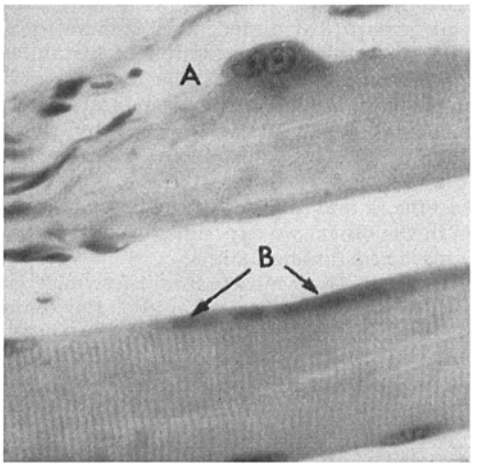
tissue histamine[3] led to this investigation of the effects of the drug on skeletal muscle.

These experiments have demonstrated that skeletal muscle fibers from mice treated with histamine are consistently smaller in diameter than those from control animals (Tables I and II). Basophilic sarcoplasm in fibers and blister-like evaginations containing groups of nuclei which were present in treated mice were never seen in control animals. Some of the histological changes which were seen in histamine-treated mice have been described in mice of the same strain (strain 129) with hereditary muscular dystrophy. Mice with this genetic affectation have skeletal muscle defects such as reduced fiber size, degenerating muscle fibers, and central rowing of nuclei in the fiber[7]. Regenerating muscle fibers, which are also present and are characterized by basophilic sarcoplasm, have been described in radioautographs[8] and in diffusion chamber implants of dystrophic muscle[9]. It should be emphasized that the mice used in these experiments were from litters of homozygous normal mice and that they did not carry the gene for muscular dystrophy.

Therefore, these drug-induced changes cannot be attributed to genetic factors. The occurrence of muscle fibers with smaller mean diameters in histamine-treated Swiss Yale albino mice, which belong to a different strain, unrelated to strain 129, further accentuates that these variations from the control findings are the result of histamine administration.



Blister-like evagination of basophilic sarcoplasm containing several nuclei (A). Basophilic sarcoplasm (B) in lower fiber contains several nuclei not in focus at this level. × 1000.

Résumé. Des souris reçurent des injections intrapéritonéales de bichlorure d'histamine. Le diamètre moyen des fibres musculaires s'en trouva nettement réduit et des modifications histologiques furent notées chez les souris traitées.

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Department of Anatomy, University of Texas Medical Branch, Galveston (USA), December 16, 1963.

[7] A. M. MICHELSON, E. S. RUSSELL, and P. J. HARMAN, Proc. Nat. Acad. Sci. *41*, 1079 (1955).
[8] B. E. WALKER, Anat. Rec. *139*, 283 (1961).
[9] W. K. O'STEEN, Lab. Invest. *11*, 412 (1962).

Esterases in Avian Sera: Species Specific Pattern and Individual Variations

This report summarizes preliminary results concerning esterase activities in bird sera. The purpose of this investigation was to determine whether species differences could be observed in zymograms of birds, as they have been described for mammals[1].

Individual samples of serum were collected from adult specimens of both sexes. After agar gel electrophoresis, the esterases were revealed by incubation in one of the media described by URIEL[2]: (1) indoxyl acetate, barbital buffer pH 8.2; with Cu acetate as activator, (2) 1-(α) or 2-(β)-naphthyl acetate or β-carbonaphthoxycholine as substrates in phosphate buffer pH 7 and Diazo Blue B as dye-coupler, (3) butyrylthiocholine as substrate with tetrazolium salt Nitro BT, (4) inhibitors: prostigmin (eserin) M 10^{-4} and DFP M 10^{-6}.

The results with indoxyl as well as with α- or β-naphthyl acetates were the same, unlike what was seen in mammals[2–5]. The first reaction was hence most often used; its advantage is a good colour contrast between the indigo blue and the red of Ponceau S, the stain used for revealing proteins[6]. When both reactions are applied on the same plate, a direct and easy localization of esterases with respect to proteins is realized.

The sera studied were:

I. Gallinaceae:		
(1) Hens	(a) Rhode Island Red, strain M44	16
	(b) White Leghorn, without pedigree	12
	(c) without specification of race	12
(2) Guinea-fowls (blue)		16
(3) Pheasants, *Ph. colchicus*		12
Pheasants, *Ph. colchicus* var. *obscurus*		4
(4) Turkeys, black		8
Turkeys, white		2
(5) Quails of different origins but mainly from families with pedigree		80
II. Anatidae:		
(1) Ducks	(a) Pekin, mainly from an inbred strain	200
	(b) Khaki without specification	200
	(c) hybrids	200
(2) Barbary ducks		6
(3) Goose		1
III. Columbidae:		
Pigeons	(a) White Peacock	8
	(b) grey (Paris)	4
	(c) Cauchois (hybridized)	12

Five groups of esterase spots were recorded, according to electrophoretic migration:

(1) β_2-γ region: A slow esterase is present in some sera of pigeons and ducks[7]; it is inhibited by prostigmin M 10^{-5} for duck sera but not inhibited even by M 10^{-4} for pigeons. A somewhat faster esterase is present in all examined sera of guinea-fowl; it is inhibited by prostigmin.

(2) β_1 region (around the reservoir): Present in all sera; variable in intensity; more important in Gallinaceae than in other species; the most intense for pheasants and guinea-fowl sera; inhibited by prostigmin; the only spot coloured by butyrylthiocholine.

(3) α_2–α_1 region: Present in all sera; variable in intensity and mobility; the most important in Anatidae and pigeons; in these species no individual variations were observed; for turkeys, pheasants and hens (with exception of the M44 strain) small variations in intensity; in guinea-fowl sera there are two spots variable in intensity; in quail's sera, significant variations in number, intensity and mobility were encountered: some sera contain a slow α-esterase, some a fast α-esterase, some both of them and others none. All α-esterases are inhibited by the DFP and they are resistant to prostigmin.

(4) Albumin region: Some quail sera contain an esterase covering partially the albumin area.

(5) ϱ (rapid): This esterase, faster than albumin, is less frequent and less intense than the preceding one; not observed in hen, turkey or guinea-fowl sera, quite important in pheasants, it is also present in ducks and pigeons.

Although for some species there are individual variations, on the whole significant differences between species were observed. In some cases these were more striking than differences between proteinograms. Thus the esterase pattern appears as a sensitive technique for differentiation of bird species.

The intra-species variations observed might, on the other hand, be ascribed to the genetically determined families of components, as for example, transferrins, haptoglobins etc. An investigation is in progress on quail.

In some species quite uniform patterns were found which could be interpreted in terms of biological homogeneity of the strain examined, as for instance the M44. In the case of duck sera, however, the remarkable constancy of results was obtained with a considerable number of samples from animals of various races, and, as such, certainly not closely related. As their protein patterns were previously found variable[6,8], this discrepancy between esterases and main proteins would indicate that they do not correspond to the same phase of phylogenetic evolution[9].

Résumé. Les taches colorées correspondant à l'activité estérasique des sérums d'oiseaux, après électrophorèse en gélose, diffèrent entre elles en nombre, localization et relative intensité de coloration dans les sérums provenant de familles différentes: Poules, Canards, Pigeons ou d'espèces d'une même famille. Chez certaines espèces, on peut également observer des variations individuelles.

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1 J. PAUL and P. F. FOTTRELL, Ann. N.Y. Acad. Sci. *94*, 668 (1961).
2 J. URIEL, Ann. Inst. Pasteur *101*, 104 (1961).
3 N. TALAL, G. HERMANN, C. DE VAUX ST. CYR, and P. GRABAR, J. Immunol. *90*, 246 (1963).
4 G. HERMANN, N. TALAL, C. DE VAUX ST. CYR, and J. ESCRIBANO, J. Immunol. *90*, 257 (1963).
5 M. KAMINSKI and H. BALBIERZ, in preparation.
6 M. KAMINSKI and E. GAJOS, in *Proteins in Biological Fluids* (Elsevier, Amsterdam 1964), in press.
7 M. KAMINSKI, C. R. Acad. Sci. *257*, 2745 (1963).
8 M. KAMINSKI, Bull. Soc. Chim. Biol. *1964*, in press.
9 All Gallinaceae and ducks studied came from the Laboratoire de Photobiologie du C.N.R.S., Gif-sur-Yvette. Thanks are due to the whole staff for help in collecting blood samples.

The CO_2-Binding Capacity of Rat Brain Tissue *in vivo*

There has been no systematic determination of the CO_2-binding capacity of brain tissue *in vivo* and the scattered values which exist have been based on tissue material subjected to *post mortem* changes. It has been shown that accurate analysis of the acid-base metabolism of the brain requires that the tissue is frozen *in situ*[1,2]. Carbon dioxide dissociation curves on brain tissue homogenates, on the other hand, do not truly reflect the *in vivo* buffer capacity of the tissue, since this capacity may involve active transport mechanisms as well as metabolically induced changes of the tissue buffer systems. The present study, which is part of a systematic investigation of the acid-base metabolism of brain tissue, describes the CO_2-binding capacity of rat brain tissue exposed to arterial carbon dioxide tensions of 12–107 mm Hg.

Methods. The experiments were performed on rats of the Sprague-Dawley strain which were anaesthetized with Nembutal (40–50 mg/kg body weight) and tracheotomized. The rats were exposed to various carbon dioxide concentrations for 5–180 min. Low arterial carbon dioxide tensions were induced in some animals by artificial hyperventilation with a Palmer Miniature Ideal Respiration Pump. The other rats were allowed to breathe spontaneously and they were given gas mixtures containing 0–12% carbon dioxide in an open system. The acid-base parameters in arterial blood were measured in samples drawn from a cannula in the femoral artery, using the micromethod of SIGGARD ANDERSEN et al.[3]. Blood analyses were performed before and at repeated intervals during the exposure of the animals to the altered P_{CO_2}. At the end of the experiment the head of the animal was dipped into liquid nitrogen. The total carbon dioxide content of the brain was determined with the method recently

1 U. PONTÉN and B. K. SIESJÖ, Exper. *19*, 312 (1963).
2 U. PONTÉN and B. K. SIESJÖ, Acta physiol. scand., in press.
3 O. SIGGARD ANDERSEN, K. ENGEL, K. JÖRGENSEN, and P. ASTRUP, Scand. J. clin. lab. Invest. *12*, 172 (1960).