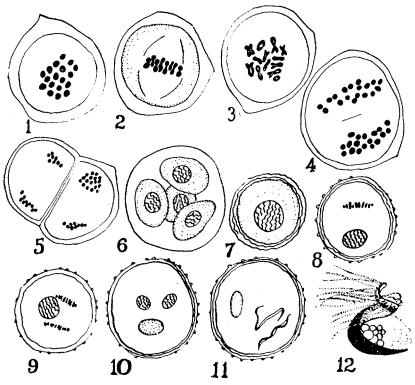

Figs. 1–12. Ragweed pollen.

every bivalent chromosome (Fig. 3). At the end of the first anaphase, the partition wall begins to appear between the two newly formed chromosome groups (Fig. 4); at the end of the second division, it is clearly apparent (Fig. 5).

When the tetrad is formed, each cell changes into a pollen grain with a thick cell wall (Figs. 6 and 7). After the first division of the pollen nucleus, a vegetative nucleus and a germ nucleus are produced, and the germ nucleus once more divides into two male nuclei (Figs. 8–10).

Each of the two male nuclei gradually change into the long, banded, sharp-pointed structures. One side of the banded nucleus is stained especially deeply. the band-shaped nuclei form spirals and resemble the spindle-shaped spermatozoids of a fern or a moss (Fig. 11). I have studied the cytomorphological features of many species of ferns (*1*), and a spermatozoid of *Alsophila martensiana* (Fig. 12) shows some resemblance to the male nucleus of ragweed (*2*). The male nucleus of Angiospermae corresponds to the spermatozoid of a fern or a moss, and it is interesting from the viewpoint of phylogeny that the male nuclei of an *Ambrosia* should show a spiral form.

In *Fritillaria* (*3*), in *Lilium* (*4*), and in *Monotropa* (*5*), the male nuclei become spiral-shaped after they enter the embryo sac, but this is the first instance in which the male nucleus of a higher plant has been found to exhibit a spiral form in the pollen.

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* Fulbright grantee (1955) from the University of Tokyo.

3 October 1955

Genus Haemagogus in the United States

We have been engaged during the past 6 years in the study of the tropical American mosquitoes of the genus *Haemagogus* (Diptera, Culicidae) that are associated with the wave of sylvan yellow fever that passed through Panama during the period 1948–51 and in 1954 reached the north coast of Honduras.

In the course of field work in Middle America, we came to realize that this genus, which had been studied primarily in the tropical rain forests of South America, includes species characteristic of very different ecological situations. In southern Mexico, near Tuxtla Gutierrez, we found two species of *Haemagogus* at elevations in excess of 4000 feet, associated with a semiarid scrub-type of vegetation. This led us to believe that there were members of the genus that might inhabit similar situations at lower elevations to the north of the Tropic of Cancer. We have been interested in determining the northern limits of the distribution of these mosquitoes because of their implication in the transmission of sylvan yellow fever.

After reviewing available information on the physiography, climatology, and vegetation of the Mexican gulf versant, we selected several areas in the Rio Grande basin for survey in late August and early September of last year, when rainfall and temperature conditions would be most favorable for the breeding of *Haemagogus* (*1*). One of these areas was the delta region of the Rio Grande in the vicinity of Brownsville, Tex. This area is largely under intensive cultivation, but we were able to find occasional patches of thorny scrub vegetation along relatively moist depressions that are locally known as "resacas." Larvae and pupae of *Haemagogus equinus* were collected from water in three tree holes in a patch of thorn scrub off Texas State Highway 48 near the intersection with Farm Road 1792, 5 miles northeast of Brownsville (4 and 6 Sept. 1955); and from a tree-hole 15.7 miles east of Brownsville on Boca Chica Boulevard (6 Sept. 1955). By 8 Sept., adult males and females had already emerged. This material will be deposited in the United States National Museum and the collection of the Gorgas Memorial Laboratory. Because of the pressure of other field work scheduled in Mexico, no attempt was made to seek *Haemagogus* futher north in Texas.

Haemagogus equinus, which occurs at least as far south as Colombia, is a proved vector of yellow fever in the laboratory, but virus has not been recovered with certainty from it in nature. It was, however, the only species of *Haemagogus* found by us in immediate association with the epizootic of yellow fever on the northern coast of Honduras in 1954 (*2*).

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27 September 1955

Loss of Sebaceous Glands in Skin of Thiamine-Deficient Mice

To study the effects of thiamine deficiency in mouse skin with resting or growing hair follicles, approximately 60 young adult C57 black mice of both sexes were used. The mice were kept in individual wire metabolism cages and offered tap water ad libitum.

Two kinds of thiamine-deficient diets were used—diet 227 (Table 1), obtained from Paul Fenton of Brown University (*1*) and a diet purchased from General Biochemicals, Inc. (Table 2). Pair-fed animals—that is, animals fed the normal diet in amounts equal to those consumed by their respective paired mates that were fed the thiamine-deficient diet, served as controls.

Biopsy specimens were removed on approximately the 7th, 14th, and 21st days of the deficiency regimen. The skin was shaved, and approximately 1 cm^2 was removed, spread on a piece of cardboard, cut in half, and fixed in 10-percent formol calcium. One half was prepared for histological study and stained in an

Table 1. Components of diet 227. In the control diet, 10 mg of thiamine was added to each 1000 g of diet 227.

Component	Amount
Casein (vitamin-free)	300 g
Dextrin	500 g
Salts (Hubbel *et al.*)	50 g
Corn oil	50 g
Vitamin supplement per 1000 g of diet	
Pyridoxine	10.0 mg
Riboflavin	20.0 mg
Nicotinic acid	50.0 mg
Biotin	0.2 mg
Folic acid	5.0 mg
Pantothenic acid	100.0 mg
Vitamin A	10,000 U.S.P. units
Vitamin D	1,000 U.S.P. units
Vitamin E	50.0 mg
Vitamin K	10.0 mg
Choline	1.50 gm

Table 2. Components of diet purchased from General Biochemicals. In the control diet, 10 mg of thiamine was added to each 1000 g of the GBI diet.

Component	Amount
Sucrose	68%
Casein, vitamin-free (GBI)	18%
Vegetable oil (hydrogenated)	10%
Salt mixture U.S.P. XIV	4%
Vitamin supplement (g/100 lb)	
Vitamin A conc. (200,000)	4.500 g
Vitamin D conc. (400,000)	3.000 g
Alpha-tocopherol	10.215 g
Choline chloride	272.400 g
Niacin	27.240 g
Inocitol	13.620 g
2-Methyl-1, 4-naphthoquinone	0.1021 g
Pyridoxine HCl	0.9534 g
Riboflavin	0.9534 g
Calcium pantothenate	2.0430 g

aqueous solution of toluidine blue, and the other half was stained for lipids in a solution of sudan black B. A total of 95 biopsy specimens was studied.

To insure that the mice would possess hair follicles at the proper stage of hair growth throughout the course of the experiments, club hairs were plucked from the resting follicles. Plucking of club hairs initiated a new wave of hair growth that required approximately 19 days for completion (*2*) in the plucked area only. The mice were placed on the thiamine-deficient diet at 21 days after plucking. The hairs at this time had just completed their growth and entered the resting phase and would remain in the resting phase for about 1 month (*2*). After 7 or 14 days of the deficiency regimen, one half of the dorsum of each mouse was again plucked, and in this replucked area hair growth was again initiated. Thus, throughout the remainder of the experiment, one half of the dorsum of each mouse had growing follicles and the other half had resting follicles.

Mice that were fed the deficient diet tended to increase in weight throughout the first week and level off during the second. About the 14th day, a progressive loss of weight began, and death occurred between the 21st and 25th days. Concomitant with the decrease in weight in the third week, the hair coat lost its smoothness, and the hairs felt dry to the touch. Although the pair-fed control animals lost much weight, their hair coat remained smooth and oily.

Biopsy specimens of skin from both deficient and pair-fed control animals showed essentially a normal appearance 7 days after the initiation of the deficiency regimen.

Skin that possessed growing or resting follicles, removed from both groups of mice at 14 days, exhibited a general atrophy that is primarily the result of a decrease in the size of the panniculus adiposus. The epidermis and its appendages were not appreciably reduced in size, and there was abundant lipid within the sebaceous gland.

Between the 21st and 25th days of the deficiency, a marked atrophy of the skin was seen, irrespective of whether the skin had growing or resting follicles. The panniculus adiposus had disappeared, the epidermis was reduced to a single thin layer of cells covered by a thin film of keratin, and the hair follicles, both growing and resting, were decreased in size. The sebaceous glands had atrophied, occasionally leaving a thin shell of flattened cells comprising the peripheral, basal, and undifferentiated cells of the gland. Lipid could not be demonstrated in these remnants of the glands (Fig. 1). Occasionally, however, a plug of lipid was seen in the duct of the gland and hair canal, which probably represents sebum previously synthesized but not extruded.

The pair-fed controls also showed a loss of the panniculus adiposus and a general decrease in size of the epidermis and its appendages. However, the sebaceous glands, although reduced in size, were intact, and intracellular lipid was demonstrable (Fig. 2).

One possible explanation for the loss of the sebaceous glands is that they are holocrine glands. In order to synthesize sebum, they need a constant supply of energy and materials to be used for mitosis and for the synthesis of the sebum. It is now established that the energy for mitosis of the cells of the epidermis and its appendages is produced to a great extent by the activity of the Krebs citric acid cycle (*3*). Since thiamine is an integral component of the Krebs cycle and of steps in intermediary metabolism leading into the cycle, a deficiency of this key component could conceivably disrupt or impair the function of the energy-producing mechanism. The loss of available energy would make continued mitosis impossible, and therefore, without continued cell replacement, the sebaceous gland would atrophy.

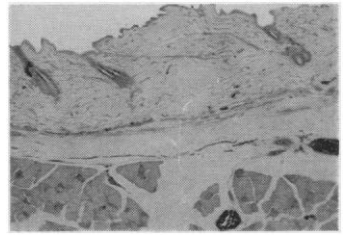

Fig. 1. Sudan black B preparation of skin 24 days after the initiation of the deficiency regimen. No sebaceous glands are present. × 50.

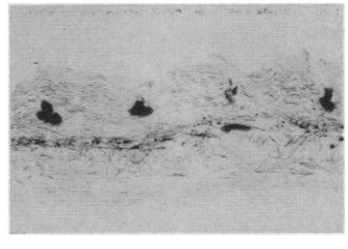

Fig. 2. Pair-fed control 22 days after initiation of deficiency regimen. Black areas are portions of the sebaceous glands stained for lipids with sudan black B. × 50.

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Congenital Galactosemia, a Single Enzymatic Block in Galactose Metabolism

The pathway of galactose to glucose-1-phosphate includes the following steps (compare Leloir, *1*, and Munch-Petersen *et al.*, *2*): (i) phosphorylation of galactose; (ii) incorporation into nucleotide; (iii) inversion of the 4-hydroxyl group; and (iv) release of glucose-1-phosphate. The following scheme summarizes these consecutive reactions (*3*):

$$\text{Gal}^* + \text{ATP} \rightharpoonup \text{Gal}^*\text{-1-P} + \text{ADP} \quad (1)$$

$$\text{Gal}^*\text{-1-P} + \text{UDPG} \rightleftharpoons \text{G-1-P} + \text{UDPGal}^* \quad (2)$$

$$\text{UDPGal}^* \rightleftharpoons \text{UDPG}^* \quad (3)$$

$$\text{UDPG}^* + \text{PP} \rightleftharpoons \text{UTP} + \text{G}^*\text{-1-P} \quad (4)$$

The enzymes that catalyze these four reactions are galactokinase, PGal-uridyl transferase, Gal-waldenase, and PP-uridyl transferase, respectively. Because the galactose was labeled with C^{14} (the asterisks indicate the C^{14}-labeling of the hexose moiety), the equations illustrate how this sugar is finally brought into the general carbohydrate metabolism.

Schwartz *et al.* (*4*) have described that galactose administration to infants who are afflicted with congenital galactosemia brings about a marked accumulation of a galactose-l-phosphate in the erythrocytes.

Most recently it has been found (*5*, *6*) that hemolyzates from infants with congenital galactosemia are devoid of the enzyzme in Eq. 2, PGal-uridyl transferase. However, it was not possible at that