virus following administration of normal allantoic fluid.

Similar interference experiments conducted in mice by the intranasal injection of partially inactivated virus preparations, followed 5 hours later by the active agent, have given results indicating that the same phenomenon may be demonstrated in this species. Protection against as much as 250 50-per-cent.-mortality doses was noted.

Interference of one virus with another has been observed repeatedly. The viruses may be quite unrelated or very closely related, as is the case with neurotropic and non-neurotropic strains of influenza Type A virus.[2] Interference of inactivated bacteriophage with the active agent of the same strain has been observed recently[3] and the present results extend these observations to the influenza viruses. It seems very likely from the data presented that virus having been inactivated during the process of cultivation may cause such interference phenomena and account for the difficulties encountered in the propagation of some strains of influenza virus. A more extensive report will be published elsewhere.

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THE EFFECT OF CASTRATION AND TESTOSTERONE PROPIONATE ON d-AMINO ACID OXIDASE ACTIVITY IN THE MOUSE[1]

IN recent reports from this laboratory we have indicated the effect of castration and testosterone propionate on the activity of three hydrolytic enzymes.[2, 3] We now wish to report findings with respect to an oxidative enzyme, d-amino acid oxidase.

The mice were of an inbred stock, Buffalo-Marsh strain.[4] Castration and implantation of the testosterone propionate[5] pellets were performed when the mice were 18 ± gms body weight. The enzyme activity was determined by a modification of Elvehjem's method. The pyruvic acid formed in the presence of arsenite was determined by the 2,4 dinitrophenylhydrazone method.

The results in Table 1 demonstrate that the mouse kidney loses part of its ability to oxidatively deaminate d-alanine as a result of castration. The administration of testosterone propionate not only restores this property but increases it above normal. These data provide further evidence for our program to elucidate the nature and purpose of the protein anabolic properties of certain steroids originally observed in this laboratory in castrate dogs.[6]

TABLE 1

THE EFFECT OF CASTRATION AND TESTOSTERONE PROPIONATE ON THE d-AMINO ACID OXIDASE ACTIVITY OF MOUSE KIDNEY

Treatment*	No.	Weight of kidneys gms	Pyruvic acid formed			
			Total		Per gram	
			micro-moles	Per cent.	micro-moles	Per cent.
Castrate ..	6	0.259	20.1	−70	75	−54
Normal ...	5	0.402	65.5	..	164	..
Cast. and T. P.	3	0.545	108.0	+64	198	+21
Normal and L. P.	3	0.537	102.0	+55	190	+16

* Body weight at castration 18 ± gms. Treatment for 130 ± days.

The substrates incubated with mouse liver brei in no instance showed the presence of pyruvate. Either the mouse liver possesses no d-amino acid oxidase or it has a different mechanism than the rat for metabolizing pyruvate. We have been able consistently to find pyruvate in good amounts in substrates incubated with rat liver brei according to exactly the same procedure used for the mouse experiments. This difference in enzyme activity between the two species is not too surprising, for we have noted marked differences in the arginase and phosphatase activities in these same species.

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DIFFERENTIAL INHIBITION BETWEEN NORMAL AND TUMOR (CROWN GALL) TISSUE IN BEET ROOTS

A DISTINCT difference has been found by the authors in the action of resorcinol and of cyanide upon the rate of oxygen uptake in the tissues of normal beets and of beet root tumors induced by inoculation with *Phytomonas tumefaciens*.

With normal beet tissue an inhibition of 12 to 14 per cent. is obtained with 0.0166M resorcinol, whereas in tumor tissue this amounts to 20 to 23 per cent.

2 C. H. Andrewes, *Brit. Jour. Exp. Path.*, 23, 214, 1942.
3 S. E. Luria and M. Delbrück, *Arch. Biochem.*, 1: 207, 1942.
1 This investigation was aided by grants from the Ciba Pharmaceutical Products, Inc., Summit, N. J., and the Josiah Macy, Jr., Foundation, New York, N. Y.
2 C. D. Kochakian and L. C. Clark, Jr., *Jour. Biol. Chem.*, 143: 795, 1942.
3 C. D. Kochakian and R. P. Fox, *Endocrinology*, 30: S1033, 1942.
4 The mice were provided by the Biological Station, Springville, N. Y., through the courtesy of Drs. W. S. Murray and S. G. Warner.
5 The testosterone propionate (Perandren) was supplied by the Ciba Pharmaceutical Products, Inc., through the kindness of Dr. E. Oppenheimer.
6 C. D. Kochakian and J. R. Murlin, *Jour. Nutrition*, 10: 437, 1935; *Am. Jour. Physiol.*, 117: 642, 1936; C. D. Kochakian, *Endocrinology*, 21: 750, 1937.

inhibition. Similarly, it was found that cyanide in concentrations of 0.0166M inhibits the oxygen uptake to the extent of 84 to 86 per cent. in normal beets and of 79 to 80 per cent. in tumor beet tissue.

Previous work in this laboratory[1] has shown a differentiation between the healthy part and the tumorous part of the same beet, whereas the above work shows a differentiation between tumorous tissue and the tissue of an entirely healthy non-infected beet root.

Upon addition of cyanide to resorcinol, and *vice versa,* in the case of both healthy and tumorous beet root tissue, there is an increase in inhibition of 6 to 8 per cent. above that due to cyanide alone. These results are summarized in Table 1.

TABLE 1

EFFECT OF RESORCINOL AND CYANIDE ON THE RESPIRATION OF NORMAL AND TUMOR BEET ROOT TISSUE

Substance added	Per cent. inhibition Beet tissue slices	
	Normal	Tumor
Resorcinol	12–14	20–23
Cyanide	84–86	79–80
Resorcinol and cyanide ..	92	86

They suggest: (*a*) Tumors as well as healthy beets may have the following types of respiratory mechanisms: (i) cyanide insensitive system, (ii) resorcinol insensitive system, (iii) cyanide plus resorcinol insensitive system, as well as the corresponding sensitive systems, the relative proportion of the three systems in tumor and healthy tissue being different in each case. (*b*) The inhibitions brought about by cyanide and by resorcinol function to a certain degree independently of each other. (*c*) It appears possible that different active centers of the same enzyme (probably a heavy metal compound) are attacked by both inhibitors but to a different degree.

Experiments are now in progress to determine the nature of this differentiation between tumor and normal tissue.

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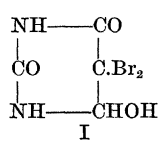
SCIENTIFIC APPARATUS AND LABORATORY METHODS

A SENSITIVE COLOR REACTION FOR THE DETECTION OF URACIL AND CYTOSINE[1,2]

THE author will describe in this paper a modification of the well-known Wheeler and Johnson color test for the detection of the pyrimidines uracil III and cytosine[3] II, which has met with wide application since the date of its discovery in the Yale Laboratory.

The original test is based on the action of bromine on either of these two pyrimidines in aqueous solution, which reacts with them quantitatively with formation of 5,5-dibromoxyhydrouracil I. This hydropyrimidine is characterized by its reactivity towards barium hydroxide with which it reacts in aqueous solution to form (1)—isodialuric acid V and (2)—by rearrangement dialuric[4] acid VI. Both of these pyrimidines give insoluble, deep-purple colored barium salts by neutralization with an excess of barium hydroxide.[5]



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Bromine water or liquid bromine are necessary reagents for the application of this useful test. There are, however, limitations in applying successfully the color test when uracil and cytosine are present in very small quantities; and furthermore, bromine is not always available in clinical laboratories for experimentation. The author also has experienced unexpected difficulties in detecting uracil and cytosine in the study of natural products containing mixtures of carbohydrates, purines and pyrimidines. He has, therefore, devised a new technique or modification of the original Wheeler and Johnson procedure[3] for testing for these two naturally occurring pyrimidines, which is an improvement on the original experimental procedure.

The new technique calls for only two common laboratory reagents, namely, concentrated hydrochloric acid and superoxol.[6] Advantage is taken, in this new application of the color test, of the known reactivity of hydrogen peroxide towards hydrochloric acid as is expressed in the equation below. The hypochlorous

$$HCl + H_2O_2 = HOCl + H_2O$$

acid formed in this reaction reacts immediately and quantitatively with uracil or cytosine if present in

[1] A. C. Neish and Harold Hibbert, *forthcoming publication.*

[1] Contribution from the Department of Chemistry, Yale University.

[2] "Researches on Pyrimidines," clxxx.

[3] Wheeler and Johnson, *Jour. Biol. Chem.,* 3: 183, 1907.

[4] Behrend and Koch, *Ann.,* 315: 246, 1901.

[5] Behrend and Roosen, *Ann.,* 251: 244, 1889.

[6] Hydrogen peroxide 30 per cent.—Merck and Company, Rahway, N. J.