to be expressed quantitatively); secondly, that it is easily repeatable.

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Unusual Urinary Compound in Patients Homozygous for Hereditary Atypical Serum Cholinesterase

URINE from patients homozygous for hereditary atypical serum cholinesterase (HASC) and from normals (mainly laboratory personnel) was collected and refrigerated immediately. The cholinesterase genotype, as shown in Table 1, was determined by reported methods^{1,2} on blood specimens drawn at the same time. The urine was lyophilized and reconstituted with distilled water to one-fifth the original volume. The concentrates were extracted by a slight modification of the method of Biran and Bartley³, as shown in Fig. 1.

Fig. 1. Method of extraction of urine

The final methanol phase was evaporated *in vacuo* to a small volume and separated by thin-layer chromatography on 'Silica Gel G', in a solvent system of chloroform-methanol-water (65:25:4).

Inspection of the chromatograms under ultra-violet light (maximum emission 254 mμ) revealed a very strong yellow fluorescence, R_F 0.65, in the extracts from all HASC patients. Only one normal urine contained this material, and it was generally present in small amounts and erratically. This normal individual, as shown in Table 1, is not a heterozygote for the atypical gene. The urines from two patients with myasthenia gravis under treatment with pyridostigmine were also studied and the fluorescent material was demonstrated in all specimens. One normal individual was given pyridostigmine bromide, 60 mg every 6 h for 10 days, and his urine was examined

Table 1. OCCURRENCE OF UNUSUAL URINARY COMPOUND

Urine source	Sex	Dibucaine No. (%) (refs. 1, 2)	Benzoylcholine Acetylcholine (ref. 2)	No. of urine specimens	Yellow fluorescence*
Normals					
Di.	M	73.0	0.70	2	0
Si.	M	76.9	0.56	1	0
Lu.	F	78.4	0.67	4	0 to ++
N. N.	M	80.1	0.71	1	0
Os.	F	79.0	0.69	1	0
Do.	F	72.3	0.78	1	0
Bo.	M	83.9	0.62	1	0
Ru.	F	85.1	0.69	3	0
H. R.	M	74.6	0.71	1	0
HASC patients					
Hug.	F	29.6	2.74	17	++++
Pa.	F	32.9	3.96	1	++++
Oe.	M	37.6	3.68	2	++++
Pr.	M	37.9	3.31	2	++++
Ar.	F	29.4	3.91	1	++++
Ro.	M	17.2	2.76	1	++++
M. G.	F	21.1	4.00	1	++++
T. M.	M	35.5	3.98	1	++++
Myasthenia gravis, treated†					
Br.	M	80.0	0.67	4	++++
Ca.	M	75.6	0.75	3	++++
Normal treated with pyridostigmine†					
Har. before	M	74.0	0.68	1	0
Har. after	—	—	—	1	++

* Intensity of fluorescent band graded 0 to + + + +.

† Serum cholinesterase assays while on pyridostigmine showed moderate to marked decrease in activity.

before and at the end of the treatment period. The initial urine did not contain the fluorescent material but the follow-up urine did. All the observations so far are summarized in Table 1.

At present, experiments are being carried out with silica columns to prepare sufficient purified fluorescent material to characterize it. Identification of this compound, which is present in states with diminished cholinesterase activity, may throw some light on the function of the normal enzyme.

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Serum Lipids in some purely Herbivorous Wild Animals

THE hypothesis that there is a correlation between the pathogenesis of atheroma and the metabolism of lipids appears to have sprung from the fact that the earliest lesion which the pathologist sees in atheroma is the accumulation of lipid material in the intima and sub-intimal connective tissues. Among the various substances classed as lipids that may play a part in causing atheroma, cholesterol and its esters are the most highly favoured suspects. This suspicion has been harboured since Vogel¹ established in 1847 the invariable presence of cholesterol in atheromatous lesions, and this idea was much strengthened by the production of experimental atheroma in rabbits by giving them pure cholesterol in oil^{2,3}.

We have previously found atheroma in some wild animals^{4,5}, even in pure herbivores which appeared to have no less atherosclerosis than the omnivores, and we are now determining the serum lipids in a variety of species. Particular interest is attached to animals having a life-