on coupling or biosynthetic steps beyond the initial event of hormone-receptor interaction. Altered intracellular potassium concentrations during high sodium intake could also be an important factor in modifying the aldosterone secretory response of the adrenal cortex to angiotensin II.

In addition to the possible allosteric effect of a cation site on receptor affinity, other explanations for the action of sodium ions on angiotensin II binding in the particulate adrenal receptor preparation should be considered. Sodium ions may cause an alteration of the conformation of angiotensin II, or may stimulate an enzyme which catalyzes the breakdown of a compound inhibiting high-affinity angiotensin binding. In the experiments reported here, it was observed that the extent of the maximum stimulation of angiotensin II binding by sodium ions varied from as little as 10 percent to as much as 100 percent in different receptor preparations. Further investigation of this variability led to the demonstration that guanyl nucleotides also influence angiotensin binding, and are effective at nanomolar concentrations; these studies have been reported in detail elsewhere (*9*).

The dependence of angiotensin II binding to adrenal membranes on multiple factors, including sodium and potassium ions and guanyl nucleotides, indicates that modulation of the interaction between the peptide hormone and specific adrenal receptor sites is a complex process. It will be necessary to determine the extent to which these factors, which have been demonstrated to influence hormone uptake in vitro, are concerned in the regulation of angiotensin II binding and target cell activation in vivo.

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Ethnic Classification of Mexican-Americans

In epidemiological studies as well as in other demographic or population studies, it is useful, and often essential, to make analyses within racial groups. The classification of racial hybrids, such as American Blacks and Mexican-Americans, however, may pose problems here. Thus Menck *et al.* (*1*), in their study of possible effects of air pollution on risk of lung cancer in Los Angeles County, claim to restrict their analysis to Caucasians but they include Mexican-Americans in this group. The problem here is that about one-third of the ancestry of Mexican-Americans in California is Mexican Indian, a fact which may be relevant to this and other epidemiological studies.

A good estimate of the amount of Caucasian ancestry in young Mexican-American adults residing in the Oakland, California, area in the 1960 to 1965 period can be made from published data (*2*). This estimate should be appropriate for Mexican-Americans of other urban areas of the state. The *r (cde)* gene of the Rh blood group system is especially useful here since it is essentially absent in "pure" Mexican Indians (*3*), but is common in Caucasians so that its frequency in the hybrid Mexican-Americans will reflect the amount of Caucasian ancestry (*M*, proportion of genes from Caucasian ancestors).

A maximum estimate of M is obtained by assuming that the frequency of r in the ancestral Mexican Indians was in fact zero, and that no other populations contributed genes (*4*). Then, with the use of the Oakland frequencies for r of 0.3849 ± 0.0039 for persons of Caucasian ancestry (*5*) and 0.2615 ± 0.0213 for Mexican-Americans, and making the usual assumption (*6*) that population mixture was the only process determining the frequency of r in modern Mexican-Americans, the calculated value of M is $0.2615/0.3849 = 0.679 \pm 0.056$ (*7*). The minimum amount of Indian ancestry is therefore estimated to be $1 - M = 0.321 \pm 0.056$.

I have no information concerning the importance of this amount of Indian ancestry to the study of Menck *et al.*; it may well be negligible. My point is that the actual genetic composition of hybrid populations should be recognized and considered in analysis. Important ethnic differences in disease susceptibility exist, as Menck *et al.* recognize. It seems prudent to consider the possibility that biracial hybrids may also have distinctive disease susceptibilities.

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4. Evidence for African ancestry in Mestizos of Central Mexico has been presented by M. H. Crawford and P. Workman [*Am. J. Phys. Anthropol.* **40**, 133 (1974)], who estimate ancestry in their sample to be 70 percent Indian, 22 percent Spanish, and 8 percent African. But the R° (*cDe*) (Rh gene) frequency, which is about 0.60 in West Africans, between 0.00 and 0.15 in "pure" Mexican Indians (*3*), and 0.0280 ± 0.0019 in Oakland Caucasians (*2*), has a frequency of 0.0481 ± 0.0148 in the Oakland Mexican-Americans (*2*). This low frequency indicates little, if any, African ancestry.
5. The frequency of r in modern Spaniards, whose ancestors were presumably the major contributors of Caucasian genes, is within 1 percent of that of the Oakland Caucasians [A. E. Mourant, *The Distribution of Human Blood Groups* (Blackwell, Oxford, 1954), p. 384].
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7. $M \pm$ standard error of M. Standard error calculated by formula in (*6*).

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