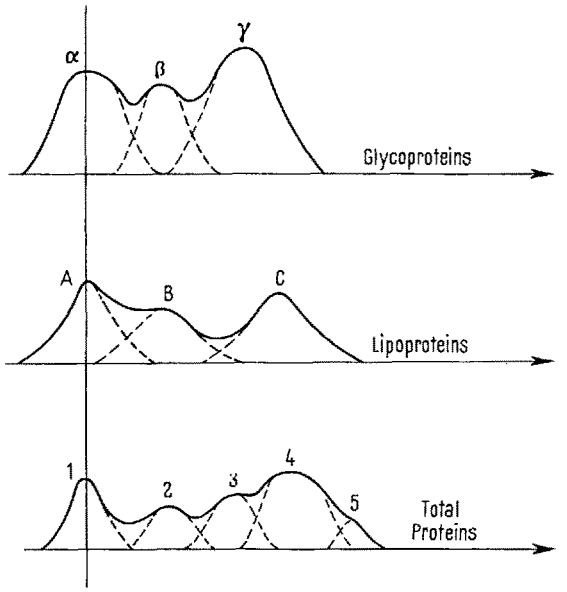
the pattern of the soluble proteins from rat liver mitochondria is, however, different from that of rat muscle sarcosomes as recently reported by DIANZANI MOR[8]. The lipoprotein pattern consists of at least 3 components. The first of them (A) occupies on the strip the same position as peak No. 1 of the total protein pattern. The second one (B) occupies the same position as peak No. 2; the position of the third one (C) corresponds to that of peaks No. 3 and 4. Relative percentages of lipoprotein components were respectively: 37·1 ± 7·9 for A, 32·2 ± 4·7 for B, and 30·7 ± 5·3 for C.



Total, lipo-, and glycoprotein diagrams of rat liver mitochondria extracts. The values on the abscissa represent the translation in mm; those on the ordinata represent the photometric absorption measurements. A factor 4 was applied in the case of glycoproteins.

The glycoprotein pattern consists of 3 peaks. Their position on the strip corresponds to that of lipoproteins peaks. Relative percentages were: 27·4 ± 5·7 for α, 26·3 ± 4·4 for β, and 46·2 ± 1·4 for γ.

In order to investigate the possible influence of sucrose on the concentration of glycoproteins in the mitochondrial extract, some experiments were run in which mitochondria were isolated from homogenates prepared with 0·125 *M* KCl instead of 0·25 *M* sucrose. No difference was found with respect to the mitochondria isolated from sucrose medium.

G. UGAZIO

Istituto di Patologia generale, Università di Cagliari (Italy), January 30, 1960.

Riassunto

Mediante elettroforesi su carta si dimostra la presenza nei lisati di mitocondri di fegato di ratto di almeno 5 componenti proteici. Le colorazioni specifiche dimostrano la presenza di 3 frazioni lipoproteiche e di altrettante glicoproteiche. I lisati dei mitocondri vennero ottenuti qer trattamento delle particelle con Triton X-100.

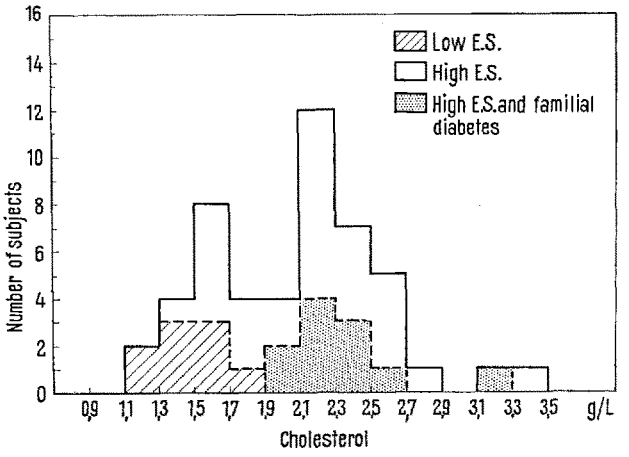
Normal Values of Plasma Potassium, Sodium, Cholesterol and Proteins and of Blood Glucose in South Indian People, in Reference to Western Standards

No systematic studies have been made so far to compare the normal blood composition of South Indian people with western standards, used as a reference. The present work is a preliminary investigation in this field.

Venous blood was taken before breakfast from 49 male Indians of Madras, all students or physicians, except for 9 who were servants of low economic status. Plasma, separated from heparinized blood by centrifugation, was analyzed for K and Na by flame photometry, for cholesterol by CRAWFORD's modification of the method of ZAK[1] and for protein by KJELDAHL. Blood glucose was determined by the FOLIN and WU method[2]. A few determinations were also made with 11 males of the white race (European or American) living in Madras.

Results are summarized in the Table. The individual values are spread symmetrically around their mean, and no significant difference is observed between Indian students or physicians, Indian servants, and white people, except for cholesterol. As shown in the Figure, these latter values are divided into two groups. The 9 Indians of low economic status are in the group of lower values, while eleven people with a familial diabetic background are all in the higher group. This distribution is statistically significant.

Our results fall in the normal range of western standards. Nevertheless, a group of 20 male Belgians of the same mean age as our Indian population show K values significantly lower (15.1 ± 1.59 mg %; HENROTTE[3]). The similarity of K values in Indians and Westerns in Madras



Distriubtion of cholesterol values (in g/l) in the Indian population. Low E. S. = Indian servants of low economic status. High E. S. = Indian students or physicians of high economic status. High E. S. and familial diabetes = same as the latters but revealing the occurrence of one diabetic or more in their family.

[1] W. CRAWFORD, Clinica chim. Acta *3*, 357 (1958).
[2] O. FOLIN and H. WU, J. biol. Chem. *41*, 367 (1920).
[3] J. G. HENROTTE, unpublished results.
[4] W. RADSMA, Festschrift Nocht (Hamburg 1937), quoted in H. C. FRIEDMAN, Lancet *2*, 262 (1954).
[5] J. LESCHI, *Races mélanodermes et leucodermes*, Thèse Sciences 1951 (Masson, Paris 1952).
[6] J. G. HENROTTE, G. KRISHNAMURTHI, and G. RANGANATHAN, to be published in Nature.
[7] W. S. SPECTOR, *Handbook of Biological Data* (W. A. Saunders Cy 1956).

	Indians				*Westerns*			
	m	σ	E. V.	*n*	*m*	σ	E. V.	*n*
K, mg %	17.90	1.54	21.23– 14.68	45	17.80	1.36	19.60– 15.88	9
Na, mg %	335.6	14.9	364.7 –302.0	47	334.7	14.3	350.0 –327.5	11
Glucose, mg %	92.01	10.48	115.0 – 76.0	41	91.0	6.12	100.0 – 83.0	10
Cholesterol, mg %								
total	206.8	46.8	318.0 –129.0	49	221.0	50.4	299.0 –134.0	11
lower group	156.0	19.0	180.0 –129.0	18	—	—	—	—
higher group	236.0	29.5	318.0 –190.0	31	—	—	—	—
Proteins, g %	7.56	0.45	8.33– 6.66	44	7.36	0.40	7.92– 6.74	11
Age, years	23.4	5.85	38 – 18	72	31.9	6.5	45 – 25	11

m = mean; σ = standard deviation; E.V. = extreme values; *n* = number of subjects; % = 100 per cm³

indicates that the slight hyperpotassemia observed in Madras by comparison with our European series depends upon a climatic factor, as confirmed by RADSMA[4] in Indochina. However, in Dakar, LESCHI[5] finds higher K values in Negroes than in western people and suggests the racial origin of this difference. The hyperpotassemia found in tropical countries might be in relation to a lower adrenal gland activity (HENROTTE *et al.*[6]).

On the other hand, the low cholesterol group of Indians has values much inferior to our results and to western data (195 to 230 mg%, SPECTOR[7]; 233 ± 40 mg%, HENROTTE[3]). Age and food habit (vegetarian or non-vegetarian diet) are similar in the two Indian groups and cannot explain their difference of cholesterolemia. Only 6 of the 9 servants have a diet poorer in calories and fats. Possibility of a racial factor in this matter is under further investigation.

Our research also indicates a correlation between high plasma cholesterol and diabetic familial background.

We acknowledge with thanks the technical facilities provided for our work by Professor K. P. ANANDAN and N. VERGHESE.

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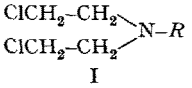
Department of Physiology and Biochemistry, Madras Medical College, Madras, and Laboratoire de Pathologie et Thérapeutique générales, University of Liège (Belgique), le 21 mars 1960.

Résumé

La composition sanguine d'une cinquantaine d'Indiens du Sud, mâles, adultes et en bonne santé, est comparée aux standards occidentaux. Les valeurs de glucose sanguin, protéines et sodium plasmatiques des Indiens sont semblables à celles des Occidentaux, leur potassémie est plus élevée et la cholestérolémie montre un groupe de valeurs très basses. La signification de ces différences est brièvement discutée.

Propos sur une nouvelle moutarde azotée

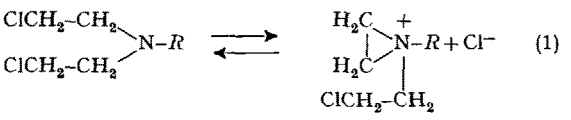
Il est bien connu que les moutardes azotées (I) ont une action cytostatique nette. Malheureusement, la toxicité de ces composés en rend le maniement très délicat.



I

On a cherché à synthétiser des moutardes azotées dont l'action cytostatique ainsi que la toxicité soient dissimulées sous un «masque» qui puisse tomber plus facilement dans les cellules cancéreuses que dans les cellules normales de l'organisme. Mais, jusqu'à présent, les résultats ne sont pas satisfaisants.

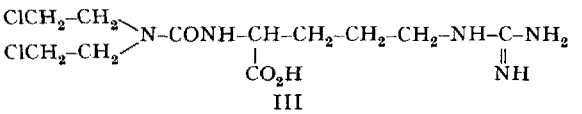
Il est bien connu que la molécule de moutarde azotée est en équilibre avec un ion de cyclodiméthylène-ammonium (II), et que l'activité de la moutarde azotée est due aux réactions du ion II avec les groupements actifs des protéines (NH_2, SH, CO_2H).



I II (1)

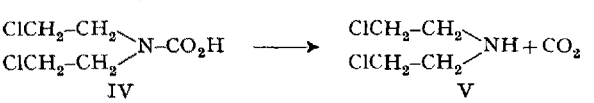
Donc, la moutarde azotée sera active dans la mesure que l'équilibre (1) sera déplacé vers la droite. Cet équilibre peut être rétrogradé si on fait diminuer la basicité de l'atome d'azote, en formant un groupement carbonamide.

La N-α-di(β-chloréthyl)-carbamyl-L-arginine (III) formera *si difficilement un ion du type II, qu'elle sera pratiquement dépourvue tant d'action cytostatique que de toxicité.*



III

Mais dans l'organisme, sous l'action de l'amidase appropriée, III sera scindé en L-arginine et en acide di(β-chloréthyl)-carbamique (IV), composé extrêmement instable, qui se décarboxylera immédiatement en passant en di(β-chloréthyl)-amine (V). Or, V est extrêmement actif et pas tellement toxique.



IV V

Lorsque l'activité amidasique du tissu néoplasique est bien plus intense que celle du tissu sain correspondant[1,2] dans les cellules cancéreuses il se formera toujours une quantité bien plus élevée de composé V que dans les cellules normales de l'organisme.

La teneur en L-arginine du tissu néoplasique est supérieure à celle du tissu sain correspondant. La L-arginine accroît l'index mitotique du tissu néoplasique, mais ne modifie pas l'index mitotique du tissu sain[3]. Par consé-

[1] J. P. GREENSTEIN and F. M. LEUTHARDT, J. nat. Cancer Inst. *8*, 77 (1947).

[2] P. C. ZAMECNIK and M. L. STEPHENSON, Cancer Res. *7*, 326 (1947).

[3] S. J. BACH and I. LASNITZKI, Biochem. J. *40*, xlviii (1946); Enzymologia *12*, 198 (1947).