The amine oxidase reaction was linear with enzyme concentration over a range of 0 to 0.20 ml of enzyme preparation.

The rate of tryptamine-2-C^{14} oxidation by homogenates prepared from epicotyls of Alaska (tall) and Little Marvel (dwarf) peas was reduced 40 to 65 percent (Table 2). The inhibition for treated Alaska pea epicotyls was 51 percent at 88 hours and 43 percent at 163 hours. A similar comparison of Little Marvel control and treated pea epicotyls showed 65 percent and 47 percent inhibition of the rate of tryptamine-2-C^{14} oxidation at 88 and 163 hours, respectively. Practically no tryptamine-2-C^{14} oxidation (less than 3 percent of the 88-hour-old epicotyls) was detectable in epicotyl homogenates of control or treated peas at the 54-hour stage of development. The rate of tryptamine-2-C^{14} oxidation was substantially higher in homogenates from tall pea epicotyls than in similar enzyme preparations of dwarf epicotyls at both the 88- and 163-hour stages. Similar experiments were performed with Alaska and Little Marvel peas which had been treated when 9 days old by applications of 400 μg of B-995 per plant to the shoot apices. A marked effect of B-995 on the capacity of shoot tips to oxidize tryptamine-2-C^{14} was revealed when the shoot tips were assayed 8 days after application of B-995.

The effect of UDMH on the rate of tryptamine-2-C^{14} oxidation by homogenates of control Alaska pea epicotyls 88 hours old was investigated by using a range of concentrations from 1×10^{-8} to $1 \times 10^{-4}M$ UDMH. A concentration of approximately $3.3 \times 10^{-7}M$ of UDMH resulted in 50-percent inhibition of the rate of tryptamine-2-C^{14} oxidation (Fig. 1). Thus, hydrolysis of less than 0.1 percent of the administered B-995 at the hydrazide group could produce a level of UDMH adequate to cause a 50-percent inhibition of tryptamine oxidation. Work by Martin *et al.* (*8*) with C^{14}-labeled B-995 indicated that the growth retardant underwent a slow decomposition in apple seedlings. Clarke and Mann (*9*) established the oxidation of tryptamine to indoleacetaldehyde by diamine oxidase purified from pea seedlings. Therefore, we assume that tryptamine oxidation in our experiments occurred via diamine oxidase.

We conclude that the inhibition of shoot elongation in peas by B-995 can be correlated with the inhibition of tryptamine oxidation by way of diamine oxidase.

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Hereditary Absence of Sebaceous Glands in the Mouse

Abstract. *An autosomal recessive mutation, characterized by an absence of sebaceous glands, and by hyperkeratosis, alopecia, and single (rather than the usual multiple) hair-follicle units, has occurred spontaneously in the BALB/c strain of mouse. Studies in which reciprocal transplantations of skin were made between normal and mutant mice suggest that some diffusible substance(s) synthesized by normal skin can stimulate hair growth and alleviate the hyperkeratosis characteristic of the skin syndrome.*

Disturbances of the keratinization process constitute an important group of skin diseases of man, and in many cases the disorders are known to be genetically controlled (*1*). Mutations that affect the skin, hair, or both, also occur in laboratory animals. However, such mutations, particularly those affecting the keratinization process, are not common, and descriptions of the disorders have been limited to morphology (*2*). The role of skin appendages, such as the sebaceous gland, in maintaining or promoting keratinization is not known. We were therefore interested in the spontaneous appearance, in an inbred strain of mice, of a previously undescribed mutation in which the animals affected were characterized by the complete absence of sebaceous glands. In this report we describe the physiology and dermatopathology of the condition during the life of an affected animal and present evidence for the mode of inheritance.

The mutant mice originated from a sibmated colony of strain BALB/cCrglGa (albino) maintained by one of us (A.H.G.). The spontaneous mutation initially appeared at F_{74} in litters from two different pairs of parents which were two generations removed from a common ancestor. Test matings provided unequivocal evidence (Table 1) that the condition is controlled by a single (autosomal) recessive gene with complete penetrance. The gene has been named *asebia* (without sebum) and the letters *ab* have been adopted provisionally to symbolize the mutation (*3*). Brother-to-sister matings, with forced heterozygosity of the gene, have been continued since initial discovery of the mutation. The subline has now been maintained for six generations and will be designated BALB/cGa-+*ab*.

Normal (heterozygous) and mutant mice in infancy and adulthood are illustrated in Fig. 1, *A* and *B*. The condition may be recognized as early as

Table 1. Number of mutants lacking sebaceous glands and total offspring from various matings. Proportion of mutants observed and expected (based on the hypothesis that a single recessive gene is responsible for the condition).

Type of mating	No. of offspring		Proportion of mutants		χ^2	p
	Mutant	Total	Obs.	Exp.		
Mutant × mutant	45	45	1.00	1.00		
Mutant × normal (F_1)	0	87	0.00	0.00		
Carrier × carrier (F_2)	37	122	.30	.25	1.8	>.1
Carrier × mutant (backcross)	88	176	.50	.50		

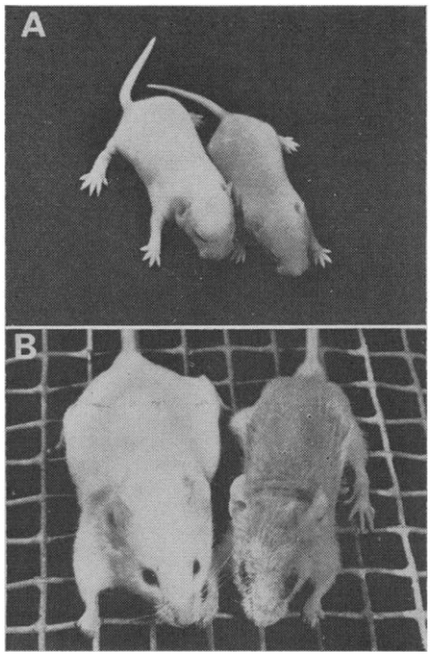

Fig. 1. Comparison of littermate female mice homozygous for the sebaceous gland defect (right) with normal heterozygotes (left). Darkened appearance of the mutant mice results from skin showing through the short, sparse, albino hairs. Wire mesh background is of ½-inch (1.27-cm) squares. (*A*) Mice aged 7 days, the earliest age for phenotypic classification; normal mouse on left weighed 6.2 g and the mutant, 4.8 g. (*B*) Mice aged 11 months, the mutant (right) with hyperkeratosis and eyelid lesions typical of old age; the normal mouse weighed 35.3 g and the mutant, 26.6 g, both nonpregnant.

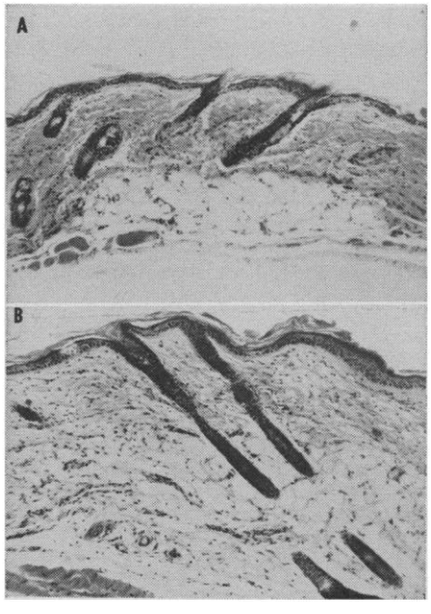

Fig. 2. Comparison of stained (hematoxylin-eosin) dorsal skin sections from normal (*A*) and mutant (*B*) adult mice (× 200). Mutant mouse skin shows no normally developed sebaceous glands. The hair follicles contain strongly basophilic material and extend deep into the fat tissue.

7 days of age by impaired growth of the coat (Fig. 1*A*), but in retarded litters, classification is not possible until 9 days. Alopecia increases until adulthood, when loose, fine scales appear among the very sparse hairs. As the animals age, blindness ensues. Pruritus is apparently limited to the eyes where, in older animals, inflammation and definite scratch marks can frequently be seen (Fig. 1*B*). The teeth, claws, and vibrissae are normal. Generally, the variability in severity of the disease is small; however, animals with a more severe degree of scaling occasionally appear.

The mutant animals show no evidence of sebaceous-gland development (Fig. 2), the one pathological feature thus far found to be present throughout life (complete serial sections have been made). The base of the follicle sometimes exhibits excessive development, but hair production is faulty. The centers of many follicles are plugged with keratotic material. The follicles extend deep into fat tissue and single hair-follicle units rather than the usual multiple hair-follicle units are observed.

The mutants are usually retarded in growth throughout their lifespan. At weaning, they weigh, on the average, 1 g less than normal littermates (average weight, 13 g for normal females 3 weeks old; litter size, 6). Growth is most retarded when the young are born to mutant mothers. Compared to normal littermates, affected offspring are more susceptible to infantile diarrhea, the incidence approaching 100 percent in litters from mutant mothers. Retarded growth is considered to be a secondary characteristic since it is a concomitant feature of other known alopecia mutants in the mouse.

When male and female mutants are paired at random, only infrequent copulations occur. This results largely from the long diestrous periods characteristic of those females which are developmentally retarded. However, after rigorous selection for near normal health and body size at weaning (discarding about 40 percent of the weanlings) a reasonable degree of fertility is observed. All (of 49) males so selected proved to be fertile. Among 32 mutant females similarly selected at weaning, 81 percent bore litters (average of 4.8 young); among 38 control heterozygous females 100 percent bore litters (average of 6.2 offspring). Although, on the average, fertility is reduced among mutant females, some selected individuals appear to be fully normal in their reproduction.

Figure 3 shows typical results of reciprocal transplantation of skin between normal and mutant mice. The normal mice which received skin from mutant donors were F_1 hybrids of the cross between strains 129 and BALB/c and bore coats of gray hair because of their heterozygosity for chinchilla and albinism. Mutant hosts received normal skin from donors of the albino strain BALB/c. All graft beds were 1 cm square. New hair growth occurred on both types of grafts (Fig. 3), but was retarded for several weeks in grafts of skin from mutant donors. Of particular interest was the fact that increased hair growth and absence of hyperkeratosis were noted on the skin of mutant recipients along a margin 0.3 cm wide around the graft. This marginal growth of hair may be seen in the lower mouse in Fig. 3; the graft was so placed as to orient its hair growth at right angles to that of the host. The increased hair growth on mutant skin occurred without the development of sebaceous glands (determined by complete serial sectioning). In control homotransplantations, no stimulation of hair growth or prevention of scaling was observed. These findings show that wounding alone was not responsible for the new hair growth and suggest that some diffusible substance(s) which can partially alleviate the abnormal symptoms in the mutant skin syndrome may be synthesized by normal skin.

Mutant mice totally lacking sebaceous glands can be of considerable value in studies of epidermal lipid synthesis. Chemical analyses of skin surface lipids,

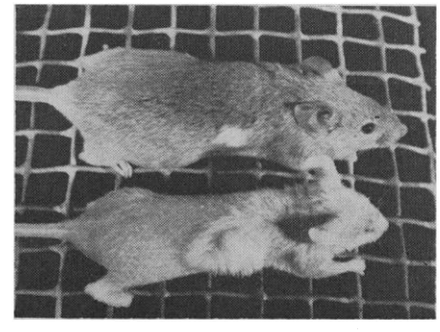

Fig. 3. Results of reciprocal transplantation of skin, performed 5 months previously. (Top) Normal mouse (genotype $c^{ch}c$) bearing graft from a mutant donor (albino). (Bottom) Mutant host (albino) received skin from a normal albino donor. Hair growth on the mutant recipient's own skin occurs along a margin 0.3 cm beyond the graft bed as identified by the orientation of the hair shafts.

uncomplicated by the relatively large and unkown contribution of sebaceous-gland products (*4*), were not possible previously.

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Paired Comparison Method for Measurement of Sugar Preference in Squirrel Monkeys

Abstract. *The glucose preference of four squirrel monkeys was determined by presenting 5-, 10-, 20-, 30-, and 40-percent solutions according to the paired comparison method. Scaling of the data for the amounts of each solution consumed yielded a preference function which increased monotonically with the concentration of the solutions.*

In previous studies of food preference it has not been uncommon for the results to vary according to the method used. The two methods most often used as an indication of the kind of food preferred involve measuring the amount of each type of food consumed or observing the operant (bar-pressing) behavior expended to obtain the different foods. These two methods often yield conflicting conclusions [see Young and Greene (*1*) as opposed to Stebbins *et al.* (*2*)] and this is the case especially within variations of the latter method (*3*). An example of a difference in preference produced by a difference in procedure is furnished by Guttman (*3*), who found that the rats rewarded at 1-minute intervals would bar-press more for a 32-percent sugar solution than they would under a continuous reinforcement schedule.

When the amount of sugar solution consumed is measured, the results indicate that most animals prefer liquids of low to intermediate concentrations. When the response rate in operant situations is measured, it increases with higher concentrations of sugar solutions. (It is usually assumed that a higher response rate indicates a greater preference or value attached to the food offered as a reward for the bar-pressing.)

Although based on measurements of substances consumed, the paired comparison method (*4*) allows relative intake of the different concentrations to be scaled, and thus may provide a link between preference functions obtained by the original two methods. Therefore, we have evaluated the paired comparison method as a means of determining sugar preference in squirrel monkeys.

Five concentrations of glucose solution, 5-, 10-, 20-, 30-, and 40-percent, were made by mixing anhydrous glucose with tap water on a weight-by-volume basis suggested by Pfaffmann *et al.* (*5*). Four young male squirrel monkeys (*Saimiri sciureus*) (*6*) were tested in their home cages 2 to 4 hours before they were given their daily meal. Every day, each subject was allowed to drink from a pair of solutions which were presented simultaneously in graduated cylinders with metal drinking tubes. The pairings were random, such that over a period of 25 days every solution was paired with every other solution twice and with itself once, thus allowing counterbalancing of position and determination of position preference. During the 1-hour test period, the animals had no access to water and readings were taken at 10-minute intervals.

Figure 1 shows that the curves representing the mean amounts of each solution consumed after 10 minutes and 60 minutes are nearly parallel, with almost 80 percent of the total intake occurring in the first 10 minutes. Wagner (*7*) has also found that satiation influences rate of intake over extended time periods but does not affect relative preference for sugar solutions. The fact that these consumption curves have maxima at intermediate concentrations indicates that they are typical for the measure used and that squirrel monkeys have generally the same preference function as other, better-tested subjects. Since the curves for 10 minutes and 60 minutes resemble each other closely, and since the data for 60 minutes are confounded by a satiation effect, the remainder of this report is based on the data obtained after 10 minutes.

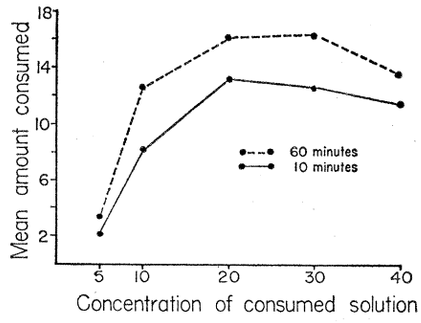

Fig. 1. Mean amount of each glucose solution consumed after 10 minutes and after 60 minutes.

The effect of solutions presented for comparison can be seen in Fig. 2, where a general tendency is shown for less of any given solution to be drunk when the solution with which it is paired increases in concentration. It is of interest that when each solution, except the 5-percent solution, was paired with itself the amounts consumed were nearly equal, and only a slight tendency to

Table 1. Comparison of obtained proportions with estimated proportions for choices of solutions with the higher concentrations.

Percentage concentration of comparison solution	Percentage concentration of consumed solution				
	5	10	20	30	40
5		0.875 *.999**	1.000 *0.999*	1.000 *0.999*	1.000 *0.999*
10	0.125 *.000*		1.000 *.999*	.875 *.999*	.750 *.999*
20	.000 *.000*	.000 *.000*		.750 *.993*	.750 *.998*
30	.000 *.000*	.125 *.000*	.250 *.000*		.625 *.667*
40	.000 *.000*	.250 *.000*	.250 *.002*	.375 *.323*	

* Numbers in italics are estimated proportions.