for two periods of half an hour and subsequent drying, or air drying. Also, according to his table, air-dried yeast did not cause fermentation but did reduce triphenyl tetrazolium chloride. These results are rather puzzling. Drying yeast in the air and then re-suspending it in water with substrate may result in no fermentation at first, but there is nothing to prevent the yeast from growing from a spore stage and eventually causing fermentation. Also I found that soaking yeast in either acetone or ethyl alcohol solutions of more than 50 per cent concentration for 1 hr. with subsequent drying and re-suspension in water resulted in complete inactivation of the reduction of triphenyl tetrazolium chloride, while soaking the cells in 16 per cent acetone or alcohol for 24 hr., followed by drying and re-suspension, did not inactivate them. Cells of onions soaked in concentrated acetone for 1 hr. with subsequent washing and immersion in 0·5 per cent triphenyl tetrazolium chloride did not show the reduction reaction in the dark.

It appears, therefore, that although reduction of triphenyl tetrazolium chloride may not always be a good indication of viability in the sense that the entire respiratory mechanism is still functional, the lack of reduction in cells normally reducing it is a fairly certain sign that death has occurred. Avoidance of light is especially important in the case of triphenyl tetrazolium chloride, but not in that of blue tetrazolium. Oxygen tension must not be excessive.

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Maximum Likelihood Calculation of *Rh* Gene Frequencies in Pacific Populations

In general, the calculation of maximum likelihood[1,2] estimates of gene frequencies requires the preliminary calculation of trial values, followed by the computation of corrections to the trial values, by a method which is essentially Newton's method of successive approximations for getting a root of an equation. In many Pacific populations (Gilbertese, Fijians, etc.), only four or five *Rh* types *CDe/C* (R_1R_1), *CDe/c* (R_1r), *CDE/c* (R_1R_2), *cDE* (R_2), and occasionally a little *cDe* (R_0) are present[3], and the composition of the population can be explained by assuming that only three genes, R_1, R_2, and R_0, are present. For such cases the maximum likelihood equations can be solved explicitly, and the best estimates of the gene frequencies obtained without any process of successive approximations. If we designate the frequencies of the five phenotypes, in the order just mentioned, as $\overline{H}$, $\overline{K}$, $\overline{P}$, $\overline{T}$ and $\overline{Q}$, the solution of the maximum likelihood equations gives the following formulæ:

$$R_1 = \overline{H} + \overline{K}/2 + \overline{P}/2$$

$$R_2 = \frac{4(1-R_1) + \overline{P} - \sqrt{[4(1-R_1) + \overline{P}]^2 - 16(\overline{P}+\overline{T})(1-R_1)}}{4}$$

$$R_0 = 1 - R_1 - R_2$$

If desired, standard errors can be calculated in the usual way.

The derivation of these formulæ, and illustrative calculations, will be published elsewhere.

If the gene R_z is present, as in Indonesians[4], an additional phenotype, *CDE/C* (R_1R_z), whose frequency we may designate as $\overline{J}$, is present, and the above formulæ will not work. Williams[5] has published a modification of Fisher's method of free parameters which is applicable to such data. The same results can be obtained a good deal more simply by using the formulæ published[6] for dealing with the *MNS* system, to which this particular *Rh* system is exactly similar. It is only necessary to make use of the following equivalences:

Short symbol	Phenotype	Equivalent *MNS* phenotype
H	*CDe/C*	*M*
J	*CDE/C*	*MS*
K	*CDe/c*	*MN*
P	*CDE/c*	*MNS*
Q	*cDe*	*N*
T	*cDE*	*NS*

Taking $R_z = m_S$, $R_1 = m_s$, $R_z = n_S$ and $R_0 = n_s$, trial frequencies which may be found by Mourant's methods[3] may now be inserted into the formulæ given for the *MNS* system and corrections found so as to obtain the maximum likelihood estimates.

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Release of Histamine in Hepatectomized Dogs during Anaphylactic, Peptone and Tryptic Shock

It is well known that in dogs the liver is the main source of the histamine released during certain types of shock, such as anaphylactic and peptone shock[1]. However, shock can be obtained in hepatectomized dogs[2] and some evidence, particularly recent work by Feldberg and Schachter[3], indicates that under certain conditions tissues other than the liver release relatively large amounts of histamine.

I have found that histamine occurs in the blood of hepatectomized dogs during anaphylactic, peptone and tryptic shock. Studies have also been made of the blood coagulation time, the amount of platelets and the hæmatocrit during shock.

The histamine in blood was estimated according to Code's method. Hepatectomy was carried out by