high concentrations of intermedin[6]. The submaximal dispersion of melanin frequently observed in operated animals was, however, not due to excess secretion of intermedin from the denervated neuro-intermediate lobe, because injection of intermedin into such animals produced maximal dispersion of melanin.) It can therefore be concluded that if a whitening hormone is secreted in response to a white background it is present in the blood in amounts too small to modify visibly the effect on the melanin dispersion of even small amounts of intermedin. Paling upon transfer to a white background must result from the disappearance of intermedin from the blood. The fact that two animals, *X*.4 and *B*.9, did respond to the background after the operation does not necessarily disprove this conclusion. The persisting colour response can be due to incomplete denervation of the neuro-intermediate lobe.

It is generally agreed that intermedin secretion from the pars intermedia is controlled by inhibitory nerves, because lesion of hypothalamus or transplantation of the pars intermedia has been observed to darken the operated animal permanently[7]. We found, however, that the rate of intermedin secretion from a denervated neuro-intermediate lobe can vary from high to zero, as judged from the state of the melanophores. The toads especially operated during the breeding season were pale after the operation. The normal secretion of intermedin is therefore probably regulated by both inhibitory and secretory nerves.

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HÆMATOLOGY

M_1, a Subdivision of the Human Blood-Group Antigen M

A 'new' antibody, anti-M_1, has been found as a component in six out of twenty available human anti-M sera: in one of them practically all the antibody was anti-M_1. No anti-M_1 could be detected in anti-M sera from eight rabbits and from one horse, nor in an extract of the seeds of *Iberis amara* (which contains anti-M): nor could it be found in a wide range of other antisera. We are calling the antibody anti-M_1, because in several ways it behaves like anti-A_1 of the ABO system and anti-P_1 of the P system.

The corresponding antigen, M_1, differs qualitatively from ordinary M. It is not merely strong M, because our two best anti-M_1 sera are insensitive to the greater amount of M known to be present in cells representing the genotype *MM* than in those representing the genotype *MN*; and, conversely, because two anti-M sera in our collection which are particularly sensitive to this 'dosage' difference make no distinction between M_1 and ordinary M. The Ss antigens do not appear to be playing a part in the M_1–M_2 difference.

Preliminary tests established that the antigen M_1 is commoner in Negroes than in Whites: they also showed that there is considerable variability in the strength of the antigen and that the classification of the weaker reactions may be left in doubt. Because of this difficulty we decided to confine the present report to classifications based on the results of rather laborious quantitative tests in which strictly comparable cell samples were used. When better anti-M_1 sera are available we hope it will be possible to classify routine cell samples without using quantitative methods.

Samples of blood from 32 New York Negroes and 32 Whites were taken on the same day at the Knickerbocker Foundation and sent by air to London. There they were tested against titrations of six sera containing anti-M and anti-M_1 in different proportions, and against titrations of three anti-N sera, one anti-S and one anti-s. The results given in Table 1 indicate that M_1 is about four times more frequent in Negroes than in Whites.

Table 1. Results of Testing Samples of Blood from 32 Negroes and 32 White People with Anti-M_1

		Negroes			Whites		
		M	MN	N	M	MN	N
Anti-M_1	+	3	5	0	2	0	0
	−	4	12	8	8	15	7

A genetic background for these antigenic differences can be provided most simply by postulating three alleles, M_1, M_2 and N, the approximate frequencies of which are:

	M_1	M_2	N
Negroes	0·13	0·35	0·52
Whites	0·03	0·52	0·45

Recombined, these figures give expectations in the five phenotype (six genotype) classes which agree very closely with the numbers observed in both the Negro and the White series. This, to say the least, does not contradict the hypothesis that two kinds of *M* alleles are responsible for the M_1–M_2 antigenic difference; but we hope for more direct support when appropriate families can be tested.

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