of tops is appreciable and the elongation of roots is very slight. In mixed solution of $N/5000$ $CaCl_2 + N/50$ NaCl, the injurious property of sodium is entirely counteracted. Thus, calcium is a powerful antidote for sodium that is present in injurious concentrations."

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We are aware of the distinction between saline and sodic conditions. However, by definition, sodic soils bear on their negatively charged exchange surfaces high proportions of exchangeable sodium ions (more than 15 percent). Bernstein (*1*) has given many instances in which responses of plants were specifically related to soil exchangeable sodium, that is, to the sodic condition. This condition did not prevail in our experiments which were done in solution culture in the absence of soil or other cation exchangers.

Bernstein states, "By definition, salin soils contain enough calcium to meet ordinary nutritional requirements of plants." Salinity of soils is defined in terms of the conductivity of the saturation extract, without any reference to calcium (*2*).

We disagree that our results are due to the short period of observation, mild growing conditions, and a neglect of subtle effects on growth and yield. (i) In the presence of adequate calcium, we have grown plants at the same salinity (50 m*M* NaCl) to maturity, and obtained a normal yield of beans. (ii) We have not studied the effects of climatic conditions systematically; the experiment reported by us was done under midsummer conditions at Davis; temperatures in the greenhouse sometimes exceeded 35°C.

Bernstein says that our results can be attributed to our "having followed the erstwhile frequent practice of studying salt uptake by plants from single-salt solutions in short-term experiments." He errs; the plants in our experiments grew in complete nutrient solutions, as has been stated in our report.

We are aware that there are some prior mentions of the role of calcium in salt toleration by plants [see, for example, references 10, 11, and 12 in (*3*) and the note from Shear]. We called attention to this matter because it is frequently neglected when the effects of salt on plants are studied. We specifically focus on mechanisms of salt damage to plants, including the propensity of sodium ions at high concentrations to displace calcium from root cells and, especially, from their membranes. This phenomenon cannot usefully be studied in experiments in which the ratio of Na to Ca is kept constant, as has frequently been the practice (*4*).

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References and Notes

1. L. Bernstein, in *The Problems of the Arid Zone* (UNESCO, Paris, 1962), p. 139.
2. L. A. Richards, Ed. "Diagnosis and Improvement of Saline and Alkali Soils," *U.S. Department of Agriculture Handbook No. 60* (U.S. Government Printing Office, Washington, D.C., 1954), pp. 4–5.
3. O. E. Elzam and E. Epstein, *Agrochimica* **13**, 187 (1969).
4. L. E. Francois and L. Bernstein, *Agron. J.* **56**, 38 (1964).

19 January 1970

Intermixture and Selection

Reed (*1*) has reviewed studies designed to estimate the proportion (*M*) of Caucasian ancestry in different American Negro populations, and, in particular, he provides a criticism of studies (*2, 3*) in which it was suggested that a comparison of the *M* values for different genes could indicate which of those genes may have been affected by (directional) natural selection during the past 300 years.

Reed's discussion of estimates of *M* for the ABO blood group system shows some misunderstanding of the statistical principles involved. His analysis (*4*) of gene frequency data from a study of Negroes in Oakland, California, shows that $M(\text{ABO}) \cong M(Fy^a)$ and that $M(\text{A}) \cong M(\text{B}) \cong M(\text{ABO})$. From these results, and the hypothesis that the frequencies of the Fy^a allele have not been affected by natural selection, he concludes that the estimate of *M* for the ABO locus is not greatly disturbed by selection and that there is no suggestion of selective differences between the *A* and *B* alleles. These observations are, however, not "pertinent" to a consideration of differential selective pressures on the A_1 and A_2 alleles, as he suggests they are, since, if there are differences in selective forces acting on the alleles at a locus, his joint likelihood estimates of *M* for the ABO locus, or the *A* "allele," need not reflect such differences. For interallelic comparisons it is the relation between estimates of *M* for different alleles that is of interest and not their numerical values per se. Similarly, if we wish to detect differences in directional selection pressures among genes at different loci, joint likelihood estimates of *M* are inappropriate and we must consider the magnitude of an estimate of *M* relative to that of some other gene (Fy^a or R^0) presumed to be unaffected by directional selection.

For example, Workman (*3*) suggested that an examination of the distribution of frequencies of the A_1, A_2, and *B* alleles in several West African, Western European, and American Negro populations indicated that $M(\text{A}_2) \gg M(\text{A}_1)$ in American Negro populations. Reed's own data from Oakland would appear to support this hypothesis. The $\text{A}_1\text{A}_2\text{BO}$ gene frequencies given by Mourant (*5*) show that, in West Africa, the frequency of A_1 is almost always greater than 0.095 and that of A_2 is generally less than 0.05. Using these values and the frequencies of the genes in Oakland Negroes and Western Europeans (*4*) we find that $M(\text{A}_2)/M(\text{A}_1) \cong 2$. This is the minimum value likely if the range of published West African frequencies is considered. However, if frequencies of A_1 and A_2 more commonly observed in West Africa (*5*) are considered—say, 0.10 and 0.05, or 0.105 and 0.045—then the ratios would be $(0.578)/(0.194) = 2.98$ or $(0.685)/(0.142) = 4.82$, respectively.

Reed suggests that, by his examination of the hypotheses involved in obtaining estimates of *M*, results which formerly provided claims that selection has been demonstrated now appear only "suggestive but far from conclusive." In fact, all of the underlying assumptions and the restrictions they place on our ability to draw conclusions were discussed by Workman (*3*). For example, one could test the hypothesis that $M(\text{A}_2)/M(\text{A}_1)$ is greater than 1 by means of a sign test of the ratio of the *M* values from a series of populations, but, as discussed elsewhere (*3*), even when a ratio such as that noted for the A_1 and A_2 alleles is observed in several different American Negro populations, the conclusion that selection is involved still depends upon assumptions about ancestral African frequencies.

Finally, a concern with standard errors is appropriate when the problem

is to obtain a single estimate of M most likely to represent the true amount of Caucasian ancestry in a Negro population, since the analysis of historical intermixture must, of necessity, depend upon such statistical results. On the other hand, recognition of various sources of error in the analysis of intermixture and selection (*1, 3*) led to a suggestion that the examination of the relative magnitude of M values from a population can never be more than a first step toward the demonstration of ongoing selection. This use of estimates of M should serve primarily as a screening technique by which we hope to determine which loci will prove to be the most rewarding subjects for subsequent medical and biological analyses.

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References

1. T. E. Reed, *Science* **165**, 762 (1969).
2. P. L. Workman, B. S. Blumberg, A. J. Cooper, *Amer. J. Hum. Genet.* **15**, 429 (1963).
3. ———, *Hum. Biol.* **40**, 260 (1968).
4. T. E. Reed, *Amer. J. Hum. Genet.* **20**, 142 (1968).
5. A. E. Mourant, A. C. Kopeć, K. Domaniewska-Sobczak, *The ABO Blood Groups* (Blackwell, Oxford, 1958).

5 December 1969

Workman raises several points which require comment. Before replying to them, however, I wish to stress that, in spite of other differences, we do not differ in believing that the study of natural selection in American Negroes is important and that M estimates can be a useful method for this study. We do differ in our ways of obtaining M estimates and in the use we make of them. In particular, we differ (i) in the choice of African gene frequencies, and (ii) in the details of calculating (and then using) M from the three gene frequencies, African (q_a), Caucasian (q_c), and U.S. Negro (q_n).

I find Workman's choice of African data very puzzling since he is well aware of the importance of obtaining a good estimate for q_a. In spite of extensive data showing that the primary slaving area of Africa, for slaves brought to the United States, extended from Senegal to Southwest Africa (*1*), he consistently refers to "West Africa" as the area from which the slaves were drawn, and he uses data from only that area (*2*). There is also considerable doubt as to how representative of that area the data are (*3*). Workman limits himself to "West Africa" despite the fact that about 25 percent or more of American slaves came from the area south of Nigeria (*1, 4*). It is simply wrong to state or imply that West Africa is the only area of genetic importance in calculating M. If Workman disagrees with this statement he should present his evidence.

As I have discussed at some length (*1*), there are only a few genes for which we have reasonable frequency data over the entire slaving area. These include Fy^a (Duffy blood group) and A (actually A_1 plus A_2) and B of the ABO system, but *not* A_1 and A_2 separately. This is why I did not use A_1 and A_2 but did use A and B. Workman misunderstands my position here. It is that, since the mean African A_1 and A_2 frequencies, over the slaving area, are not known, he cannot meaningfully calculate $M(A_1)$ and $M(A_2)$. I was not implying that my evidence for lack of strong selection on the A "allele" ($= A_1 + A_2$ alleles) was directly pertinent to his A_1 and A_2 results. When $M(A_1)$ and $M(A_2)$ can justifiably be calculated, however, my A "allele" results will be indirectly pertinent because selection on A_1 and A_2, if any, is then required to be of opposite sign, and with magnitudes adjusted for the greater frequency of the A_1 allele.

The problem of the correct African gene frequency is critical when A_1 or A_2 is used to obtain an estimate of M. This is best shown by giving the actual numbers in the calculations Workman presents for $M(A_2)$. Using the Oakland frequencies for Negroes (q_n) and West European Caucasians (q_c), and an "estimated" q_a of 0.05, Workman estimates $M(A_2)$ as $(q_n - q_a)/(q_c - q_a) = (0.0585 - 0.05)/(0.0647 - 0.05) = 0.578$. The facts that "0.05" is uncertain, that it is within 0.01 of q_n, and that the numerator and denominator are of the same order of magnitude as the standard errors of q_n and q_c (0.0032 and 0.0028) do not seem to disturb Workman. If q_a (the weighted mean over the entire slaving area) were actually 0.056 (and we have no evidence that it is not) $M(A_2)$ would then become $0.0025/0.0087 = 0.29$, close to estimates from other genes; this estimate is almost meaningless, however, since the numerator, 0.0025, is *less* than its standard error, 0.0032. The calculation of $M(A_1)$ is similarly tenuous, being $(0.1160 - 0.10)/(0.1823 - 0.10) = 0.194$ for an *assumed* q_a of 0.10. The ratio of these two M's is the basis for Workman's belief in selection at the A_1A_2BO locus. Workman is willing to make and use such calculations; I am not.

His statement that it is only the relative magnitudes of $M(A_2)$ and $M(A_1)$ that matter is misleading when there is such extreme uncertainty in the estimates. His further implication that standard errors are not important here and might be replaced by a sign test on "M values from a series of populations" seems inappropriate since he in fact does not make a sign test, or any other statistical test, on his M values. Workman has presented no evidence that in this area we can abandon our usual concern with the statistical reliability of numbers (as well as with the *origin* of the numbers) and be guided only by intuition. Others who have done this have been led to false conclusions (*5*).

I would like to conclude more harmoniously by reiterating that Workman and I have no differences with regard to the importance and potential utility of M estimates. He was one of the first to use these estimates as a tool for detecting selection in racial hybrids, and my critical remarks should not obscure the possibility that "some of these selective genes may already have been identified" (*1*).

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References and Notes

1. T. E. Reed, *Science* **165**, 762 (1969).
2. The A_1A_2BO data which Workman uses [A. E. Mourant, A. C. Kopeć, K. Domaniewska-Sobczak, *The ABO Blood Groups* (Blackwell, Oxford, 1958), pp. 228–229] include "West Africa," Gambia, French West Africa (Touaregs), Ghana, and Nigeria, but, of these, only the data for Ghana and Nigeria are relevant here (see *1*, table 1). About 60 percent of American slaves came from some region other than Ghana or Nigeria (*1*, table 1).
3. The total number of individuals sampled in Ghana and Nigeria [Mourant *et al.*, *The ABO Blood Groups* (Blackwell, Oxford, 1958)] is 1536; 853 (56 percent) of these are from one study on Ewe people, which has a very strong indication ($P << .001$) of either genetic inhomogeneity or mistyping or both. More important, the Ewe peoples are of negligible importance in estimating M, having constituted no more than about 1 percent of the slaves in the North American slave trade (*1*, table 1; P. D. Curtin, personal communication). The Ewe are quite distinct culturally from neighboring peoples, such as those in the Akan linguistic group [G. P. Murdock, *Africa: Its Peoples and their Culture History* (McGraw-Hill, New York, 1959), pp. 252–253], which *were* important in the slave trade.
4. W. S. Pollitzer, *Amer. J. Phys. Anthropol.* **16**, 241 (1958).
5. For example, J. E. Bowman, H. Frischer, F. Ajmar, P. E. Carson, and M. K. Gower [*Nature* **214**, 1156 (1967)], using gene AK^2 at the adenylate kinase locus, estimated M in the Chicago area as 0.13 and concluded, without statistical evidence, that it was really lower than other estimates for M in northern Negroes. In fact, however, the 95-percent confidence interval for their estimate is about $0.03 - 0.23$ (*1*), hence this conclusion is unwarranted.

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