larity between serum and mitochondrial beta lipoproteins in their affinity for TTC parallels the affinity of the beta lipoproteins in these two locales to complex with a large variety of enzymes(14,15).

Summary. The localization of tetracycline labeled with tritium in the subcellular fractions of rat liver cells has been investigated. Ultracentrifugal analysis of cellular components revealed a significant and characteristic concentration of the drug in all fractions. Electrophoresis on paper and starch blocks of detergent-solubilized mitochondrial proteins revealed a distribution of the tetracycline, as determined by fluorescence and radioactivity, which coincided with the beta lipoprotein fraction of the mitochondrial lysate. Tetracycline in the presence of calcium ions is capable of precipitating the beta lipoprotein fraction of mitochondrial lysate as well as that of serum. The localization of TTC in fractions other than mitochondria is probably not related to complexing with lipoprotein.

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The Merocrine Component of Apocrine Secretion.* (28431)

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Apocrine sweat gland function has been divided into 2 separate and distinct activities, apocrine sweating and apocrine secretion(1). Apocrine sweating, the appearance of apocrine sweat on the skin surface, occurs when the apocrine myoepithelium is stimulated to contract, with resultant expression of preformed sweat from the apocrine tubule. Implicit in this concept is the belief that no active formation or secretion of sweat takes place at the time of apocrine sweating. Apocrine secretion, the formation of apocrine sweat by the secretory cells, is believed to be a slow, virtually continuous process, requiring hours to a few days to fill the tubular reservoir. It has been contrasted with the secretion of eccrine sweat which is not pre-formed but is rapidly produced at the time of eccrine sweating. Although apocrine secretion is presumed to be regulated or paced by hormonal substances, as yet not precisely defined, the cellular mechanisms involved in the formation of apocrine sweat have never been elucidated.

Recently we have been examining the excretion of locally introduced fluorescein sodium by the apocrine sweat glands. Within 3 minutes after its local intracutaneous injection, fluorescein may be detected in apocrine sweat. This prompt incorporation or excre-

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tion of dye in apocrine sweat suggests that apocrine sweat formation includes a fluid component produced immediately at the time of stimulation of apocrine sweating.

Materials and methods. The shaved axillae of 8 Negro and 4 white healthy, adult males were used as the test sites. The dome or central portion of the axillae of each man was atropinized (0.2 mg atropine sulfate) to prevent an eccrine response. Ten minutes later, an injection of 0.5 cc of 10% fluorescein sodium in physiological saline, with sodium bicarbonate added (final pH – 7.8), was made into the deep dermis or subcutis of one axilla of each subject. Diffusion and distribution of the dye to the skin and throughout the test area generally required 3 minutes and was observed under Wood light (3600Å). Apocrine sweating was then stimulated by the intradermal injection of 0.05 cc of 0.1% epinephrine hydrochloride in physiological saline in the central portions of the dye-treated and opposite axillae.

Observations were made grossly and under stereoscopic microscopy (10-30 ×). Apocrine sweat was identified by the criteria enumerated previously, *viz.*, appearance primarily of follicular orifices, turbidity, shape of sweat droplet and rapid drying to produce glue-like residue(1). Samples of sweat were also collected in capillary tubes to aid in identification.

Results and discussion. Within 30-120 seconds following the epinephrine injection, the normal latent period for apocrine sweating, fluorescein-stained apocrine sweat droplets were evident in the dye-treated axillae of 7 of the Negro and 3 of the white subjects. After an additional 1-2 minutes, the other Negro and white men also showed dye-stained apocrine sweat. As they dried in glue-like caps over the follicular orifices, these dye-stained droplets were wiped away, and new fluorescein-tinted apocrine sweat droplets appeared. This process of drying of apocrine sweat, its removal and the emergence of more apocrine sweat from these orifices continued, with ever diminishing amounts of sweat, for 20-40 minutes following the single injection of epinephrine. The overall response was generally more dramatic in the Negroes, who characteristically have larger apocrine sweat glands and more profuse apocrine sweating(1).

In all of the subjects it was noted that some apocrine sweat droplets were not dye-stained initially, but as the first-formed droplets dried and were wiped away, subsequent sweat droplets appearing at these follicular orifices contained fluorescein. Some follicular orifices, however, while clearly showing an adequate apocrine response, never demonstrated sweat droplets stained with fluorescein, possibly because of poor diffusion of dye to these glands. It is significant that as the dye and epinephrine diffused in pseudopod-like extensions into the surrounding axillary skin, new dye-stained apocrine sweat droplets could be seen. Other peripheral glands, however, to which dye had apparently not diffused, as noted by the absence of fluorescein-staining of the skin in the region of these glands, produced only unstained sweat droplets.

The opposite axilla of all subjects showed a classic apocrine response but was without evidence of fluorescein in the sweat. Although a few Negro subjects showed some fluorescence of sweat droplets in the axilla not receiving local fluorescein, this was the typical dull yellow fluorescence seen normally in some samples of apocrine sweat and was easily differentiated from the brighter orange or yellow-green fluorescence due to the fluorescein seen in the opposite axilla.

The axillae of these subjects were reexamined daily for 7 days, after daily stimulation with epinephrine. Increasingly-reduced concentrations of fluorescein continued to appear in the apocrine sweat of the dye-treated axilla of most of the subjects through 3-4 days. After 5-6 days, none could be detected. The opposite axilla failed to show any fluorescein-stained apocrine sweat throughout the 7-day observation period.

To date the processes of apocrine and eccrine sweat secretion have been examined chiefly from a histologic or cytologic approach(2,3). Interpretation of these microscopic observations seems to separate apocrine from eccrine sweat formation and suggests different secretory dynamics for each

gland. Rapid excretion of locally-introduced dye by the eccrine sweat glands, as described earlier(4), is consonant with the physiologic activity of these glands in the formation, over long periods, of large quantities of a fluid composed principally of water. Prompt excretion of locally introduced dye by the apocrine sweat glands suggests that these glands also have the capacity to form sweat, in part at least, immediately, *i.e.*, at time of apocrine sweating. However, the apocrine sweat glands produce a turbid or milky secretion containing significant concentrations of protein, lipid and carbohydrate in addition to ammonia and electrolytes. It seems likely that these substances are formed or secreted slowly by the secretory cells with a fluid or merocrine component added, perhaps by simple diffusion, at the time of apocrine sweating as indicated by the dye excretion studies. Variations in turbidity, color and natural fluorescence of apocrine sweat in the same individual could readily be explained on the basis of this theory of apocrine secretion.

In view of these observations, it would be well carefully to reexamine the apocrine sweat gland microscopically in an effort to delineate the cytologic mechanisms responsible for the proposed phases of apocrine sweat secretion. Histologic study of dye-treated skin is in progress but has not yet revealed the presence of selective channels, canaliculi, secretory vacuoles or other structures which could readily explain the merocrine component of apocrine sweat secretion.

Summary. The prompt excretion of locally-introduced fluorescein sodium in axillary apocrine sweat has been described. This observation suggests that the secretion of apocrine sweat includes a fluid or merocrine component formed at the time of apocrine sweating.

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The Citrate Content of Teeth. (28432)

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Fluoride has been shown to increase the "crystallinity" of bone apatite of the rat (1-3) and of the human(4,5) with a concomitant decrease in the citrate content of the bone(6-7). The increase in "crystallinity," *i.e.*, a decrease in specific surface and/or reduction in strain would produce a less reactive and a more stable crystal structure. If a similar situation obtains in dental enamel (8), another parameter would be introduced to help elucidate the efficacy of fluoride in reducing caries. Preliminary information, however, is desirable on the citrate content of dental tissues of various species before the relation of "crystallinity" to fluoride may be studied. Data on citrate content of human teeth appear to be limited to the work of Free(9), Zipkin *et al.*(10), Stack(11) and Hartles *et al.*(12) indicating that dentin and enamel contain about 0.9% and 0.1% citrate, respectively. In addition, Thunberg(13) has reported about 0.5% citrate in the teeth of cats, cattle, and swine, and Free(9) found 0.54% and 0.11% citrate in dog's dentin and enamel, respectively. Leaver *et al.*(14) recently reported that rat incisor dentin and enamel contained 0.046% and 0.032% citric acid, respectively, while the molar dentin con-