Résultats. En ce qui concerne l'utilité des trois différents sels de tétrazolium, nos résultats confirment ceux des auteurs précédents. En effet, l'INT, bien qu'il se réduise très facilement, donne lieu à la formation de cristaux grossiers de monoformasan rouge brique et ne rend possible qu'une localisation histochimique. En revanche, les fins dépôts granuleux du produit de réduction de NT et du nitro-BT permettent une localisation cytochimique. Avec ces deux derniers ditétrazoliums, nous avons pu observer, par places, une coloration rouge dont la signification n'est pas élucidée.

Quant à la distribution des formosans dans les différentes parties du tissu oculaire, nous avons obtenu les résultats suivants:

Cornée: Dans la cornée, c'est l'épithélium et l'endothélium qui ont montré une activité enzymatique importante (Figure 1a, b). Dans l'épithélium, nous avons pu constater une activité plus intense dans les couches basales, activité qui diminue progressivement vers les couches superficielles (Figure 1a). Dans le parenchyme, seuls les éléments cellulaires ont donné un résultat plus ou moins positif.

Corps ciliaire: Au niveau du corps ciliaire, une grande quantité de formasan a pu être mise en évidence dans le muscle ciliaire, d'une part, et dans le protoplasme de l'épithélium, d'autre part. Quelquefois nous avons pu constater une image striée au niveau du muscle ciliaire. Cet aspect pourrait correspondre au fait que l'activité déhydrogénasique est absente au niveau des ondes contractiles des fibres ciliaires, tandis qu'elle est maximale à proximité de celles-ci (DICULESCO et al.[7]).

Cristallin: Dans le cristallin, l'activité succino-déhydrogénasique s'est limitée à une mince couche superficielle englobant le reste du cristallin. Dans cette couche superficielle, la capsule du cristallin s'est révélée négative, tandis que l'épithélium faisait preuve d'une activité prononcée (Figure 2). Cette activité ne s'est toutefois pas exclusivement bornée à l'épithélium. En effet, les cristaux de formasan ont été retrouvés dans les fibres superficielles de l'écorce cristallinienne. Néanmoins, à cet endroit, la spécificité de la réaction peut être mise en doute.

Rétine: Une très forte réaction peut être observée dans la rétine au niveau de l'article interne des éléments photorécepteurs. En outre, une très faible activité peut être mise en évidence dans les couches des cellules ganglionnaires, ainsi que dans la couche réticulaire interne. Les résultats obtenus chez l'homme et chez le lapin sont identiques.

Ces expériences, dont nous avons donné un bref aperçu, confirment et complètent les résultats obtenus par d'autres auteurs au cours de l'étude de l'activité déhydrogénasique dans le tissu oculaire.

Summary. The author has studied, with different tetrazolium techniques (NT, INT, nitro-BT), the succinate dehydrogenase activity in the ocular tissue (cornea, ciliary body, lens, retina).

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Clinique ophtalmologique de l'Université de Genève (Suisse), le 19 mars 1962.

[7] J. DICULESCO, J. VERNE et R. WEGMANN, Ann. Histochem. *6*, 47 (1961).

Asthma and Mast Cells of Bronchial Connective Tissue

We have already demonstrated in previous studies that the bronchial connective tissue of man is rich in mast cells, mostly with abundant metachromatic granulations; and that opposite to such abundance in normal man, or in subjects affected with non-asthmatic bronchitis, there is an evident scarseness in asthmatics, in whom the rare mast cells still visible are mostly degranulated or disrupted with the metachromatic granules scattered in the ground substance of the connective tissue[1–4].

In the present study, we report the findings of a comparative research on the distribution and on the reactivity of bronchial mast cells to the compound 48/80 in the following two groups: (1) patients affected with pure bronchial asthma; and (2) patients affected with non-asthmatic bronchitis.

The mast cells were observed in bronchobiopsy specimens taken from the orifice of the middle lobe bronchus, stained with toluidine blue 01% at pH 6.5. In all cases, a first specimen was taken before treatment with the histamine liberator 48/80 and a second one from 3 to 30 h after the last administration, taking care to excise it far from the first cicatrice. The compound 48/80 was administered intravenously in daily doses proportional to body weight, progressively increasing from an initial minimum of 10–35 μg/kg to a maximum of 35–60 μg/kg. To both groups the same total quantity of 48/80 was administered, used in a strength of 1 mg/ml of physiological saline.

Counting of mast cells for each patient was made in 30 microscopical fields for each one of the 5 histological sections examined, using ocular × 10 and objective × 100. The average number of mast cells visible in the area of 30 microscopical fields of each group was obtained dividing the total number of mast cells counted for each group by 55 (number of histological sections examined for each group).

Tab. I

		Mast cells	Mean number of mast cells in 30 microscopical fields	% value
Asthmatics (11 cases)	before	non-degranulated	14.24	26.29
		degranulated	39.91	73.71
		total population	54.15	100.00
	after 48/80	non-degranulated	3.89	12.56
		degranulated	27.09	87.44
		total population	30.98	100.00
Non-asthmatics (11 cases)	before	non-degranulated	40.80	55.81
		degranulated	32.31	44.19
		total population	73.11	100.00
	after 48/80	non-degranulated	20.55	41.08
		degranulated	29.47	58.92
		total population	50.02	100.00

[1] G. SALVATO, Minerva Med. *49*, 2868 (1958).
[2] G. SALVATO, Exper. *15*, 308 (1959).
[3] G. SALVATO, Allergie Asthma *6*, 22 (1960).
[4] G. SALVATO, Int. Arch. Allergy *18*, 348 (1961).

Tab. II. Analysis of variance

Source of variation	Degrees of freedom	Deviance	Variance	F	Critical values of F for $P < 0.01$
Between asthmatics and non-asthmatics	1	19855.00	19855.00	37.32	6.76
Between before and after 48/80	1	29418.47	29418.47	55.30	6.76
Interaction	1	0.53	0.53	0.000996	6.76
Experimental error	216	114906.00	531.97	—	—
Total	219	164180.00	—	—	—

The results reported in Table I show that this average number was higher in non-asthmatics (73.11) than in asthmatics (54.15) with a percentage of degranulated cells respectively 44.19% and 73.71%. After treatment with the histamine liberator 48/80, the average number of mast cells decreased both in non-asthmatics (50.02) and in asthmatics (30.98); the percentage of degranulated cells rose in both groups (non-asthmatics: 58.92%; asthmatics: 87.44%).

The significance of the data thus obtained was verified using the *variance-ratio F test.* As shown in Table II, it is practically certain that the diverse distribution of mast cells in the two groups of patients is due to systematic factors connected with bronchial asthma. The F value was in fact greater than the value corresponding to $P < 0.001$: the probability of a casual divergence between the average numbers of mast cells in asthmatics and in non-asthmatics is therefore lower than one per thousand. The difference between the average numbers of mast cells before and after treatment was also still significant at the level $P < 0.001$. A higher reactivity of mast cells to the compound 48/80 in asthmatics was, on the other hand, excluded with practical certainty by probabilistic analysis ($F = 0.000996$).

This study confirms the findings of our previous work, i.e. that the numerical reduction and the degranulation of mast cells are a characteristic phenomenon of the hyperergic-patergic reactivity of bronchi in asthmatics; these alterations are absent, or of much lower intensity, in normergic inflammation characteristic of non-asthmatic bronchitis.

The reliability of these conclusions is supported by the rigorous method applied. The significance of sampling is validly proved by its homogeneity: in fact, on drawing a frequency distribution curve of the quantities of mast cells, a nearly perfect Gaussian curve was obtained[5].

Riassunto. Lo studio comparativo dei mastociti bronchiali in 2 gruppi di pazienti: (1) bronchitici non-asmatici, (2) asmatici, dimostrò che in questi ultimi le «Mastzellen» erano meno numerose e prevalentemente degranulate. Il 48/80 produsse nei 2 gruppi diminuzione cellulare e degranulazione. Il *test F* dimostrò l'alta significatività dei suddetti reperti, mentre esluse una interazione tra asma e reazione al farmaco.

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City Hospital, Merano (Italy), March 16, 1962.

[5] Thanks are due to Prof. I. SCARDOVI, statistician of Bologna University, for his kind co-operation in ascertaining the probability of the experimental data.

Effect of Dibenzyline on Serum Cholesterol in Rabbits[1]

Much interest has been shown in the hormonal regulation of lipid metabolism. Amongst other problems which have received attention, is that of the influence of sympathomimetic drugs on serum cholesterol. Animals treated with adrenalin or serotonin show often an increased level of serum cholesterol[2–6]. On the other hand, it has been reported that some ganglion blocking agents, used for treatment of hypertension, not only lower blood pressure but also depress the level of serum cholesterol[7,8]. Our experiments on rabbits with adrenergic substances (serotonin and nor-adrenalin) and with a ganglion blocking substance (Dibenzyline), gave different results in short term tests. In view of this discrepancy the following observations are believed worth reporting.

Method and Results. In the present experiments adult healthy rabbits of either sex were used. Nor-adrenalin bitartarate was given intramuscularly, 0.6 mg in an aqueous solution once a day for 2 days to 26 rabbits. 20 mg of serotonin were given intramuscularly to 12 animals in an aqueous solution of 5-hydroxytryptamine creatinine sulfate once a day for 2 days. Dibenzyline (N-phenoxyisopropyl-N-benzyl-β-chlorethylamine hydrochloride, Smith, Kline & French Labs., Phila.) in doses of 2.5 mg/kg of body weight was injected into the ear vein of 17 animals. The intravenous injections were given slowly, over periods of 20 min. When signs of blocking appeared (as hypersalivation, enophthalmos, etc.), the speed of the injection was still more decreased. Notwithstanding this precaution, all the animals were weak and listless at the end of the injection, but recovered within 2 h. One rabbit died during the injection, and one other rabbit showed

[1] This work was done under a Fellowship of the Canadian Life Insurance Officers Association.

[2] A. DURY and L. D. MOSS, J. Gerontol. *9*, 287 (1954).
[3] A. KAPLAN and M. GANT, Fed. Proc. *14*, 82 (1955).
[4] P. T. WERTLAKE, A. A. WILCOX, M. I. HALEY, and J. E. PETERSON, Proc. Soc. exp. Biol. Med. *97*, 163 (1958).
[5] E. SAFRIR, K. E. SUSSMAN, and D. STEINBERG, J. Lipid Res. *1*, 109 (1959).
[6] M. FRIEDMAN and S. O. BYERS, Amer. J. Physiol. *199*, 995 (1960).
[7] Q. B. DENING, M. E. HODES, A. BALTAZAR, J. G. EDREIRA, and S. TOROSDAG, Amer. J. Med. *24*, 882 (1958).
[8] A. C. LEVY and E. R. RAMEY, Proc. Soc. exp. Biol. Med. *99*, 637 (1958).