Rh SUBGROUPS IN SOUTH INDIANS

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THE incidence of the Rh factor in Indians has been worked out by various authors. Most of the investigators have used anti-Rh serum of anti-D specificity only. Wiener[1] and Greval[2] tested Indians with anti-D, anti-C and anti-E sera. Ranganathan *et al.*[3] tested 294 South Indians with anti-D, anti-C and anti-c sera. Prasad *et al.*[4] tested 105 Indian students in London with anti-C, anti-D, anti-E, anti-c and anti-e sera. The present investigation has been undertaken with a view to get sufficient data about the distribution of the Rh groups in South Indians.

The tests were carried out on 100 medical students. The red cells were tested with anti-Rh sera, anti-D, anti-C, anti-E and anti-c sera. Dried sera and preserved liquid sera were employed. Landsteiner's tube method was used for the tests. The results were read macroscopically and confirmed by microscopic examination. Reliance was placed only on the microscopic reaction because on a number of occasions a negative macroscopic reading gave an unmistakable presence of agglutinates when viewed under the microscope and a positive macroscopic reading on rare occasions showed no agglutination under the microscope. The specimen of cells which gave a negative reaction with the anti-D serum was retested for confirmation.

The results obtained with the various antisera have been tabulated in Table I which includes the results obtained by Prasad *et al.*[4] for Indians, and the results for Hebrews in Canada[5] for comparison.

TABLE I
Incidence of Rh subgroups

Reactions with anti-sera				South Indians (present study)	Indians (Prasad *et al.*)[4]	Hebrews[5]
Anti-C	Anti-c	Anti-D	Anti-E			
+	+	+	−	30	32·38	35·00
+	−	+	−	32	35·24	29·29
−	+	−	−	8	7·62	7·86
−	+	+	+	6	3·81	5·00
+	+	+	+	20	16·19	17·14
−	+	+	−	3	2·86	3·57
−	+	−	+	0	..	0·00
+	+	−	−	0	1·90	1·43
−	−	+	+	1	..	0·71
+	+	−	+	0	..	0·00
+	−	−	−	0	..	0·00
Percentage of positives in South Indians with the sera :						
82	67	92	27	100	100·00	100·00

Table II shows the Rh chromosome frequencies of South Indians compared with those obtained by Prasad *et al.* for Indians.

TABLE II
Rh Chromosome frequencies expressed as percentages

Chromosome	South Indians (present investigation)	Indians (Prasad *et al.*)[4]
CDe	56·57	56·64
cde	28·28	24·77
cDE	9·40	10·48
cDe	4·88	4·27
CDE	0·87	0
Cde	0	3·85

Our results show a frequency for D-negative of 8 per cent The figures obtained by other workers vary from 2 to 10 per cent. as pointed out by Prasad *et al.* The variation might be due to different workers testing different populations, and probably also due to technical errors. The chromosome frequencies obtained by us agree closely with those obtained by Prasad *et al.* except for Cde and CDE.

When the distribution of the phenotypes in South India is compared with the incidence of phenotypes in the different races in the world,[5] there is a striking resemblance with the phenotypes in Hebrews in Canada while some similarities and some differences occurred with some other races. We are not able to explain this interesting feature as we have insufficient ethnological data.

When the Kahn tubes after the tests were soaked in 1 : 1,000 nitric acid overnight and then cleaned in tap water, rinsed in distilled water and autoclaved for use, washed red cells in saline suspension in such tubes showed clumping even without the addition of the stock serum, due evidently to trace impurity of serum sticking to the wall of the narrow tube. But when a saline suspension containing unwashed cells was put into the tube no agglutination occurred. These were consistently seen in several observations spread over some days. This leads us to suspect that the serum may contain an inhibitor factor against agglutination, and porbably when this is present in high

concentration the prozone phenomenon occurs. Further work on the suspected inhibitor factor is in progress. Tubes soaked overnight in dichromate sulphuric acid mixture, brushed, cleaned in tap water and rinsed in distilled water and .autoclaved did not give any false reactions.

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1. Wiener, A. S., *et al., J. Immunol.*, 1945, **50**, 341. 2. Greval *et al., Nature*, 1946, **157**, 411. 3. Ranganathan *et al., J. Ind. Med. Assoc.*, 1948, **17**, 162. 4. Prasad *et al., Am. J. Phy. Anthr.*, 1949, **7**, N.S. No. 4, 553. 5. Race and Sanger, "Blood Groups in Man," Table 28, *Blackwell Scientific Publications*, Oxford, first edition, 1950.

NOBEL AWARD FOR MEDICINE AND PHYSIOLOGY—1953

THE NOBEL PRIZE FOR MEDICINE AND PHYSIOLOGY for 1953 has been awarded jointly to Prof. H. A. Krebs, Professor of Physiology in the University of Sheffield and Director of the Medical Research Council Unit for Research in Cell Metabolism, and Dr. F. Lipmann, Head of the Biochemical Research Laboratories, Massachussetts General Hospital.

Prof. Krebs has mostly been concerned with the study of metabolic processes by experiments *in vitro*. The first of his two greatest contributions to biochemistry was from Freiburg in 1932, when he elucidated the mechanism of urea synthesis in the liver, by discovering the participation of ornithine, citrulline and arginine through a cyclical process—a concept of unprecedented nature His subsequent observations on the deamination of amino acids demonstrated D-amino acid oxidase, and laid the foundations for future studies of the L-acids. In Cambridge in 1935 he proved the synthesis of glutamine from glutamic acid and ammonia in tissue slices After moving to Sheffield, he announced in 1937 his second major contribution, the citric acid cycle. Before this, the path of oxidation of carbohydrates from pyruvate onward was unknown; although information was available, its significance was not appreciated. Krebs supplied the missing evidence and the idea, again that of a cycle, and the problem was solved The citric acid cycle has stood the test of time, requiring only amplification of detail; it is concerned with oxidation of fat and protein as well as carbohydrate, provides paths for gluconeogenesis and amino acid synthesis, and is the chief source of metabolic energy. In recent years, Krebs's laboratory has been particularly concerned with the movement of substances across biological membranes, and he has studied the uptake of glutamic acid and potassium by tissue cells, processes driven by energy from metabolism. He has also employed isotopes in the quantitative investigation of oxidative phosphorylation and ion transfer.

After studying the problems of muscle metabolism in Meyerhof's laboratory and of fermentation in Carlsberg Laboratory, Lipmann set the pattern for his future work when in 1937 he began to analyse the oxidation of pyruvate to acetate by bacteria. He found that the oxidation is accompanied by phosphorylation, and announced from Cornell University in 1939 that the 'energy-rich' ester acetyl phosphate is an intermediate His celebrated article, "Metabolic Generation and Utilisation of Phosphate Bond Energy" (1941) organized and developed existing ideas and had the most profound influence on subsequent biochemical thought and research. Moving to Boston, Lipmann realized that acetyl phosphate is not formed in pyruvate oxidation in animal tissues; some other substance had to be sought By a happy coincidence his own researches led to its identification. Studying the biological acetylation of sulphanilamide, he discovered a new coenzyme, coenzyme A (coenzyme of acetylation). Finding that it is a derivative of the vitamin pantothenic acid and a general constituent of living organisms, he quickly realized that it is of fundamental importance in carbohydrate and fat metabolism. By 1946 it was clear that some acetyl derivative stood at the entry to the citric acid cycle, on the paths of carbohydrate and fat oxidation and ketone-body formation. The idea grew that this was the acetyl derivative of coenzyme A, and was confirmed after the isolation of this substance from yeast by Lynen and Reichert in 1951. In the meantime Lipmann persevered in the purification of coenzyme A and as a result its structure has recently been settled.—(*By courtesy of Nature.*)