: 1067, 1121, 0578, 0733, and 0719. When the discriminant was repeated with all nonzero values of kinship replaced by 1, a different set of five sibships was significant: 1067, 0765, 0988, 0578, and 0922. Affinal relationships among these founders suggest that all the genes for achromatopsia may derive from sibship 1067, which is ancestral to every known carrier [14]. Apparently the significance of other founder sibships is due to segregation among descendant lines.

Two brothers in sibship 1067 left survivors of Typhoon Lengkieki. Semenuhwe had one child, while Mwahuele left seven children by three wives, one of whom married a cousin, the daughter of Semenuhwe. Thus the most economical hypothesis is that Mwahuele was the only carrier of achromatopsia in his generation. Since the gene is recessive, the selection pressure against it in low frequency is

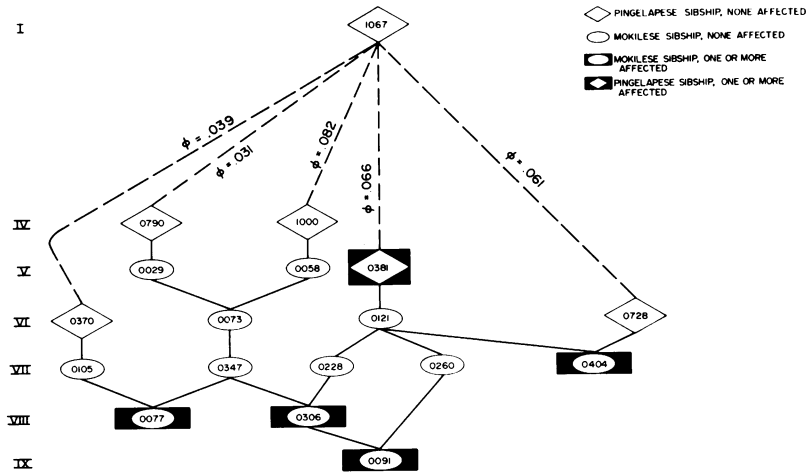


FIG. 1.—Achromatopsia in Mokilese, showing relationship to Pingelapese sibship 1067

small, and so it is unlikely that Mwahuele carried a fresh mutation. Most probably the gene was present but rare before Typhoon Lengkieki, descending from an earlier mutation, and by chance was transmitted through Mwahuele to several of the few survivors.

## IDENTIFICATION OF CARRIER FOUNDERS

Our initial studies by stepwise regression suggested a more reliable method to detect carrier founders. The most significant founder is defined as the one with the largest $\chi^2$ for the $2 \times 2$ contingency table of factor $\times$ kinship ($N_{++}N_{00} > N_{+0}N_{0+}$), where factor is a binary variable indicating presence or absence of an attribute and kinship is also binarized (0, +). With the same restriction, the second most significant founder then gives the largest $\chi^2$ when sibships with kinship to the most significant founder are excluded. In the computer program PHYLON this process is continued until all carrier sibships have kinship with at least one significant founder, or all founders have been called significant, or the number of significant founders reaches 25, or all nonsignificant founders have indeterminate $\chi^2$, or $N_{++}N_{00} < N_{+0}N_{0+}$.

This procedure was applied to Pingelap and Mokil, using all 99 autochthonous and migrant founders in the first five generations since Typhoon Lengkieki. For achromatopsia, sibship 1067 was the only significant founder, since all known carriers are descended from 1067. The result is clearer than by stepwise regression, showing the validity of our algorithm.

If the evidence for a single carrier founder were less convincing, we might want to test for presence of the gene among autochthonous founders, here defined as Pingelapese and Mokilese survivors of Typhoon Lengkieki. A rigorous test of significance is not feasible in such interrelated material, but the following procedure suggests itself. Let $a$ be the minimum number of carrier founders identified from

a mixture of autochthonous and migrant founders, and let $b$ designate the minimum number of carrier founders when only the 54 migrant founders are included in the analysis. Then $\chi = (b-a)/\sqrt{a}$ may be treated approximately as a normal deviate testing goodness-of-fit to a Poisson distribution with mean $a$. For example, achromatopsia gives $a = 1$, $b = 7$, $\chi = 6.0$, and so we reject the null hypothesis that the gene was absent among autochthonous founders. It is clearly implausible to assume that seven unrelated immigrants were carriers of achromatopsia, when a single autochthonous carrier is the alternative hypothesis.

Especially when autochthonous founders are excluded, the analysis may terminate with some carrier sibships still not accounted for because they are not descended from any of the migrant founders, or $\chi^2$ from the ancestral carrier is indeterminate, or $N_{++}N_{00} < N_{+0}N_{0+}$. If the number of such unexplained carrier sibships is large, the null hypothesis that the gene was absent among autochthonous founders may be rejected, whether or not $\chi$ is significant; if the number of unexplained carrier sibships is small, they may well be due to parentage errors in the pedigree.

## THE GM SYSTEM

Tests on Gm factors 1, 2, 3, 5, 6, 13, 14, and 21 revealed low frequencies of the $Gm^{1,5,6}$ and $Gm^{1,2,21}$ alleles [15]. The former seems always to derive from African ancestry, while the latter comes from Caucasian and Oriental ancestors. A binary variable representing presence or absence of the factor Gm (6) among tested sibs was regressed stepwise on kinship to migrants. Two coefficients were highly significant, the first of which was for the parents of Captain Wick, a name assumed by a foreign resident of Mokil who fathered Sera, the ancestress of all known carriers of $Gm^{1,5,6}$ among Mokilese and Pingelapese [16]. The other highly significant coefficient, for Sera's Gilbertese husband Paybuki, was negative. Since Sera transmitted her $Gm^{1,5,6}$ allele to children by two husbands, the regression analysis correctly and with surprising force indicates that Paybuki was not a carrier (fig. 2). PHYLON identifies Captain Wick as the only carrier founder.

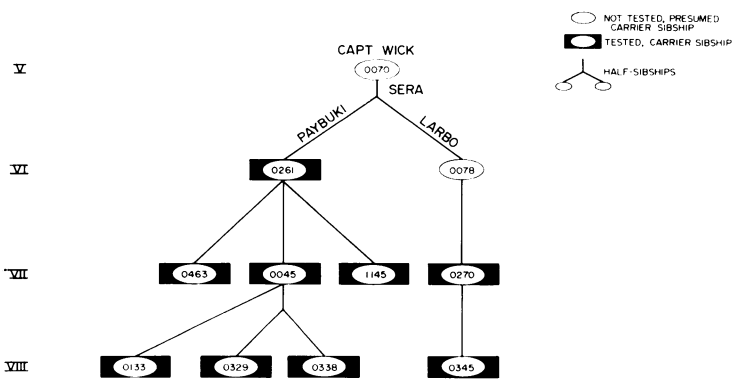


FIG. 2.—The Gm (6) factor in Mokilese and Pingelapese, showing descent from sibship 0070

There can be no doubt that Sera transmitted a $Gm^{1,5,6}$ allele from a non-Micronesian (presumably mulatto) parent. Without this evidence we would be uncertain whether immigration, mutation, or remote African affinity accounts for this allele in the Ponape district.

A binary variable representing presence or absence of the Gm (2) factor was regressed stepwise on kinship of migrants. Three coefficients were highly significant, corresponding to disjoint lines of descent from an American, a Portuguese, and a Japanese, which are also identified by PHYLON (fig. 3). Clearly the

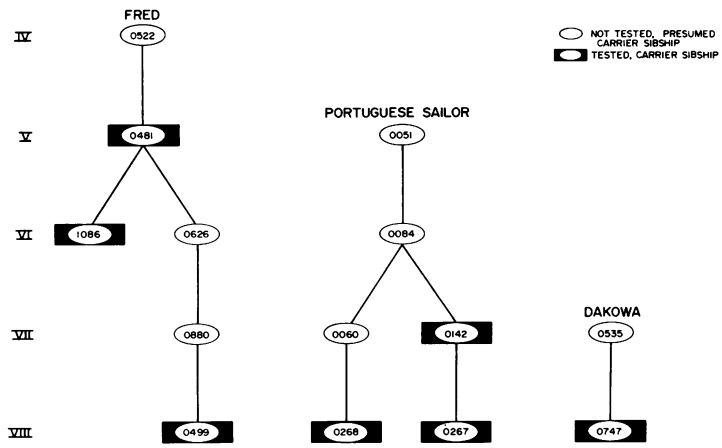


FIG. 3.—The Gm (2) factor in Pingelapese and Mokilese, showing oligophyletic descent

$Gm^{1,2,21}$ allele was introduced into the Ponape district by recent immigration.

The $Gm^{1,21}$ allele is several times more frequent than the above two rare alleles. It can be inferred in the two phenotypes Gm (−2, +21) and Gm (+2, −5, +21). A binary variable representing these phenotypes was significantly associated with pooled kinship to migrants, but not to any particular migrant. Apparently the number of founders with the $Gm^{1,21}$ allele is too large to be revealed by stepwise regression. PHYLON identifies five carrier founders, of whom three (including the most significant) were autochthones and the others were a Gilbertese and a Caucasian. It would require a minimum of $b = 11$ migrant carrier founders ($\chi = 2.7$), leaving one contemporary carrier sibship not accounted for. Evidently the $Gm^{1,21}$ allele existed in the Eastern Carolines before Typhoon Lengkieki, but its current frequency has been augmented by immigration.

## BLOOD GROUPS

By stepwise regression, the A factor is not clearly associated with individual or pooled migrants. PHYLON indicates that a minimum of 13 founders, both autochthones and immigrants, are required to account for all sibships with the A factor. The most significant founders are autochthonous, and the number of unexplained carrier sibships when autochthonous founders are excluded is too large (36) to

be dismissed as parentage errors. We conclude that the A factor was present among the survivors of Typhoon Lengkieki.

Migration is significant for the B factor. Twelve migrant carriers are required (four Marshallese, three Caucasians, three Gilbertese, one Kusaiean, and one Mortlockese), leaving two carrier sibships unaccounted for, of which one has a Marshallese parent. The B factor was apparently absent among the survivors of Typhoon Lengkieki.

The *Ms* allele is significantly associated with migrant ancestors, but the effects of individual migrants are not clear. PHYLON indicates a minimum of 12 founders, including some autochthones and many immigrants. The number of carrier sibships not accounted for by migrant founders (15) seems too large to suppose that the *Ms* allele was absent among the survivors of Typhoon Lengkieki.

The S factor is associated by stepwise regression with migrants. PHYLON indicates a minimum of seven carrier founders. Excluding autochthonous founders, the minimum number of carrier founders is eight (three Caucasians, two Gilbertese, a Japanese, a native of New Guinea, and a Marshallese). There are five carrier sibships not accounted for, of which one has a Ngatikese parent.

Some of our blood grouping was done in the field with inadequately stored reagents [17]. Tests in our Honolulu laboratory showed that field typing with saline reagents was accurate, but there had been errors with the two reagents which had been potentiated with bromelin, mostly false negative reactions with anti-E and false positive reactions with anti-c. Obviously, the field results for the Rh system cannot be used to determine gene frequencies. However, since errors on each atoll should be random with respect to ancestry, even such imperfect data can be used to detect carrier founders. Both the E and c factors are associated with kinship to migrants. PHYLON identifies a Caucasian, a Gilbertese, and a Marshallese as the most significant carriers of a *ce* allele, which was probably *cde* for the Caucasian and *cDe* for the Micronesians. No Rh-negative individual was detected. The *cDE* allele ($R^2$) is associated with two Caucasians, two Marshallese, a Gilbertese, a Mortlockese, and a Kusaiean. Of the five carrier sibships not accounted for when autochthonous founders are excluded, two have migrant parents.

Apparently the B, S, c, and E factors were absent among the survivors of Typhoon Lengkieki.

### HAPTOGLOBIN AND ISOZYMES

These systems were studied by Yamamoto and Fu [18]. The $Hp^2$ allele is associated with a Japanese, a Kusaiean, and four autochthones. Evidently the haptoglobin system was polymorphic on these atolls before Typhoon Lengkieki.

In three isozyme systems there is an allele which seems to have been absent among the survivors of Typhoon Lengkieki. The allele $PHs^A$ for erythrocyte acid phosphatase is associated with four Caucasians, two Marshallese, and a native of New Guinea. At the phosphoglucomutase locus $PGM_1$, the allele $PGM_1^2$ is associated with three Caucasians, three Japanese, two Marshallese, and a Gilbertese. At the 6-phosphogluconate dehydrogenase locus, the allele $PGD^B$ comes from a Caucasian, three Gilbertese, a Nukuoran, and a Mortlockese.

COLORBLINDNESS

Sixteen sibships had males with red-green colorblindness. Their maternal sibships were scored as positive and all others as negative. Among sibships with at least one examined member, the trait as defined was found to be associated with

TABLE 5

SEGREGATION ANALYSIS OF LEPROSY UNDER FALCONER'S MODEL

| Sibship Size ($s$) | No. Affected ($r$) | No. Observed ($N_{sr}$) | No. Expected |
|---|---|---|---|
| 1 | 0 | 118 | 103.37 |
| 1 | 1 | 0 | 9.63 |
| 2 | 0 | 46 | 51.45 |
| 2 | 1 | 15 | 9.14 |
| 2 | 2 | 0 | 0.41 |
| 3 | 0 | 29 | 30.98 |
| 3 | 1 | 8 | 8.26 |
| 3 | 2 | 3 | 0.73 |
| 3 | 3 | 0 | 0.02 |
| 4 | 0 | 27 | 25.61 |
| 4 | 1 | 8 | 9.10 |
| 4 | 2 | 1 | 1.21 |
| 4 | 3+ | 0 | 0.07 |
| 5 | 0 | 21 | 22.21 |
| 5 | 1 | 12 | 9.87 |
| 5 | 2 | 1 | 1.75 |
| 5 | 3+ | 0 | 0.17 |
| 6 | 0 | 15 | 15.00 |
| 6 | 1 | 8 | 8.00 |
| 6 | 2 | 2 | 1.78 |
| 6 | 3+ | 0 | 0.22 |
| 7 | 0 | 7 | 7.71 |
| 7 | 1 | 5 | 4.80 |
| 7 | 2 | 2 | 1.28 |
| 7 | 3+ | 0 | 0.21 |
| 8 | 0 | 7 | 7.59 |
| 8 | 1 | 6 | 5.40 |
| 8 | 2 | 2 | 1.68 |
| 8 | 3+ | 0 | 0.33 |
| 9 | 0 | 3 | 2.32 |
| 9 | 1 | 0 | 1.86 |
| 9 | 2 | 2 | 0.66 |
| 9 | 3+ | 0 | 0.16 |
| 10 | 0 | 1 | 0.85 |
| 10 | 1 | 1 | 0.76 |
| 10 | 2+ | 0 | 0.38 |
| 11 | 0 | 1 | 1.18 |
| 11 | 1 | 1 | 1.15 |
| 11 | 2 | 1 | 0.51 |
| 11 | 3+ | 0 | 0.16 |
| 12 | 0 | 0 | 0.36 |
| 12 | 1 | 0 | 0.38 |
| 12 | 2 | 1 | 0.19 |
| 12 | 3+ | 0 | 0.07 |
| Total | ... | 354 | 354.00 |

NOTE.—Parental affection unspecified ($h^2 = 0$; $A = .0816$).

a Caucasian, a Gilbertese, a Marshallese, and a Mortlockese. Of the two carrier sibships not accounted for by migrant founders, one had a Ngatikese mother.

## LEPROSY

We are indebted to Dr. Norman Sloan for making his data on leprosy available to us. For Pingelapese sibships there is no significant association between leprosy and kinship to any founder, or to pooled immigrant founders. Sibships with at least one examined member were analyzed for unspecified parental affection by Falconer's quasi-continuity model [19]. Simultaneous estimation gave a prevalence of .0816 ± .0081 and a heritability of 0 ± .14 (table 5), reflecting the lack of any pronounced concentration of cases within particular families. As always in human material, heritability is defined in the broad sense to include environment common to sibs. This null estimate of heritability, together with absence of a founder effect, indicates that genetic susceptibility does not explain the focus of leprosy on Pingelap, which may be due to contacts in the phosphate mines of Nauru at the beginning of this century [20]. We see that the methods of historical genetics are capable of excluding as well as elucidating hereditary factors.

## DISCUSSION

During the first five generations after Typhoon Lengkieki, there were 13 Caucasians, seven Japanese, and 30 Pacific Islanders from outside the Eastern Carolines (mostly Marshallese and Gilbertese) who left descendants on Pingelap and Mokil. Their contribution to sibships with at least one examined member is given in table 6. The impact of long-range migration on these remote atolls is striking.

TABLE 6

CONTRIBUTION OF LONG-RANGE IMMIGRANTS DURING GENERATIONS 1–5 TO SIBSHIPS WITH AT LEAST ONE EXAMINED MEMBER

| | $4\Sigma\phi_{it}$ | | |
|---|---|---|---|
| POPULATION | Caucasian | Japanese | Oceanic |
| Pingelapese | .020 | .010 | .056 |
| Mokilese | .099 | 0 | .258 |

Alleles in populations are distinguished as idiomorphs, polymorphs, or monomorphs according to whether their frequencies are less than .01, between .01 and .99, or greater than .99, respectively. Alleles may also be distinguished by their origin as monophyla, oligophyla, or polyphyla according to whether they are identical by descent from the same, a few, or many founder genes, respectively. To be specific, we tentatively suggest that few means that more than one but less than six founders account for at least 90% of known carriers. These concepts do

## TABLE 7

ORIGIN OF ALLELES ON PINGELAP AND MOKIL

| ORIGIN AND ALLELE | MINIMUM NO. FOUNDERS | | CONTEMPORARY CARRIER SIBSHIPS | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Admitting Autochthones $a$ | Excluding Autochthones $b$ | Total | Not Explained Admitting Autochthones | Not Explained Excluding Autochthones | Due to Five Most Significant Founders | $\frac{b-a}{\sqrt{a}}$ | PRESENT IN AUTOCHTHONOUS FOUNDERS |
| Monophylon: | | | | | | | | |
| Achromatopsia | 1 | 7 | 117 | 0 | 35 | 117 | 6.00 | + |
| $Gm^{1,5,6}$ | 1 | 1 | 9 | 0 | 0 | 9 | 0 | 0 |
| Oligophylon: | | | | | | | | |
| $Gm^{1,2,21}$ | 3 | 3 | 7 | 0 | 0 | 7 | 0 | 0 |
| $Gm^{1,21}$ | 5 | 11 | 64 | 0 | 1 | 64 | 2.7 | + |
| $R^o + r$ | 5 | 7 | 68 | 1 | 4 | 63 | 0.9 | 0 |
| $Hp^2$ | 6 | 12 | 186 | 2 | 13 | 183 | 2.4 | + |
| $PHs^A$ | 5 | 7 | 33 | 0 | 0 | 30 | 0.9 | 0 |
| $PGD^B$ | 9 | 6 | 27 | 0 | 1 | 25 | −0.7 | 0 |
| Colorblindness | 6 | 4 | 16 | 0 | 2 | 15 | −0.8 | 0 |
| Polyphylon: | | | | | | | | |
| $A$ | 13 | 15 | 343 | 1 | 36 | 291 | 0.6 | + |
| $B$ | 7 | 12 | 137 | 0 | 2 | 113 | 1.9 | 0 |
| $Ms$ | 12 | 16 | 269 | 2 | 15 | 228 | 1.2 | + |
| $PGM_1^2$ | 8 | 9 | 25 | 0 | 0 | 20 | 0.4 | 0 |
| $NS + MS$ | 7 | 8 | 63 | 0 | 5 | 55 | 0.4 | 0 |
| $R^2$ | 7 | 8 | 67 | 1 | 5 | 55 | 0.4 | 0 |

NOTE.—The alleles $O$, $NS$, $R^1$, $Gm^{1,3,5}$, $Hp^1$, $AK^1$, $Gd^B$, $PHs^B$, $PGM_1^1$, and $PGD^A$ are polyphyla present in high frequency among the survivors of Typhoon Lengkieki.

not depend on population size, but distinctions by frequency are more interesting and feasible in large populations and distinctions by origin in small populations. Classification depends on the founder and reference generations and may change by drift, mutation, or migration. Thus a single migrant or mutant may convert a monophylon to an oligophylon, which random extinction may again reduce to a monophylon. The number of founder genes in an allelic set often increases with the reference population: an allele may be monophyletic in an isolate, but polyphyletic in the region of which the isolate is a part. The apparent number of founder genes may decrease as a genealogy is extended, revealing unsuspected identity by descent. Finally, the apparent number of founders may be increased by parentage errors.

These principles are illustrated by Pingelap and Mokil (table 7). Achromatopsia and $Gm^{1,5,6}$ are monophyla. $Gm^{1,2,21}$, $Gm^{1,21}$, and many of the other alleles studied are oligophyla, the majority of carriers coming from a handful of carrier founders. The alleles $Gm^{1,5,6}$, $Gm^{1,2,21}$, $B$, $NS + MS$, $R_o + r$, $R_2$, $PHs^A$, $PGM^2_1$, $PGD^B$, and colorblindness were apparently absent from the survivors of Typhoon Lengkieki and are becoming polyphyletic by migration.

SUMMARY

Methods of historical genetics in isolates are developed and illustrated. The subequal effective sizes of Pingelap and Mokil, with census sizes in the ratio of 2:1, result from more variable fertility on Pingelap after Typhoon Lengkieki two centuries ago. Achromatopsia and $Gm^{1,5,6}$ are monophyla (descended from a single carrier founder). Many other alleles studied are oligophyla (descended from a few carrier founders). The focus of leprosy on Pingelap is not due to genetic susceptibility.

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