That the meat of lamb varies in B vitamin content from carcass to carcass is of unusual interest because lambs are known not to require the B vitamins. Sheep have been shown to synthesize thiamine, pantothenic acid, and riboflavin in the rumen (\mathcal{Z}) , and an excretion study indicates that they also synthesize niacin (3).

There are no clues as to why some of the animals had higher B content in the meat than others, because production histories could not be obtained during the emergency period when this meat was purchased. Whether the factors which control the B vitamin deposition in the meat are environmental or genetic must be determined by further research.

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The Effects of Cytochrome C in Anoxia

I. HERBERT SCHEINBERG and H. O. MICHEL

Biochemistry Section, Medical Division, Chemical Corps, Edgewood Arsenal, Maryland

Proger and his co-workers (4) have reported that intravenously administered cytochrome C is capable of mitigating the physiological effects of anoxia. In view of these reports it was thought that cytochrome C might be of value in preventing cyanide intoxication. Experiments to be reported elsewhere failed to demonstrate any significant effect of cytochrome C in cyanide-poisoned rats. Further work was then carried out in an attempt to confirm two findings of Proger and his group which were susceptible to objective and quantitative measurement: the effects of cytochrome C on the organ content of easily hydrolyzable phosphorus compounds, and the survival times of anoxic rats.

The resynthesis of adenosine tri- and diphosphate from adenylic acid and inorganic phosphate is coupled to oxidative processes (3) and should be diminished under conditions of anoxia. The tissue content of adenosine tri- and diphosphate parallels the content of easily hydrolyzable phosphorus (2, 3). In addition, under conditions of anoxia the blood level of lactic acid increases (1). In our experiments the amount of easily hydrolyzable phosphorus in heart and kidney, the blood level of lactic acid, and the measurement of survival times were used to quantitate the effect of anoxia on rats and to measure the value, if any, which cytochrome had in the treatment of anoxia.

In the first experiment both members of pairs of littermate white rats were given 2.0 cc./kg. of physiological saline intravenously. One member was then placed in an atmosphere of 3.9 per cent oxygen, and the other allowed to breath air. After 8 minutes determinations of the easily hydrolyzable phosphorus ("7-minute phosphorus") of kidney and heart and of the blood lactic acid were made. These revealed a significant difference in all three quantities between those rats which breathed 3.9 per cent oxygen and those which breathed air (Experiment 1, Table 1). In the second experiment both members of pairs of littermate white rats were placed in an atmosphere of 3.9 per cent oxygen after one had been given approximately 20 mg./kg. of cytochrome C in saline intravenously and the other an equal volume of physiological saline. Determinations of the same

TABLE 1 EFFECT OF CYTOCHROME C ON SEVERAL MANIFESTATIONS OF ANOXIA IN RATS

Experiment	No. of pairs	Treatment of animals	Mean of differ- ences between test and control*	P*
1	7	Pairs of rats (test and control) pretreated with saline. Test placed in an atmos- phere of 3.9 per cent oxygen for 8 minutes; control re- mained in air.		
		Kidney 7-minute phosphorus (mg./100 grams fresh tis- sue)	-2.37	0.025
		Heart 7-minute phosphorus (mg./100 grams fresh tis- sue)	-5.07	0.025
		Blood lactic acid (mg./100 cc. blood)	110.8	Less than 0.01
2	11	Pairs of rats (test and control) placed in 3.9 per cent oxy- gen for 8 minutes. Test pretreated with cytochrome C; control pretreated with saline.		•
		Kidney 7-minute phosphorus (mg./100 grams fresh tis- sue)	0.27	0.68.
		Heart 7-minute phosphorus (mg./100 grams fresh tis- sue)	-0.78	0.41
		Blood lactic acid (mg./100 cc. blood)	-0.64	More than 0.90
3	25	Pairs of rats (test and control) placed in 2.8 per cent oxy- gen. Test pretreated with cytochrome C; control pre- treated with saline.	<u>,</u>	
		Survival time (min.)	0.66	0.45

* The differences between the test and control animals with respect to the measured quantities were found, and from these the values of P, representing the probabilities that the observed differences were due to chance, were determined.

three quantities as in Experiment 1 revealed no significant difference between the cytochrome C and saline pretreated animals (Experiment 2, Table 1).

In the third experiment both members of pairs of white rats of the same age, weight, and sex were placed in an atmosphere of 2.8 per cent oxygen after one had been given 5 mg. of cytochrome C in saline and the other an equal volume of physiological saline intravenously. There was no significant difference in the survival times of the members of each pair (Experiment 3, Table 1).

These results do not confirm the findings of Proger and his collaborators in similar experiments.

The experiments will be reported in detail elsewhere.

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Effects of Ultraviolet Radiation on Visual Thresholds

ERNST WOLF

Biological Laboratories, Harvard University

In a recent paper (1) E. Ludvigh and V. E. Kinsey demonstrated that visual threshold light-difference sensitivity and critical flicker frequency tests in the *forea* are not affected by previous exposure for 5 minutes to the radiation of a 1,000-watt mercury vapor arc, from which most of the visible and almost all of the ultraviolet radiation shorter than 320 m μ were filtered out. These findings seem to contradict the results obtained with baby chicks (2, 3), in which extensive changes in sensitivity thresholds were obtained and which the authors cited can attribute only to the marked difference in absorption and in general physiological characteristics between the eyes of baby chicks and those of adult human beings. They conclude that ultraviolet radiations longer than 320 m μ encountered in nature are without deleterious effect on these functions of the normal human eye.

For the chick (2, 3) as well as for the human eye (4) it has been shown that pre-exposure to the radiation of a mercury vapor arc emitting ultraviolet light above 285 mµ in addition to the visible wave range raises the final dark-adapted thresholds considerably above the normal level (1.3 log unit for the chick and 0.25 log unit for the human eye), as compared with pre-exposures to the same source but with all ultraviolet filtered out. The adapting brightness is in both cases the same, and hence it is assumed that the final threshold differences are due to the ultraviolet. In Fig. 1 data are given for one human observer (light exposure, 10 minutes, with a large adapting field; test with a 12.5° square field; central fixation; presentation, 1/25 second) and for a series of baby chicks (pre-exposure, also 10 minutes). The data for the human eve are individual readings; the chicken data are averages. The figure shows the interesting fact that in both cases the cone part of the duplex dark-adaptation curves is unaffected by the pre-exposure to ultraviolet, while the rod segments are clearly altered. For the human curve the onset of rod adaptation is delayed for about two minutes, the cone segment overshooting the normal beginning of rod adaptation and remaining above the previously established level until termination of the test. For the chick the slopes of cone and rod segments are quite different from those for the human, so that, due to the steepness of the cone segment, an overshooting is not apparent. It is also found that a reduction of the extent of the ultraviolet spectrum reduces the effect on final thresholds; light containing only wave lengths above $365 \text{ m}\mu$ has no effect. Ultraviolet alone, after largely eliminating visible light, acts in qualitatively the same manner as visible light to which ultraviolet has been added.

Previously it has been pointed out (3, 4) that the effect of ultraviolet upon the cones is probably prevented by their

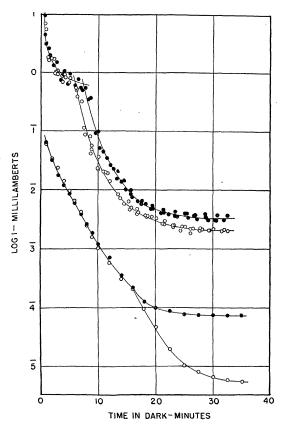


FIG. 1. The course of dark adaptation of human eye (above) and for baby chicks (below) after exposure to the radiation of mercury vapor lamps. Open circles indicate that the ultraviolet has been filtered out; black circles, that ultraviolet above 285 m μ is present.

dense pigmentation, while it acts upon the pigment-free rods. Therefore, while testing *foveal* intensity discrimination, or flicker thresholds, after pre-exposure to ultraviolet, it is obvious that an effect upon visual thresholds cannot be expected, since one is dealing with an irresponsive pure cone population of sensory units with exclusion of the rods. A test of this kind has no relevance to the problem of the presence of an ultraviolet effect upon the peripheral rod units.

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