sought through absorption experiments. Definite proof of specific antibodies in sera against kidney, liver, testicle and possibly muscle particles was obtained, as shown in Table II.

TABLE II

EFFECT OF ABSORPTION ON SPECIFICITY OF SLIDE AGGLUTINATION

Antigen	Serum absorbed with	Rabbit serum *vs.* mouse organ particles from: Lung	Kidney	Muscle	Liver	Testicle	Saline
Kidney particles	.....	1	4	1	2	0	0
	Kidney	0	0	0	0		
	Muscle	0	4	0	0		
	Liver	0	4	0	0		
	Sheep red cells	1	4	1	2		
Liver particles	.....	1	1	2	4	0	0
	Kidney	0	0	0	3		
	Muscle	0	0	0	4		
	Liver	0	0	0	0		
	Sheep cells	1	1	2	4		
Muscle particles	.....	1	1	3	3	0	0
	Kidney	0	0	±	0		
	Muscle	0	0	0	0		
	Liver	0	0	2*	0		
	Sheep cells	..	..	..	..		
Testicle particles	.....	1	1	1	2	2	0
	Liver	0	0	0	0	2	

* Apparently some specificity in this experiment, but not observed in other muscle preparations.

It is obvious from these experiments that the antibodies can be absorbed completely only by the homologous antigen, while cross-reactions apparently disappear upon absorption with any one of the heterologous antigens. The Forssman antibodies are not concerned with these cross-reactions, since no change occurs upon complete absorption with sheep cells. There was no definite relation between the mouse serum precipitins or mouse red cell agglutinins and these cross-reactions.

Using particles derived from another species—the ferret—we have observed agglutination of liver only, by mouse liver antiserum, and of brain only by mouse brain antiserum, while kidney particles were not agglutinated by any one of the sera. Ferret muscle antigen cross-reacted with several antisera, other than anti-muscle.

These experiments prove that particles derived from some normal mouse organs by high-speed centrifugation (25,000 RPM) show, in addition to the Forssman antigen, organ specific differentiation. The question of organ specific structures in tissues and cells has been studied repeatedly (*cf.* Landsteiner[5]). While in certain instances (*e.g.,* lens, brain and others) organ specificity is demonstrable without great difficulty, in other cases (kidney, liver) the results have been more or less indefinite.

[5] K. Landsteiner, "The Specificity of Serological Reactions," C C Thomas, Springfield, Ill., 1936.

As a result of these various studies, different types of organ-specific antigens have been identified.[6] One group is characterized by the fact that the same antigen is present in the homologous organ of many different species. Our results show that liver as well as brain particles conform with this type.

The kidney particle preparations employed in our experiments belong to another group of organ specific antigens which are found in one organ from one species only. However, since we have used only two species, it is obvious that these observations must be extended.

In a number of instances, it has been possible to ascribe organ specific reactions to alcohol extractable material.[7] The particles used in this study contained high percentages of lipoids, but the relation of these fractions to the specificity is still under investigation.

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STABILIZATION OF IODINE IN SALT AND FEEDSTUFFS

ON the Industrial Fellowship on iodine at Mellon Institute we have had occasion to investigate broadly the factors responsible for the loss of iodine from iodized salt and iodized mineral feeds. These mixtures may contain any or all of the following substances: ferric oxide, copper sulfate, cobaltous nitrate, sodium chloride, manganese sulfate, calcium carbonate, calcium phosphate, sodium sulfate, sulfur, potassium iodide, volatile flavors, organic meals and vitamin concentrates. Potassium iodide is furnished to the feed manufacturer in the form of either an iodized mineral mixture, a concentrated iodized pre-mix containing the essential minerals or an iodized salt. The primary cause of loss of iodine is through oxidation of the iodide to free iodine with subsequent volatilization. Another important factor is the absorption of potassium iodide by the fabric or cardboard containers. The formation of free iodine not only results in a loss of iodine but also causes a decrease in the vitamin C content of the feed.

Iodized mineral feed mixtures lose between 9 per cent. and 20 per cent. of their iodine content during four months' storage under ordinary conditions. Oxidation occurs mainly through the catalytic action of iron, copper and manganese compounds present in the mixtures. These reactions take place only in the presence of moisture and are accelerated by the action of light. An important synergism is observable in this catalytic action. Ferric oxide becomes appreciably

[6] E. Witebsky, Report of Proceedings, 2nd Intern. Congress of Microbiol., London, 1937.

[7] E. Witebsky, *Zeitschr. f. Immunitätsf.*, 62: 3, 1929.

[8] Nemours Foundation Fellow.

soluble in the presence of soluble manganous compounds, and the catalytic activity of iron and copper together is much greater than the additive effects of iron and copper separately.

The loss of iodine from iodized salt depends upon the oxidizing impurities in the salt, chiefly chlorate, nitrate and ferric chloride. Iodized salt for animal feeding, which contains large amounts of potassium iodide, loses iodine with great rapidity. Iodized salt for human consumption, which contains 0.02 per cent. potassium iodide, loses 40 per cent. or more of the iodine in eighteen months. If the salt is freshly prepared, approximately 15 to 20 per cent. of the iodine is lost during the first month.

Stabilization by the use of alkaline agents and reducing agents has been recommended and extensively used. But this treatment has not been entirely successful because of the inability to obtain adequate contact between the reacting components in a dry powder. The employment of a reducing agent in conjunction with a soluble pyrophosphate is more effective. Pyrophosphate forms an inactive complex with oxidized iron; it also destroys the synergism between iron and copper.

A new and simplified procedure for stabilizing the iodine reinforcement of comestibles has come from our work. The process consists of milling 100-mesh alkali iodide with a small portion of a non-toxic metallic soap. The milling of 92 parts of potassium iodide with 8 or more parts of calcium stearate in the form of an impalpable powder is recommended; the powder density of the calcium stearate should be as low as possible. The resulting product is a stable free-flowing powder, coated with calcium stearate and practically insoluble in water. The coating is rapidly emulsified in the presence of bile. Calcium stearate is non-toxic and may be ingested in reasonable amounts with complete physiological safety. Various grades of the impalpable powder are available commercially.

A mineralized salt, containing 10 per cent. ferric oxide, 2 per cent. copper sulfate, and 4 per cent. potassium iodide coated with calcium stearate, has lost only 0.9 per cent. of its iodine content during storage for four months, while the same formula without calcium stearate has lost 15 per cent. of the iodine. An iodized livestock mineral containing 0.21 per cent. stearate-coated potassium iodide lost 0.5 per cent. of the original iodine content during two months, while an unstabilized mineral containing the same ingredients lost 14 per cent. of its iodine content. Absorption of the stearate-coated potassium iodide by cardboard, paper or fabric containers does not occur.

Calcium stearate also functions to prevent caking in table salt. As iodized table salt contains 0.02 per cent. potassium iodide, it is necessary to employ 0.2 per cent., or ten times as much calcium stearate as potassium iodide, to be effective in preventing caking. The cost is comparable to that of present methods.

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A NEW FACTOR OF THE VITAMIN B COMPLEX REQUIRED BY THE ALBINO MOUSE

IN view of the wide use of the albino mouse as an experimental animal, particularly in medical research, it is surprising that so little is known about its dietary requirements. The work that has been done[1,2,3,4,5] indicates that the nutrition of the mouse differs markedly from that of the rat. It was stated by Birch, György and Harris[6] that mice on the Goldberger black-tongue diet supplemented with 5 International Units of thiamin and 15γ of riboflavin injected twice a week failed to grow and developed skin lesions and loss of hair, whereas rats on the same diet grew normally.

In the study of the requirements of the albino mouse for the several factors of the vitamin B complex, a basal diet consisting of purified casein, 18 per cent., sucrose, 75 per cent., cod liver oil, 1 per cent., butter fat, 2 per cent., and Osborne and Mendel's salt mixture, 4 per cent., is being used, supplemented with known factors of the B complex.

When crystalline pyridoxin, thiamin, nicotinic acid and riboflavin are injected at various levels up to 10γ, 25γ, 25γ and 15γ respectively per day, failure of growth results in every case, and a characteristic skin lesion develops in from 30 to 40 days. If the crystalline supplements are fed with the basal diet at levels of 100γ of pyridoxin, 250γ of thiamin and nicotinic acid and 150γ of riboflavin daily, the same results are observed as when the B factors are injected.

The addition of liver or yeast, or the water or dilute (30 per cent.) alcohol extract of liver or yeast to the diet produces normal growth and maintains healthy skins in mice. However, feeding the basal diet supplemented with pyridoxin, thiamin, nicotinic acid and riboflavin with the addition of the filtrate from a fuller's earth adsorption of either yeast or liver extract neither produces growth nor prevents the appearance of skin lesions in mice when feeding amounts of filtrate equivalent to 0.1 gram and 0.2 gram of yeast daily or 0.5 gram of liver every other day. In only one case[7] have skin lesions been described on a diet adequate in thiamin, pyridoxin, nicotinic acid, ribo-

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[5] J. M. Wolfe and H. P. Salter, *Jour. Nutrition*, 4: 185, 1931.
[6] T. W. Birch, P. György and L. J. Harris, *Biochem. Jour.*, 29: 2830, 1935.
[7] P. György and R. Eckhardt, *Nature*, 144: 512, 1939.