prodigious amount of root hairs. Beta-1-naphthylalanine, glycyl-β-1-naphthylalanine, β-3-thianaphthylalanine, and β-2-naphthylalanine produced the greatest inhibition of root growth of the naphthylalanines tested.

The respiratory quotient (RQ) was obtained by standard procedures (*5*). These studies show that the RQ of germinating cucumber seeds was increased by the naphthylalanines previously mentioned (Table 1), with the exception of β-2-naphthylalanine. The RQ of the β-1-naphthylalanine, glycyl-β-1-naphthylalanines, and β-3-thianaphthylalanine treated seed can be correlated to the growth pattern observed in the roots. These compounds produced an effect similar to that observed with 1-naphthaleneacetic acid. The RQ of the seeds treated with the carbobenzoxyglycyl-β-naphthylalanines and β-naphthylalanine was approximately the same as the control whereas the growth behavior was much less than that of the nontreated seeds.

A possible explanation for these results is that the β-2-naphthylalanines may be converted into naphthyleneacetic acid by a mechanism similar to the conversion of tryptophane to indoleacetic acid in plants.

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Received August 28, 1953.

The Role of Lactones in Flavor Deterioration of Milk Fat[1, 2]

Stuart Patton, Philip G. Keeney, and Carl T. Herald[3]

The Pennsylvania Agricultural Experiment Station, State College

At an early stage in the storage deterioration of anhydrous milk fat a coconut-like flavor defect is evident (*1, 2*). This defect is manifest prior to typical oxidized fat flavors. Keeney and Doan (*1*) have suggested that lactones formed during deterioration of the fat are responsible for the off-flavor. This lipid-associated flavor defect is considered to be a principal factor limiting the acceptability of dry whole milk for beverage purposes (*2*).

[1] Authorized for publication 28 Nov., 1953, as paper No. 1845 in the Journal Series of the Pennsylvania Agricultural Experiment Station.

[2] Research undertaken in cooperation with the Quartermaster Food and Container Institute for the Armed Forces and assigned No. 457 in the series of papers approved for publication. The views or conclusions contained in the report are those of the authors and are not to be construed as reflecting the views or endorsements of the Department of Defense.

[3] We are indebted to Firmenich, Inc., and Fritzsche Bros., Inc., for supplying the lactones employed in this investigation.

Evidence from this laboratory has indicated that the flavor and odor properties of δ decalactone appear to be identical with those of the compound responsible for the coconut-like off-flavor of milk fat. A logical origin of this lactone is Δ^9 decenoic acid, a component of milk fat glycerides. This acid and Δ^9 dodecanoic and Δ^9 tetradecenoic acids occur in milk fat but apparently are absent in most other fats (*3–5*). Of a number of fats studied, including milk fat, lard, coconut, cottonseed, and soybean and peanut oils, only milk fat developed the off-flavor in question. A comparison of flavor deterioration in milk fat containing phospholipids and in that washed free of phospholipids showed development of the off-flavor in both media with equal facility. Extensive study also eliminated vitamin A, carotene, and other constituents of the unsaponifiable matter as possible origins of the flavor defect. Experiments involving low-temperature crystallization and hydraulic expression of milk fat demonstrated that the flavor defect develops most readily in the low-melting fraction. Hydrogenation of milk fat from an iodine value of 32 to a value of 12 completely prevented development of the off-flavor. These results are considered good evidence that the off-flavor originates from the unsaturated glycerides of milk fat. Observations on flavor deterioration of the methyl esters of oleic, linoleic, and linolenic acids dissolved in mineral oil revealed that these compounds yield only oily, tallowy types of off-flavors. However, storage deterioration of Δ^{10} undecenoic acid resulted in the production of an aroma reminiscent of the milk fat off-flavor. Grün and Wirth (*4*) have reported that Δ^9 decenoic acid slowly decomposes on standing and develops the odor of decalactone. This observation was confirmed. Δ^9 decenoic acid was prepared from milk fat according to the bromination-debromination method of Bosworth and Brown (*3*). The acid on standing several months gave rise to the odor of δ decalactone. In addition, it was noted that odor of the lactone could be developed in 48 hr by treating the decenoic acid with 70 percent sulfuric acid as shown by Grün and Wirth (*4*). Study of various fatty acid fractions from milk fat indicated that the C_{10} fraction is the only one that will develop the typical coconut-like aroma of dry whole milk either autocatalytically or in the presence of sulfuric acid.

Additional evidence that Δ^9 decenoic acid may be involved in the flavor defect was obtained from infrared studies of milk fat recovered from dry whole milk immediately after manufacture and after one month of storage. Among the significant spectral changes in storage was a loss in absorption in the vicinity of 3.2 μ. This change can be assigned logically to a loss of the vinyl group in Δ^9 decenoic acid (*6*).

In order for the lactones of decanoic acid to be of significance from a flavor standpoint, they should be detectable at relatively low concentrations. Results using four taste observers showed that the γ and δ decalactones are roughly equivalent in flavor potency and can be detected at a concentration of 1 to 2 ppm in milk. The γ lactones of decanoic and dodecanoic

acids impart fruity flavors to milk which also are suggestive of deteriorated milk fat.

Prevention of the coconut-like off-flavor in both anhydrous milk fat and dry whole milk has been investigated at this laboratory. Antioxygenic measures such as vacuum or inert gas packing and the addition of various antioxidants have proven ineffectual in preventing this off-flavor. The only satisfactory preventive noted thus far is low temperature storage (0° C or lower) of the product. Products stored in this manner for several months rapidly develop the off-flavor when they are warmed and held at 100° C for a few hours.

These results are of a preliminary nature and the problem is currently under intensive investigation. Most approaches to problems involving fat deterioration have embraced oxidative mechanisms as a basis. The present findings do not rule out the possibility that minute quantities of oxygen are required in production of the coconut-like off-flavor of milk fat. However, they also suggest a nonoxidative rearrangement of unsaturated fatty acids to lactones as the mechanism of this flavor deterioration.

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Received December 30, 1953.

Communications

Uptake of Radiozinc by Normal and Diabetic Rat Pancreas

INCREASING interest is being directed upon the possible importance of zinc in tissue metabolism and upon a role it may play in the etiology of diabetes. Scott and Fisher (*1*) found upon autopsy a difference amounting to one-half in the zinc content of diabetic pancreas as compared to the normal. This finding is in some conflict with that of Eisenbrand and his group who reported, on a fat-free basis, differences of some 17 percent (*2*) and later (*3*) reported no statistically significant difference. A Japanese group directed by K. Okamoto has for some time put forward the view that zinc is rather directly concerned in the production and course of diabetes. Recent papers (*4, 5*) discuss their findings based upon histochemical grounds. Considerable evidence is presented in support of their view that diabetes is reflected in a deranged apportionment of available zinc in the animal body. In their view, agents such as alloxan, oxine, and dithizone are diabetogenic because of their "specific affinity" for zinc. Root and Chen (*6*) report that 8-hydroxyquinoline possessed diabetogenic activity but discuss this action on grounds other than zinc metabolism. That zinc is present in relatively large quantities in normal pancreatic tissue has been confirmed, employing radiozinc, by Chaikoff and co-workers (*7, 8*) and by Heath and Liquier-Milward (*9*).

We felt it would be interesting to scrutinize the distribution of radiozinc in a series of normal and alloxan-induced diabetic rats. Blood sugar levels were employed to follow the course of diabetes. Accordingly, 2.0 ml of an isotonic saline solution of Zn^{65} chloride were injected intraperitoneally into each of 4 alloxan-induced diabetic rats. Twenty-four hr elapsed before the animals were sacrificed. Tissues were removed, ashed, and assayed for radioactivity, employing conventional radiochemical procedures. A wide variety of tissues were retained for counting and some whose behavior appears interesting are reported in Table 1. There appears to be considerably less Zn^{65} concentrated in the diabetic pancreas as compared with the normal. This finding would seem to offer sup-

TABLE 1. Distribution of Zn^{65}, percentage of administered dose found after 24 hr in various tissues.

Tissue	Normal rat %			Av.	Diabetic rat %				Av.
	1	2	3		1	2	3	4	
Pancreas	2.3	2.6	2.8	2.6	0.2	0.3	0.4	0.1	0.3
Liver	22.0	13.5	18.1	17.9	18.3	12.5	16.5	14.3	15.4
Stomach	6.4	5.1	5.6	5.7	4.7	3.4	2.0	5.7	4.0
Gastrointestinal tract									
upper	—	4.5	—	4.5	3.6	6.2	5.8	7.9	5.9
middle	3.5	3.8	5.7	4.3	4.3	7.4	5.4	3.4	5.1
lower	3.6	4.0	7.1	4.9	5.3	5.7	5.6	—	5.6
Kidney	2.0	2.0	0.8	1.6	2.6	4.5	3.9	3.7	3.7
Brain	0.2	0.1	0.1	0.1	0.2	0.1	0.1	0.1	0.1
Heart	0.3	0.4	0.4	0.4	0.4	0.3	0.4	0.4	0.4
Spleen	—	0.3	1.1	0.5	0.3	0.3	0.2	0.2	0.3
Lung	0.7	0.8	0.7	0.7	0.9	0.8	0.6	0.8	0.8