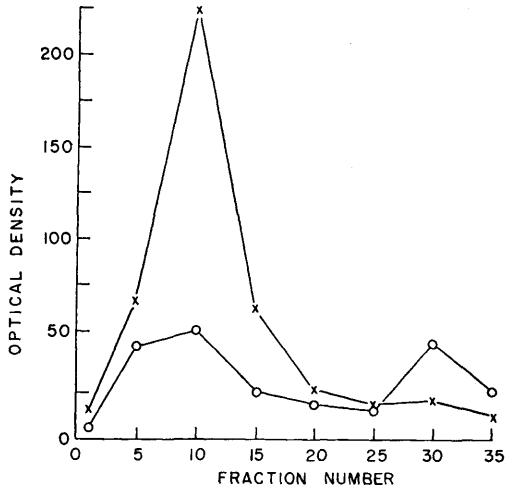

FIG. 1. Electrophoresis of aqueous extract of muscle from vit. E supplemented (×) and vit. E deficient (○) animal.

employing a dilute salt extraction procedure found that there was an increase in percentage of the most rapidly moving component peak 11 and also that the amount of the fast moving component peak 11 was constant in both groups of animals.

Summary. Three pairs of male littermate rabbits were fed a commercial vit. E deficient and a vit. E supplemented diet. When the vit. E deficient member of each pair showed typical vit. E deficient symptoms, the pair of animals was sacrificed and the water soluble muscle proteins extracted from the striated muscle. The electrophoretic pattern of the deficient animals showed a significant change in distribution of water soluble proteins as compared to the control group. This change was an increase in the fast moving peak and a decrease in the slow moving peak.

The authors wish to thank Mrs. B. A. Sullivan for assistance in the care of the animals.

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Ethanol Metabolism in Vitamin Deficiencies. I. Niacin.* (23379)

GEORGE H. NELSON AND JAMES I. ABBENHAUS (Introduced by Edwin H. Shaw, Jr.)
Department of Biochemistry, University of South Dakota, School of Medicine, Vermillion.

Westerfeld(1) recently reviewed the pathways of ethanol† metabolism, and more recently Schulman and Westerfeld(2) have proposed an additional possible pathway. Since the route of metabolism has not been definitely established, it seemed worthwhile to study the effects of various vitamin deficiencies on the rate of alcohol metabolism in an effort to evaluate the role of vitamins. The first step in alcohol metabolism appears to be its conversion to acetaldehyde probably by means of alcohol dehydrogenase with DPN acting as hydrogen acceptor. Since DPN is a niacin-containing coenzyme, this study was begun with niacin.

Methods. Weanling male rats of the Sprague-Dawley strain, 24 days old when received in this laboratory, were used throughout the experiments.

Since the rat can synthesize niacin from abundant dietary tryptophan, the tryptophan content of the diet was limited. Preliminary work in this field indicated that a true niacin deficiency could be induced in rats with the diet shown on the next page.

The control diet contained, in addition, 50 mg niacin. Some rats were maintained on a diet similar to the above except the casein-casein hydrolysate ratio was 80-120 instead of 100-100.

Immediately upon arrival of the rats in this laboratory, the animals were paired and individually caged, and the deficient group was allowed to eat the niacin deficient diet

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† Hereafter referred to as alcohol.

Casein	100	g
" hydrolysate (tryptophan free)	100	g
Sucrose	700	g
Cottonseed oil	40	g
Cod liver oil	10	g
Alphacel	20	g
Salt mixture (U.S.P. XIV)	30	g
Thiamine hydrochloride	5	mg
Riboflavin	5	mg
Calcium pantothenate	10	mg
Pyridoxine hydrochloride	10	mg
Para-amino benzoic acid	50	mg
i-inositol	250	mg
Choline chloride	1500	mg
Biotin	.2	mg
Pteroylglutamic acid	.2	mg

ad lib. At the end of each 24 hour period the food was weighed and amount of food eaten by a deficient animal was fed to his pair-fed control. A third and fourth group of animals were allowed to eat the 100-100 and 80-120 casein-casein hydrolysate control diets *ad lib.* These groups of animals were used for weight comparisons with the deficient animals. Rates of alcohol metabolism were determined only on deficient and pair-fed control animals.

When an animal was considered niacin deficient by comparison of his growth curve with that of the control animals fed *ad lib.*, alcohol metabolism was determined in the following manner: alcohol was injected intraperitoneally as a 20% W/V solution in isotonic saline in a dose of 3 g/kg. The injection was made with a calibrated syringe pipette (Vernes Rheometer). After injection the animal was allowed to metabolize for exactly 3 hours. He was then killed by a sharp blow on the back of the neck and cut into small pieces into an all-metal Waring blendor. Blood and any other remains of the animal were washed from the scissors into the container with a weight of water 7 times that of the animal. The container was sealed with its air tight metal screw cap and the 1 to 8 homogenate was made. After homogenization, the container was cooled in an ice bath before removing the cap. Alcohol concentration of the homogenate was determined by the method of Aull *et al.*(3), using the specially designed Aull alcohol apparatus.‡ Rate of alcohol metabolism was calculated by subtracting recovery of alcohol in mg/100 g rat after metabolism from 304, multiplying by 10, and dividing by 3. The rate is then expressed as mg alcohol metabolized/kg/hr. Recovery of alcohol from 12 dead rats weighing 50-68 g and injected as above was 304 $\pm$ 3.9 (mean $\pm$ standard deviation) mg/100 g rat. This value was used in the calculation of rate of metabolism as the initial dose instead of the theoretical value of 300 mg/100 g rat. Fed deficient animals had access to food until just before the injection of alcohol. Fed pair-fed animals were fed a few hours prior to the run. All fasted animals were denied access to food for 24 hours.

‡ Scientific Glass Apparatus Co., Bloomfield, N. J.

Results. The results of 19 pairs of animals are shown in Table I. In each of Groups I and II three pairs of animals were fed the diets containing the 80-120 ratio of casein-casein hydrolysate, while all other pairs were fed diets containing the 100-100 ratio. No difference was noticed between the 2 different ratios. As can be seen, some averages show a rather high standard deviation. This is in part due to the differences in age and weight within a group of animals; the younger, smaller animals tend to show a higher rate of metabolism. Nevertheless, in 18 of the 19 pairs of animals, the deficient animal exhibited a higher rate of metabolism than did his pair-fed control. The single deficient animal that showed a lower rate of metabolism is included in Group I. One might postulate that the deficient animals exhibit a higher rate of metabolism because they are smaller. However, if one calculates rate of metabolism as mg alcohol oxidized/whole animal/hr, one finds that in 16 of the 19 pairs the deficient animal still exhibits a higher rate of metabolism. It would be customary to expect a smaller animal to burn less alcohol per whole animal. Whether the animals were fed or fasted appears to have no significant effect on the over-all picture. Recent work by Smith *et al.*(4) indicated that injection of nicotinamide into mice 6 hours prior to injection of alcohol had no effect on rate of alcohol metabolism, although DPN concentration in the liver was raised 10-fold.

Summary. It has been shown that niacin deficiency in rats has the effect of increasing

TABLE I. Effect of Niacin Deficiency on Rate of Alcohol Metabolism.

Group	No. of rats	Deficient or pair fed	Wt in g (range, avg)	Age in days (range, avg)	Fed or fasted	Rate of metabolism (mg/kg/hr), avg ± S.D.	p values
I	6	Def.	40-65 49.2	44-54 49.3	Fed	493 ± 39	<.10 >.05
	6	P.F.	46-68 54.0	44-54 49.3	"	401 ±106	
II	6	Def.	38-70 50.3	45-55 49.7	"	461 ± 25	<.01
	6	P.F.	46-66 52.2	45-55 49.7	Fasted	372 ± 63	
III	7	Def.	46-53 48.1	53-64 59.7	"	370 ± 41	<.01
	7	P.F.	46-53 50.9	53-64 59.7	"	313 ± 21	
I, II, III	19	Def.	38-70 49.1	44-64 53.3	—	438 ± 64	<.01
	19	P.F.	46-68 52.3	44-64 53.3	—	359 ± 76	
I, III	13	Def.	40-65 48.6	44-64 54.9	—	427 ± 74	<.05 >.01
	13	P.F.	46-68 52.3	44-64 54.9	—	353 ± 84	

rate of alcohol metabolism in spite of the fact that DPN is generally conceded to be involved in conversion of alcohol to acetaldehyde.

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Preparation of Coated Radioautographs by Dipping Sections in Fluid Emulsion.* (23380)

B. MESSIER AND C. P. LEBLOND

Department of Anatomy, McGill University and Montreal Cancer Institute, Notre Dame Hospital, Montreal, Canada

In 1946, the coating method of radioautography was developed in collaboration with Bélanger(1). The intimate contact provided by this method between the photographic emulsion and the radioactive tissue section insured a high resolution, which has not been improved by any technical procedures, except by the use of high contrast "nuclear" emulsions. In 1947 it was proposed to mount the section directly on a photographic plate(2) or to place a stripping film over the section(3). A high resolution could also be obtained with these procedures(4), but the coating method(1,5) has been preferred by the present authors because of the speed with which large batches of slides may be handled. More recently, Joftes and Warren proposed a method in which the slide is dipped in melted emulsion(6). While several steps of the technic were felt to be cumbersome or unnecessary, the dipping itself is simple and requires a minimum of equipment. An attempt was, therefore, made to introduce a "dipping" step into the coating method routinely used here. The results proved to be highly satisfactory and the technic will now be described in detail.

Slides bearing histological sections, either stained or unstained, are placed in a 1% solution of celloidin for a few seconds ("Tissue Imbedding Solution" No. M 4700, manufactured by Randolph Products Co., N. J., di-

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