The results of blood groupings and M and N typings are given in Table 1. For comparative purposes, the data for three other races are included.

TABLE 1

	White	Colored[5]	Chinese[3]	Japanese*
	Per cent.	Per cent.	Per cent.	Per cent.
O	45.0	47.1	30.0	26.0
A	41.0	28.2	34.0	40.0†
B	10.0	19.6	25.3	23.3
AB	4.0	5.1	10.7	10.7
M	29.2	29.0	23.3	30.0
N	21.2	28.3	22.0	16.6
MN	49.6	42.8	54.7	53.4

* This study.
† Among the Japanese bloods, the ratio of A_1 to A_2 was 5:1 if the intermediate type was calculated as A_2.

The anti-Rh sera employed in this study are in the order of the frequency of positive reactions in a random white population, anti-Rh_0, anti-Rh′ and anti-Rh″. The data obtained with these three anti-Rh sera are recorded in Table 2, in which the results for the Japanese as well as for the three other racial groups are given.

TABLE 2

	White	Colored	Chinese	Japanese*
	Per cent.	Per cent.	Per cent.	Per cent.
anti-Rh_0	85–87	95.5 (L) 92.0 (W)	99.3	98.0
anti-Rh′	70–73	46.0 (L) 29.2 (W)	93.0	85.4
anti-Rh″	30	29.2 (W)	no data	61.4

(L) indicates data of Levine[2]; (W) indicates data of Wiener.[5]
* This study.

The anti-Rh″ serum was generously supplied by Dr. Wiener. In accordance with his suggestion, this serum was diluted 1:5 in order to remove the effects of a concomitant agglutinin of another specificity. However, in our hands, it was necessary to interpret some of the weaker reactions, such as ±, as negative with the anti-Rh″ agglutinin.

From a practical standpoint, the most significant finding is the exceedingly low incidence of negative reactions with the anti-Rh_0 serum among the Japanese. As in the case of the Chinese, one may conclude that the incidence of erythroblastosis fetalis and of intragroup transfusion reactions should be very low among Japanese as compared to the white race. This is amply supported by the scarcity of reports of these conditions in the Japanese medical literature.

The findings with the Japanese bloods on the basis of the several antigenic components of the Rh factor are presented in Table 3 along with the corresponding data for white and colored races recently published by Wiener.

TABLE 3

Reactions with anti-Rh_0	anti-Rh′	anti-Rh″		White	Colored	Japanese
+	+	+	Rh_1Rh_2	19.2	7.3	47.25
+	0	+	Rh_2	14.8	21.9	13.35
+	+	0	Rh_1	51.2	21.2	37.40
+	0	0	Rh_0	2.2	41.6	0.00
0	+	+	Rh′Rh″	0.0	0.0	0.66
0	0	+	Rh″	0.3	0.0	0.00
0	+	0	Rh′	0.8	0.7	0.00
0	0	0	Rh neg.	11.4	7.3	1.34

The terminology of the Rh antigens given above is that recommended by Wiener.[6]

Aside from the very low incidence of Rh negative individuals among Japanese already mentioned, the most significant finding is the absence of the type Rh_0 which is so frequent in the colored race. In striking contrast is the high incidence of the phenotype Rh_1Rh_2 in Japanese as compared to that in the white and colored races.

Almost identical differences in white and colored individuals were observed by Levine[7] in tests with an active anti-Rh″ agglutinin produced by an Rh_1 individual. In tests with a potent anti-Hr serum all Rh_1Rh_2 bloods gave negative reactions. About 60 per cent. of Rh_1 bloods of white individuals and almost all colored individuals tested possessed the Hr factor.

These preliminary findings serve to illustrate the importance of comprehensive racial studies which could be carried out as soon as all varieties of anti-Rh sera become readily available. It can be expected that the final theory of the heredity of the Rh antigenic complex will emerge from or be confirmed by the statistical analysis of the various phenotypes in many racial groups, as in the case of the four blood groups.

ROBERT K. WALLER
PHILIP LEVINE

ON THE SPECIFICITY OF CHOLINE ESTERASE IN NERVOUS TISSUE[1]

RECENT investigations have provided evidence for a new concept of the role which acetylcholine may have in the mechanism of nervous action.[2, 3] The release and the removal of the ester are considered as an intracellular process directly connected with the nerve action potential at points along the neuronal surface. The ester released by a stimulus depolarizes the membrane by rendering it permeable to all ions. Thus, flow of current is generated (action potential) which

[6] A. S. Wiener, SCIENCE, 99: 532, 1944.
[7] P. Levine: Unpublished data.
[1] This work was aided by grants of the Josiah Macy, Jr., and Dazian Foundations.
[2] J. F. Fulton and D. Nachmansohn, SCIENCE, 97: 569, 1943.
[3] D. Nachmansohn, R. T. Cox, C. W. Coates and A. L. Machado, *Jour. Neurophysiol.*, 5: 499, 1942, and 6: 383, 1943.

stimulates the adjacent region. There the process is repeated, and the impulse in this way propagated along the axon. At the nerve ending, owing to the increased surface, there is less resistance and more flow of current enabling the impulse to cross the non-conducting gap. The flow of current is the transmitting agent.

The new concept is based, to a large extent, on studies of the enzyme choline esterase. Three essential features are: (*i*) the high concentration of the enzyme in nerves. The concentration is sufficiently high to make possible a rate of acetylcholine metabolism which parallels that of the electric changes. (*ii*) The localization of the enzyme at the neuronal surface where the bioelectric phenomena occur, and (*iii*) the parallelism between the enzyme activity and the voltage of the nerve action potential developed by the electric organ. In these experiments, it was assumed that the enzyme is specific, *i.e.*, that the substrate metabolized is acetylcholine, the release of which during nerve activity has been demonstrated at several instances.

Evidence has now been obtained that the enzyme present in various nerve tissues, on which the role of acetylcholine in the mechanism of nervous action has been studied, is a specific enzyme. It differs essentially from the esterases present in other tissues or serum in which, as shown by Stedman, Stedman, and Easson,[4] only a fraction is specific choline esterase. Whatever nervous tissue is used the enzyme present has the same typical properties. It is unable to split carbaminoylcholine and benzoylcholine. That the latter compound is not split by brain esterase was already observed previously.[5] It splits butyrylcholine at a much lower rate than acetylcholine. On the other hand, acetyl-β-methylcholine which is not hydrolyzed by most other tissues is split by nerve esterase, although at a rate 20 to 40 per cent. lower than acetylcholine. Table 1 gives a few examples of significant data chosen among a number of substrates and tissues tested. The results thus provide further essential support for the new concept of the role of acetylcholine in the mechanism of nervous action.

TABLE 1

RATE OF ESTERASE ACTIVITY OF DIFFERENT TISSUES COMPARED TO THAT OF ACETYLCHOLINE (= 100)

Species	Tissue	Benzoyl = Choline	Butyryl = Choline
Rat	Brain	0	20
Squid	Ganglion	0	24
(Loligo paealii)	Fiber	0	48
Lobster (Homarus vulgaris)	Ab. chain	0	15
Electric eel (Electrophorus electricus)	Electric organ	0	3
Torpedo	" "	0	0
Rabbit	Liver	31	130
Guinea-pig	Pancreas	65	200
" "	Kidney	200	350
Human	Serum	60	272

[4] E. Stedman, E. Stedman and L. H. Easson, *Biochem. Jour.*, 26: 2056, 1932.

[5] B. Mendel, D. B. Mundell and H. Rudney, *Biochem. Jour.*, 37: 473, 1943.

This concept has been criticized by Lorente de Nó[6] because he is unable to find an effect on the frog's sciatic when put into concentrated acetylcholine solution. It is extremely difficult to reproduce an *intracellular* process by adding a compound to a living cell in a test-tube (see insulin). The failure to produce such an effect is therefore meaningless and can not be considered as contrary to a concept based on an impressive body of biochemical and physiological evidence. Acetylcholine is a quaternary ammonium base. Such compounds usually do not permeate the living cell membrane. Eserine is a tertiary ammonium base which as a free base may enter the cell. With eserine, Lorente de Nó finds a depolarization of the nerve—a point quite consistent with the mechanism suggested.

DAVID NACHMANSOHN
MORTIMER A. ROTHENBERG
COLLEGE OF PHYSICIANS AND SURGEONS,
COLUMBIA UNIVERSITY

BRAIN-WEIGHT AND BODY-WEIGHT IN THE RED SPIDER MONKEY

IN recent compilations of the brain and body weights of primates[1,2] the Cebidae are represented as having the highest brain to body weight ratio of any of the primates including man. The coefficients of cephalization computed for them are correspondingly high.

The recorded data are chiefly those of Hrdlička,[3] and the body weights which he gives for Cebus and for Ateles are very much less than the weights of adult animals for those genera with which we are familiar. Table 1 gives the weights of 19 adult red

TABLE 1

WEIGHTS OF 10 ADULT RED SPIDER MONKEYS

Males	Females	
5.19 kg	4.27 kg	5.36 kg
6.63	4.82	5.39
6.89	4.85	5.42
	5.08	5.78
	5.13	5.81
	5.22	5.87
	5.22	5.96
	5.25	6.01

[6] R. Lorente de Nó, *Jour. Cell. Comp. Physiol.*, 24: 85, 1944.

[1] S. Zuckerman, "Functional Affinities of Man, Monkeys, and Apes." New York: Harcourt, Brace and Co., 1933.

[2] G. von Bonin, *Jour. Psychol.*, 16: 379–389, 1937.

[3] A. Hrdlička, *Amer. Jour. Phys. Anthrop.*, 8: 201–211, 1925.