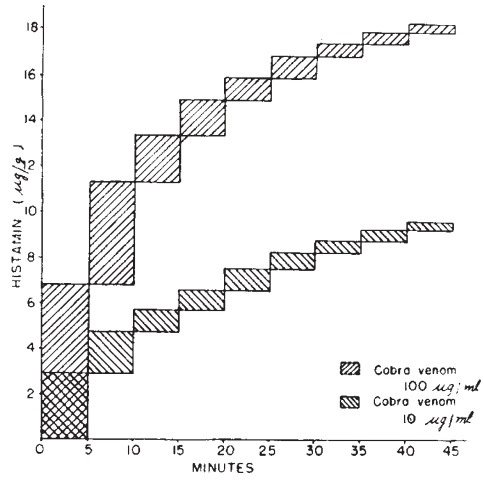

Total release of histamine from rat diaphragm when immersed for successive periods of 5 min. in Tyrode solution containing cobra venom

in Tyrode solution. Before assay, the gut was desensitized[1] to the action of cobra venom. The histamine content of the muscle was estimated[3] as required. We also found that Tyrode solution itself released small quantities of histamine from the diaphragm[2]. The histamine content of rat diaphragms (eight experiments) varied from 14 to 21 μgm./gm. of fresh tissue.

The amounts of histamine liberated by cobra venom (average of five experiments) are represented graphically; doses of 25–50 μgm./ml. (not shown) of the venom gave intermediate values. The release of histamine increased with increasing doses of the venom, and of the liberated amount 50 60 per cent was set free from the tissue during the first ten minutes. At a concentration of 100 μgm./ml. of the venom, the tissue was nearly depleted of its store of histamine in the course of 45 min., for at the end of the experiment the diaphragm was found to contain on an average 2·5 μgm. of histamine/gm. of fresh tissue. When the experiment was continued for half an hour more, the muscle was completely free from histamine.

The symptoms of poisoning in man due to snake venoms at some stages simulate that of the acute effects of histamine on tissues[4]. The release of histamine by the venom may account for such symptoms. The foregoing results suggested that antihistamines might afford some protection to the rat from the deleterious effect of the venom. 'Pyribenzamine' (CIBA, 10 mgm./kgm. subcutaneously) and 'Antistin' (5 mgm./kgm. intraperitoneally) given before or along with the venom (0·5 mgm./kgm. intravenously) did not significantly alter the survival periods (50–115 min.) of adult rats. Investigation is proceeding in order to ascertain the mechanism of histamine release by the venom and how far the liberated histamine is responsible for bringing about the death of the animal.

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Distribution of *ABO-MN* and *Rh* Types among Eskimos in South-west Greenland

In the early summer of 1951, an epidemic of measles in south-west Greenland gave us an opportunity of carrying out a number of blood-typing tests on 187 non-related Eskimos of pure race. The distribution of the blood types observed is evident from the following tabulation.

ABO and *MN* groups:

Observed phenotype distribution	*A*: 49·46 per cent		*M*: 67·38 per cent	
	B: 6·45 ,,		*N*: 4·28 ,,	
	AB: 2·69 ,,		*MN*: 28·40 ,,	
	O: 41·40 ,,			
Total number of persons tested: 187.				
Calculated frequency of gene	*p*: 0·3082		*m*: 0·8158	
	q: 0·0467		*n*: 0·1848	
	r: 0·6434			
Expected phenotype distribution calculated from the frequency of the gene	*A*: 49·14 per cent		*M*: 66·53 per cent	
	B: 6·01 ,,		*N*: 3·42 ,,	
	AB: 2·88 ,,		*MN*: 30·15 ,,	

$D_{(ABO)} = 1 - (p + q + r) = 0{\cdot}0617$. P.E.$_D$ = 0·0058
$D_{(MN)} = 1 - (m + n) = 0{\cdot}0279$. P.E.$_D$ = 0·0247

	Rhesus types				
Observed reaction types on employment of mentioned sera	anti-*c*: +	+	−	−	+
	,, *C*: +	+	+	+	−
	,, *D*: +	+	+	+	+
	,, *E*: +	−	−	+	+
Most frequent genotypes signified by the reaction types	$\frac{CDe}{cDE}$	$\frac{CDe}{cDe}$	$\frac{CDe}{CDe}$	$\frac{CDe}{CDE}$	$\frac{cDE}{cDE}$
Distribution of the reaction types:	33·55	3·17	52·53	5·06	5·70 per cent

Total number of persons tested with the four antibodies: 158

Gene distribution if all the observed reaction types only signify the above genotypes	*CDe*	*CDE*	*cDE*	*cDe*
	72·42	2·53	22·47	1·58 per cent
Calculated gene distribution (calculated as if only the above genes occur in the material)	*CDe*	*CDE*	*cDE*	*cDe*
	72·48	3·49	23·88	2·19 per cent

The frequency of $\frac{CDe}{cDE}$ is not used in the calculation of the gene distribution. According to the observations, this frequency is 0·3355, and according to the calculated gene frequencies it is 0·3461 (the deviation is about one-half of the probable maximum deviation), so it is most improbable that genes other than the above-mentioned (for example, *cde*) occur in an appreciable number in the material.

Further, only five persons were found with the reaction type $c+C+D+E-$. This frequency corresponds to the frequency of the genotype $\frac{CDe}{cDe}$ in the *Rh*-positive part of a European population. So it seems reasonable that the *Rh*-negative gene (*d*) did not occur in the present material.

The low frequency of gene *N* is a conspicuous feature. This has been found previously from the examination of Eskimos in East Greenland[1]. The same phenomenon is encountered in the Indian tribes of North America, whereas Mongoloid races—to which the Eskimos usually are referred—do not present this character.

In this material the gene *d* did not occur. This seems a common trait too for the earlier tested Mongoloid races.

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