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Preface

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Contents

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Introduction

The composition of human perspiration has been studied and reported extensively in the medical literature. The medical community has analyzed sweat for many purposes, including attempts to diagnose certain diseases, such as cystic fibrosis, and studies of skin conditions, such as acne. Even the perfume and cosmetics industry has an interest in determining the precise chemical nature of perspiration and how it might interact with their personal hygiene products. However, the information ascertained in these studies does not begin to address the issue that is most critical for forensic scientists. Knowing the precise contents of the various skin glands does not accurately represent the nature of what is actually secreted onto substrates from the fingers and palms. In operational scenarios, numerous contaminants are present in the fingerprint deposit, including material from other glands, cosmetics, perfumes, and food residues. In addition, the secreted material is almost immediately altered by oxidative and bacterial degradation mechanisms. These factors are particularly important since crime scene technicians seldom encounter latent print deposits immediately after they are deposited by a perpetrator. However, there is little information available that describes how a latent print deposit changes with time. Thus, a more thorough understanding of these transformations would allow forensic scientists to develop specific reagents for visualizing compounds known to be stable for long periods of time.

Skin Anatomy

Skin serves several functions, including regulation of body temperature, water retention, protection, sensation, excretion, immunity, blood reservoir, and synthesis of vitamin D (except where noted, the information in this section was obtained from Odland[1]). The skin of an average adult exceeds 2 m^2 in area; yet, in most places it is no more than 2 mm thick. While the average thickness of epidermal skin varies little over most of the body, the thickness on the palms and soles can be as much as 0.4 to 0.6 mm. The skin is usually divided into two distinct layers. The outer layer is a stratified

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The Dermis

The dermis is a moderately d
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epithelium called the epidermis, which has an average thickness of 75 to 150 μm. The underlying layer of skin is called the dermis, a dense fibroelastic connective tissue that constitutes the primary mass of the skin. This portion of the skin contains most of the specialized excretory and secretory glands that produce sweat. Although the dermis constitutes between 90 to 95% of the mass of human skin, the epidermis accounts for the major proportion of the biochemical transformations that occur in the skin (although structures that extend into the dermis, such as the various sweat glands and hair follicles, are also metabolically important).

The Epidermis

The epidermis (Figure 3.1) consists of several cell layers.[2] The innermost is known as the stratum germinativum (basal cell layer). It consists of one layer of columnar epithelial cells, which upon division push into the stratum spinosum. The stratum spinosum (prickle cell layer) consists of several layers that are held together by intercellular fibrils. The combined stratum spinosum and stratum germinativum are often referred to as the Malpighian layer (named in honor of Marcello Malpighi, a 17th century Italian professor and fingerprint science pioneer who first used high magnification to detail the fine structure of ridges and pores).

As these cells approach the skin surface, they begin to grow larger and form the next layer, the stratum granulosum (granular layer). Keratohyalin granules (the precursor of keratin, a fibrous, insoluble protein found in skin) are formed in this layer, which is approximately two to four cells thick. The nuclei are then either broken up or dissolved, resulting in the death of the epidermal cell and an increase in the number of cytoplasmic granules. The penultimate layer, the stratum lucidum (clear layer), is ill-defined and consists primarily of eleidin, which is presumed to be a transformation product of the keratohyalin present in the stratum granulosum. In the outermost layer, the stratum corneum (cornified layer), the eleidin is converted to keratin, which is the ultimate fate of the original epidermal cell. Keratin, which is continually sloughed off, must continuously be replaced by cells beneath it. It has been estimated that a typical individual will shed approximately 0.5 to 1 g of dead skin cells per day.[2] The total cell cycle in the epidermis is estimated to take approximately 28 days. Figure 3.2 is a stained skin section showing all of the layers of the epidermis.

The Dermis

The dermis is a moderately dense fibroelastic connective tissue composed of collagen (a fibrous protein composed of primarily glycine, alanine, proline, and hydroxyproline), elastin fibers (a fibrous protein containing primarily

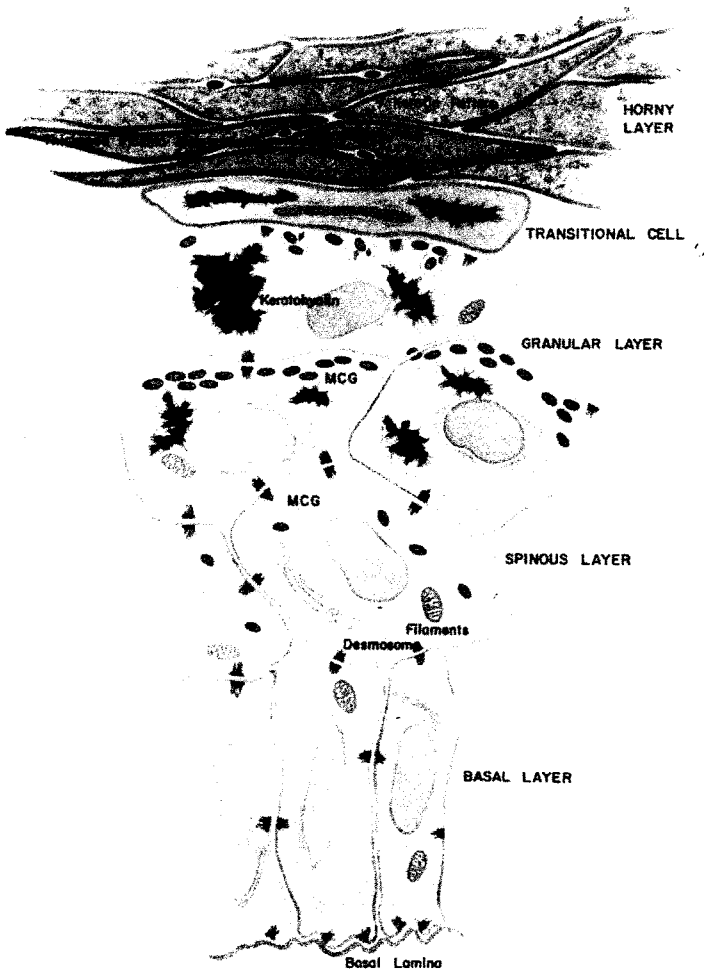

Figure 3.1 A schematic diagram showing the layers of the epidermis. (From *The Structure and Function of Skin, 3rd Edition*, Montagna, W. and Parakkal, P.F., Eds., Academic Press, 1974. With permission.)

glycine, alanine, valine, and lysine), and an interfibrillar gel of glycosamin-proteoglycans, salts, and water. This layer contains up to five million secretory glands, including eccrine, apocrine, and sebaceous glands.[2] Collagen fibers form an irregular meshwork that is roughly parallel to the epidermal surface and provides skin tensile strength and resistance to mechanical stress. Elastin gives skin its elasticity and its ability to resume its natural shape after deformation. Fibrous mats of elastin are intermeshed with collagen to give skin its tension. This tension is greatest over body areas where the skin is thin and elastin is abundant (e.g., the scalp and face). Fibroblasts, which form elastin and collagen, and histiocytes, which form interferon for protection against

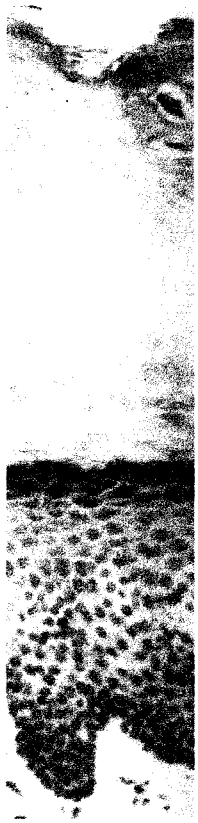

Figure 3.2 A stained section
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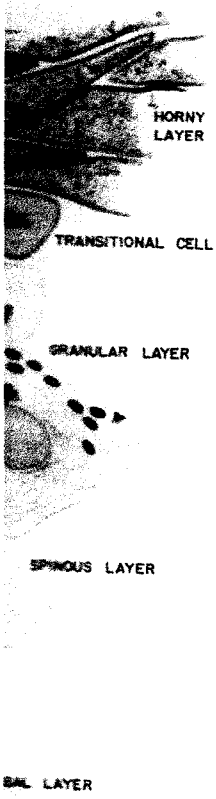

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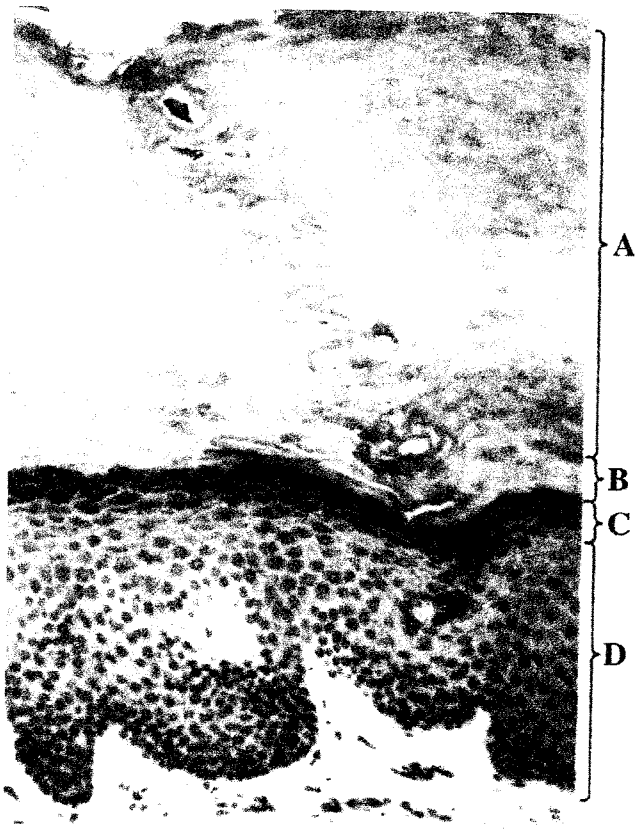

Figure 3.2 A stained section of the epidermis from the palm showing all of the layers. Section A is the stratum corneum, section B is the stratum lucidum, section C is the stratum granulosum, and section D is the stratum malpighii. The structure evident in the stratum corneum is the duct of an eccrine sweat gland. (From *The Structure and Function of Skin, 3rd Edition*, Montagna, W. and Parakkal, P.F., Eds., Academic Press, 1974. With permission.)

viral infections, are present in this layer. A system of blood, lymphatic, and nerve vessels is also present.

The dermis is divided into two anatomical regions, the pars papillaris and the pars reticularis. The papillary dermis is the outermost portion of the dermal layer and contains smaller and more loosely distributed elastin and collagen fibrils than does the reticular dermis. The papillae are supplied by numerous capillaries, which ultimately supply nourishment to the epidermis via diffusion. The second region, the reticular dermis, lies beneath the papillary dermis and comprises the bulk of this layer. It is characterized by dense collagenous and elastic connective tissue. These collagen bundles are arranged predominately in interwoven strands that are parallel to the skin surface, although some tangentially oriented bundles are present.

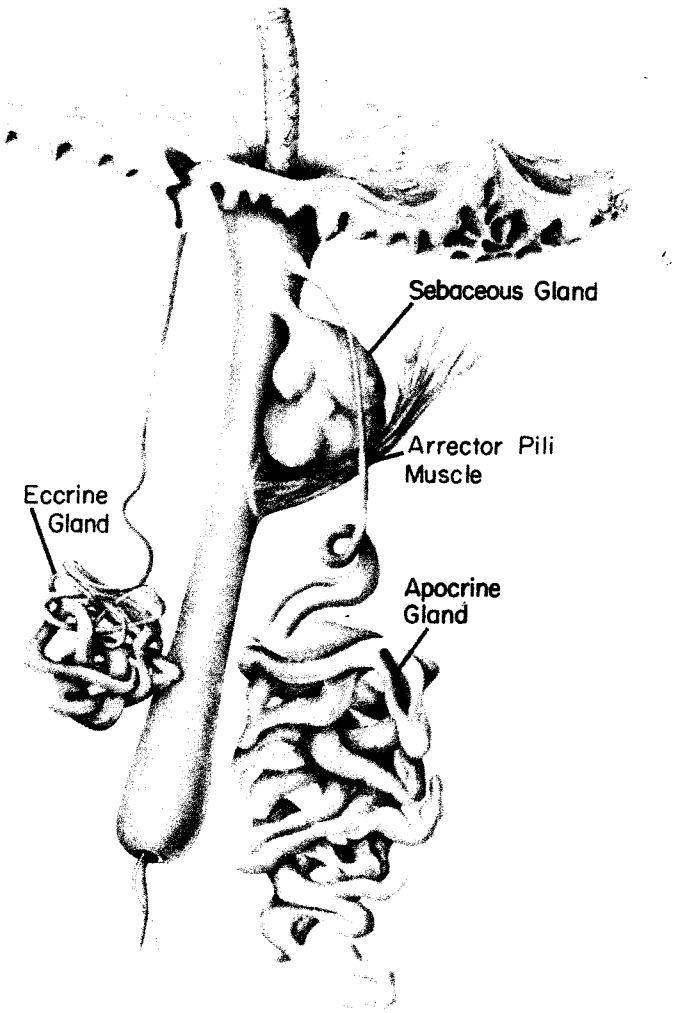

Figure 3.3 A schematic diagram of the three major secretory glands in relation to other cutaneous appendages. (From *The Structure and Function of Skin, 3rd Edition*, Montagna, W. and Parakkal, P.F., Eds., Academic Press, 1974. With permission.)

Secretory Glands

The three major glands (eccrine, apocrine, and sebaceous) responsible for the secretion of "sweat" are shown in Figure 3.3. The eccrine glands are usually found throughout the body, but the highest densities are found in the palms and soles. The sebaceous glands are typically localized to regions containing hair follicles, as well as the face and scalp. The apocrine glands

are found primarily in the
However, in most instance
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Eccrine Glands

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Inorganic Compounds

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are found primarily in the axillary regions (e.g., armpits and genital areas). However, in most instances, only the eccrine and sebaceous glands contribute significantly to the latent print deposit. Although the composition of sweat is approximately 99% water,[3] studies have shown that a considerable variety of chemical compounds are present. A recent study found approximately 346 compounds (303 of which were positively identified) present in surface skin residues.[4,5]

Eccrine Glands

There are between two and four million eccrine sweat glands distributed throughout the human body surface (except where noted, the following information was obtained from Quinton[6]). Each gland has been calculated to have an estimated weight of 30 to 40 μg, for an aggregate weight of about 100 g. In normal individuals, these glands are capable of secreting as much as 2 to 4 L of fluid per hour. The evaporation of this quantity of sweat requires approximately 18 kcal/min, which affords humans an ability to dissipate heat faster than any other animal. Sweat glands are most abundant on the soles of the feet (620/cm^2) and least abundant on the back (64/cm^2).[7] Gland formation begins around the third fetal month on the palms and soles and at about 5 months for the rest of the body. Typically, the glands have fully matured by the eighth fetal month. The eccrine gland is essentially a tubular shaped structure with a duct portion that coils in helical fashion down deep into the dermis layer. The function of the distal half of the sweat gland tubule is to reabsorb sodium, chloride, bicarbonate, glucose, and several other small solutes. Under normal conditions, this allows water to be evaporated from the skin surface without the loss of essential solutes.

Inorganic Compounds

Although eccrine sweat is usually in excess of 98% water, it also contains numerous organic and inorganic constituents. The presence of these solutes on the skin surface causes a reduction in sweat vapor pressure. These effects have been modeled and quantified.[8] Excess secretion of certain chloride salts has been reported to be a cause for increased rates of corrosion of metal surfaces by particular individuals.[9] This effect was particularly pronounced in patients suffering from hyperhidrosis, a condition which causes excess sweat production. The rate of eccrine sweating has been shown to depend on the amount of water ingested, but does not appear to exert an independent effect on the relationship of sweat composition to sweat rate.[10] Sweat has been reported to contain 0.5 to 8 m*M* total ammonia,[11] which is 20 to 50 times higher than plasma levels. In addition, trace amounts of the following inorganic substances have also been detected in sweat: magnesium, iodide

(5 to 12 μg/L), bromide (0.2 to 0.5 mg/L), fluoride (0.2 to 1.18 mg/L), phosphate (10 to 17 mg/L), sulfate (7 to 190 mg/L), iron (1 to 70 mg/L),[12] zinc, copper, cobalt, lead, manganese, molybdenum, sulfur, tin, and mercury.[13-15]

Interestingly, the eccrine gland is one of the target organs for cystic fibrosis. Historically, this condition has been diagnosed on the basis of elevated sodium chloride concentration in sweat. In general, the sweat sodium ion concentration appears to be isotonic to that of human plasma, although significant variations can be obtained depending on the method of collection (e.g., thermal vs. pharmacologically induced sweat).[16] One study found that the sodium concentration varied over a rather large range, from 34 to 266 mEq/L. Others reported the average concentration at 140 ± 1.8 mEq/L[7] and 60 mEq/L.[17] The latter source reported that the chloride concentration is generally lower than that of sodium, averaging around 46 mEq/L, and that the potassium level ranged from 5 to 59 mEq/L. In general, chloride levels are isotonic with those in plasma.[18] Other studies have determined the potassium levels to be between 4.9 to 8.3 mEq/L[16] and 8.8 mEq/L.[19] The amount of calcium in sweat was found to be about 3.4 mEq/L and the amount of magnesium was 1.2 mEq/L.

The HCO_3^--CO_2 buffer system appears to play a critical role in maintaining sweat pH. The pH of sweat isolated from human secretory coils (in the dermis) is approximately 7.2, while the pH of sweat secreted from the gland can vary from as low as 5.0 (at a low sweat rate) up to 6.5 to 7.0 (at a high sweat rate). This indicates that the duct itself acidifies the sweat, presumably by reabsorbing bicarbonate and/or secreting H^+ in exchange for a Na^+ ion.[20] At low sweat rates, this mechanism can conserve bicarbonate (and other solutes) efficiently and thus maintain a slightly acidic sweat pH. At higher sweat rates, the mechanism is overwhelmed and cannot reabsorb solutes effectively. This results in secreted sweat containing higher amounts of bicarbonate and thus it has a higher pH. The typical bicarbonate concentration has been reported to be between 15 to 20 m*M*.

Amino Acids

Of critical importance to latent print visualization with ninhydrin is the concentration of amino acids and proteins. The total amount of amino acids present in a print has been reported to be between 0.3 to 2.59 mg/L.[14] The first amino acid found in eccrine sweat was serine, isolated as β-naphthalinesulfoserine by using a microbiological method, and was reported by Embden and Tachau in 1910. A study of samples of pharmacologically induced sweat (using pilocarpine hydrochloride) collected after a hygienic bath yielded 22 amino acids.[21] Amino acid amounts in sweat have been reported to be several times higher than corresponding values in plasma.[22] One study found the most abundant amino acids to be serine and alanine,

Table 3.1 A Sum (Serine Ratio) of A

Serine
Glycine
Ornithine
(Ornithine, lysine)
Alanine
Aspartic acid
Threonine
Histidine
Valine
Leucine
Isoleucine
Glutamic acid
Lysine
Phenylalanine
Tyrosine

15.44 and 14.63 mg%, resp
participants found that in
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others.[24-26]

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acid, cystathionine, β-am
butyric acid, and carnosine

Proteins

The total protein content in
to 25 mg/dL. One study u
sensitive silver staining for
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Table 3.1 A Summary of the Relative Abundance (Serine Ratio) of Amino Acids in Fingerprint Deposits

	Hamilton[28]	Hadorn et al.[27]	Oro and Skewes[29]
Serine	100	100	100
Glycine	67	54	59
Ornithine	32	45	45
(Ornithine, lysine)	42	47	45
Alanine	27	35	28
Aspartic acid	22	11	22
Threonine	17	9	18
Histidine	17	13	14
Valine	12	10	9
Leucine	10	7	10
Isoleucine	8	6	8
Glutamic acid	8	12	5
Lysine	10	5	—
Phenylalanine	7	5	5
Tyrosine	6	3	5

15.44 and 14.63 mg%, respectively. Another study of both active and inactive participants found that in both cases, serine, glycine, and alanine were the most abundant amino acids.[23] A similar trend was also reported by several others.[24-26]

Quantitatively, amino acid concentrations can vary as much as 2 to 20 times depending on collection methods (e.g., thermally induced sweat vs. exercise-induced sweat) and by sample location on the body. A study comparing sweat samples obtained from the back and hands of subjects found some significant differences.[27] The samples from the backs of subjects showed higher amounts of amino acids involved in the urea cycle. These and other differences appeared to be independent of plasma and urine amino acid levels, suggesting that amino acids do not appear in sweat as a result of filtration from the blood plasma. Table 3.1 summarizes the relative amino acid abundance values from several different studies. One study reported a series of ninhydrin positive substances, in addition to amino acids, in human eccrine sweat.[30] Some of these substances include *o*-phosphoserine, methionine sulfoxide, α-amino-isobutyric acid, glucosamine, α-amino-*n*-valeric acid, cystathionine, β-amino-isobutyric acid, ethanolamine, γ-amino-butyric acid, and carnosine.

Proteins

The total protein content in sweat has been determined to range between 15 to 25 mg/dL. One study using two-dimensional electrophoresis and ultrasensitive silver staining found over 400 polypeptide components.[31] Some specific examples determined by sodium dodecyl sulfate polyacrylamide gel

electrophoresis (SDS-PAGE) include albumin, Zn-α_2-glycoprotein, lysozyme, and the α_1-acid glycoprotein orosomucoid.[32] An agarose gel isotachophoresis analysis of thermally induced sweat detected transferrin, fast-migrating γ-globulins, α- and β-lipoproteins, and several glycoproteins.[33] It has been determined by size fractionation HPLC that the bulk of the peptides in sweat are in the low end of the molecular weight range. Secretion of higher molecular weight proteins (i.e., in excess of 10,000 Da) has been reported to increase as the rate of sweating increases.

Lipids

The lipid content of secretions from the eccrine gland has also been investigated.[34] Contamination of samples by lipids of sebaceous and epidermal origin is a major consideration in these analyses. In this particular study, thin layer chromatography was used to separate the lipid fraction collected from both "clean" and "scraped" sweat samples. Results indicated that the "scraped" samples contained a significant amount of lipids that were consistent with those found in the stratum corneum. In contrast, the "clean" samples collected using the method described by Boysén et al.[35] contained only one significant lipid band, which corresponded to the cholesterol/fatty acid standard. In the samples collected, fatty acid concentrations ranged from less than 0.01 to 0.1 μg/mL and sterol concentrations ranged from less than 0.01 to 0.12 μg/mL. These results would indicate that "scraped" samples were contaminated by lipids from the epidermis, while "clean" samples gave a more realistic characterization of eccrine lipids.

Miscellaneous Constituents

Lactate and urea have been reported at significant levels in perspiration. The amounts of these compounds can vary from 30 to 40 m*M* at low sweat rates to as low as 10 to 15 m*M* at higher rates.[13] Other miscellaneous components of eccrine sweat include creatine, creatinine,[36] glucose (0.2 to 0.5 mg/dL), pyruvate (0.2 to 1.6 m*M*), cAMP, phenobarbitone, and immunoglobulins.[37] Numerous enzymes have also been detected in dissected sweat glands, including alkaline phosphatase, acid phosphatase, Na/K ATPase, phosphatidic acid phosphatase, monoamine oxidase, acetyl cholinesterase, and lactic, malic, glucose-6-phosphate, isocitric, and succinic dehydrogenases.

Drugs have also been found in eccrine sweat.[38] Sulfonamides, antipyrine, and aminopyrine were found to exhibit sweat concentrations that were directly proportional to plasma levels. Simple diffusion, aided by the relatively low ionization of the drugs studied within the physiological pH range, was assumed to be the mechanism by which these drugs entered the sweat glands. Another study found that L-dimethylamphetamine as well as its metabolite L-methamphetamine were found to be excreted in sweat.[39] After taking 25 mg

Table 3.2 A Summary of the Com

Inorganic (major)	
Sodium	34–266 mEq/L
Potassium	4.9–8.8 mEq/L
Calcium	3.4 mEq/L
Iron	1–70 mg/L
Chloride	0.52–7 mg/mL
Fluoride	0.2–1.18 mg/L
Bromide	0.2–0.5 mg/L
Iodide	5–12 μg/L
Bicarbonate	15–20 m*M*
Phosphate	10–17 mg/L
Sulfate	7–190 mg/L
Ammonia	0.5–8 m*M*
Organic (general)	
Amino acids	0.3–2.59 mg/L
Proteins	15–25 mg/dL
Glucose	0.2–0.5 mg/dL
Lactate	30–40 m*M*
Urea	10–15 m*M*
Pyruvate	0.2–1.6 m*M*
Creatine	
Creatinine	
Glycogen	
Uric acid	
Vitamins	
Miscellaneous	
Enzymes	
Immunoglobulins	

Note: Some compounds and species we concentrations were specified for

of the L-dimethylamphetamine found to be approximately 2 to Unlike the urine concentration, found to be independent of relatively rapid, noninvasive n ethanol (as well as other vola composition of eccrine sweat

Sebaceous Glands

The second major class of se throughout the body, except where noted, the informatio

Table 3.2 A Summary of the Composition of Eccrine Sweat

Inorganic (major)		**Inorganic (trace)**	
Sodium	34–266 mEq/L	Magnesium	
Potassium	4.9–8.8 mEq/L	Zinc	
Calcium	3.4 mEq/L	Copper	
Iron	1–70 mg/L	Cobalt	
Chloride	0.52–7 mg/mL	Lead	
Fluoride	0.2–1.18 mg/L	Manganese	
Bromide	0.2–0.5 mg/L	Molybdenum	
Iodide	5–12 μg/L	Tin	
Bicarbonate	15–20 m*M*	Mercury	
Phosphate	10–17 mg/L		
Sulfate	7–190 mg/L		
Ammonia	0.5–8 m*M*		
Organic (general)		**Organic (lipids)**	
Amino acids	0.3–2.59 mg/L	Fatty acids	0.01–0.1 μg/mL
Proteins	15–25 mg/dL	Sterols	0.01–0.12 μg/mL
Glucose	0.2–0.5 mg/dL		
Lactate	30–40 m*M*		
Urea	10–15 m*M*		
Pyruvate	0.2–1.6 m*M*		
Creatine			
Creatinine			
Glycogen			
Uric acid			
Vitamins			
Miscellaneous			
Enzymes			
Immunoglobulins			

Note: Some compounds and species were only listed as present in sweat in the literature. No concentrations were specified for these components.

of the L-dimethylamphetamine, the maximum concentration in sweat was found to be approximately 2 to 4 μg/mL, within a few hours after ingestion. Unlike the urine concentration, L-dimethylamphetamine levels in sweat were found to be independent of pH. Ethanol has also been detected. Several relatively rapid, noninvasive methods have been proposed to examine the ethanol (as well as other volatile organics) present in perspiration.[40] The composition of eccrine sweat is summarized in Table 3.2.

Sebaceous Glands

The second major class of secretory glands, sebaceous glands, are located throughout the body, except for the palms and dorsum of the feet (except where noted, the information in this section was obtained from Strauss

Table 3.3 Anatomical Variation in the Amount and Composition of Human Sebum Collected After 12 hr of Accumulation (in Weight Percent)

Site	Total lipid (μg/cm²)	CH	CE	TG	DG	FA	WE	SQ	TG+DG+FA
Forehead	288	1.1	2.7	29.6	3.5	27.2	25.9	10.1	60.3
Cheek	144	1.1	3.4	39.4	2.7	15.4	26.9	11.2	57.5
Chest	122	1.3	2.6	29.7	5.4	24.9	25.7	10.3	60.0
Back	84	2.2	2.0	35.9	4.5	17.4	27.4	10.6	57.8
Arm	76	4.8	4.3	34.3	2.4	18.4	27.7	8.1	55.1
Side	57	4.3	4.5	47.1	1.9	7.6	24.9	9.6	56.6
Leg	57	6.3	6.0	44.6	1.5	10.2	23.1	8.1	56.3

Note: CH = cholesterol; CE = cholesterol esters; TG = triglycerides; DG = diglycerides; FA = free fatty acids; WE = wax esters; SQ = squalene; and TG + DG + FA = total glycerides plus free fatty acids.

Source: Greene, R. S., Downing, D. T., Pochi, P. E., and Strauss, J. S., Anatomical variation in the amount and composition of human skin surface lipid. *J. Invest. Dermatol.*, 54(3), 246, 1970. With permission.

et al.[41]). Gland density is greatest around the face and scalp, where as many as 400 to 800 glands per cubic centimeter may be found. The sebaceous glands are generally associated with hair follicles and open inside the hair shaft canals. Unlike eccrine secretions, which empty directly onto the skin surface, the sebum produced by sebaceous glands first travels into the follicular canal and then onto the skin surface. The lipid is produced by a holocrine mechanism, whereby lipid-laden cells disintegrate and empty their contents through the sebaceous duct onto the skin surface.[42] These glands develop during fetal life between weeks 13 and 15 and have achieved a nearly full size by the time of birth.[43] The glands are fully developed and functioning before birth, probably due to stimulation by maternal hormones. At birth, with the termination of the source of these hormones, the glands soon become mostly inactive. Table 3.3 summarizes sebum production and composition for various anatomical regions.[44]

Sebaceous gland activity appears to be controlled by a somewhat complex process. It appears that mid-brain dopamine stimulates the anterior and intermediate lobes of the pituitary gland to release various hormones via certain glands (e.g., thyroid, adrenals, and gonads).[45] In turn, these glands secrete additional hormones that stimulate sebum production. Several androgens have been found to stimulate sebum production.[46] Testosterone is an especially potent stimulator of sebum production in humans. It has been reported that sebum production levels in castrated males are considerably lower than in intact men.[47] The administration of testosterone to castrated males has been reported to result in a significant increase in sebaceous gland activity.[48] However, administration of testosterone to the normal adult male does not lead to an increase in sebum production. This would indicate

Table 3.4 Th and Surface E

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Squalene
Cholesterol este
Cholesterol

Source: Downing, of surface 1974. Wi

that maximum stimulati endogenous testosterone. (face lipids after administer produced a significant inc ination of these compour appears that excretion of their urinary elimination.

Lipid Origin and Bree

Radioactive labeling studi lipids.[52] Autoradiograms incubating samples of sub tate) found in total lipid glycerides, and phospho cholesterol esters, and free of radioactivity. That wou origin rather than being in lipid classes between li in Table 3.4. Another stud two different sources, the *novo* synthesis (endogenou both of these sources rem to sebum was variable. Ex linoleate (an essential fatt erides. However, the fact t have different fatty acid makes it unlikely that the of endogenous lipids that a squalene, and wax esters.

Table 3.4 The Approximate Composition of Sebum and Surface Epidermal Lipids

Constituent	Sebum (wt%)	Surface epidermal lipid (wt%)
Glyceride/free fatty acids	57.5	65
Wax esters	26.0	—
Squalene	12.0	—
Cholesterol esters	3.0	15
Cholesterol	1.5	20

Source: Downing, D. T. and Strauss, J. S., Synthesis and composition of surface lipids of human skin, *J. Invest. Dermatol.*, 62, 231, 1974. With permission.

that maximum stimulation of the sebaceous glands is accomplished by endogenous testosterone. Other studies have found slight increases in skin surface lipids after administering testosterone.[49] Testosterone given to children also produced a significant increase in sebum production.[50] Metabolism and elimination of these compounds in human skin samples has been reported.[51] It appears that excretion of C_{19}- and C_{18}-steroids through the skin may exceed their urinary elimination.

Lipid Origin and Breakdown

Radioactive labeling studies have illuminated the formation and origin of lipids.[52] Autoradiograms from one study showed that radioactivity (from incubating samples of subcutaneous fat from scalp biopsies with [^{14}C] acetate) found in total lipid extracts was confined to squalene, wax esters, triglycerides, and phospholipids. It is significant to note that cholesterol, cholesterol esters, and free fatty acids did not contain any significant amount of radioactivity. That would imply that these compounds are of epidermal origin rather than being produced in the sebaceous gland. The differences in lipid classes between lipids of sebaceous and epidermal origin are listed in Table 3.4. Another study proposed that sebaceous lipids are derived from two different sources, the body's circulation (exogenous lipid) and from *de novo* synthesis (endogenous lipids).[53] They assumed that the composition of both of these sources remained constant, but that their relative contribution to sebum was variable. Examples of possible exogenous lipids would include linoleate (an essential fatty acid), cholesterol, cholesterol esters, and triglycerides. However, the fact that circulating cholesterol esters and triglycerides have different fatty acid compositions than their sebaceous counterparts makes it unlikely that they are incorporated directly into sebum. Examples of endogenous lipids that are not available from blood include Δ6 fatty acids, squalene, and wax esters.

Various oxidative and bacteriological changes occur after sebum is excreted. Lipolysis by enzymes derived from the epidermis or bacteria present in skin surface debris from human skin has a tendency to break down triglycerides and methyl esters.[54] That particular study reported that, in ether, triolein and tristearin were converted primarily to free fatty acids and 1,2-diglycerides and only trace amounts of 1,3-diglycerides and monoglycerides. This evidence leads to the conclusion that the majority of free fatty acids present in sweat originate from the hydrolysis of sebum triglycerides. Evidence of varying degrees of bacterial lipolysis has been offered for *Corynebacterium acnes*,[55,56] staphylococci,[57] *Pityrosporum ovale*,[58] *Pityrosporum acnes*, *Pityrosporum granulosum*,[59] Micrococcaceae, and propionibacteria.[60] Several studies have shown that treatment of skin with antibiotic compounds (e.g., clindamycin) reduced bacterial populations and led to a concurrent decrease in free fatty acids.[61-63] However, one study found that treatment with neomycin failed to affect the *C. acnes* population.[64] It is likely that certain bacteria, such as *C. acnes*, are present within the hair follicles and would be inaccessible to topical antibiotics.

Chemical Composition of Sebum

There is a considerable variety of organic compounds present in sebum. Several factors can influence a particular individual's sebum profile, including diet and genetics. It is possible that each person may have a unique scent signature, as demonstrated by the ability of certain breeds of dogs to track humans over wide areas. In addition, in animals, certain lipids may function as a means of communication. One study determined that in certain species, short-chained aliphatic acids were found to act as pheromones.[65] These compounds also allow animals to recognize members of their own social group. It is possible that a similar situation was once present in humans; however, modern hygiene practices may have diminished our ability to recognize the signals. In fact, in humans, sweat has to be broken down bacterially before it acquires a detectable, characteristic odor. A summary of sebum composition by lipid class is presented in Table 3.5.

Fatty Acids

Hydrolysis of human sebum results in the formation of a mixture of fatty acids. The amount of free fatty acids in sebum shows considerable variation, but averages between 15 to 25%. They are derived primarily from the hydrolysis of triglycerides and wax esters. It has been proposed that as the amount of liberated free fatty acids increases to a certain concentration, the pH drops sufficiently to inhibit bacterial lipases responsible for their production.[64] It has been reported that patients with acne have elevated levels of free fatty acids, typically greater than 30%.[71] It has also been observed that free fatty

Table 3.5 Summary of Sebum Composition

	Downing et al.	Lewis and Hayward	Nicolaides and	Goode and Nordstrom	Darke and

Table 3.5 Summary of Sebum Composition

	Downing et al.[66]	Lewis and Hayward[67]	Haahti[68]	Nicolaides and Foster[69]	Felger[70]	Nordstrom et al.[71]	Goode and Morris[3]	Darke and Wilson[72]
Glycerides	43.2	46.4	42.6	31.7	35.4[b]	16.1	33	30.2[c]
Fatty acids	16.4	16.0	16.2	29.6	27.2[b]	33.0	30	22.0
Wax esters	25.0	21.5	24.2[a]	21.8	22.6	25.3	22	29.3[d]
Cholesterol esters	2.1	2.9	—	3.3	2.5	2.0	2	—
Cholesterol	1.4	1.8	1.4	2.4	0.7	3.8	2	1.1
Squalene	12.0	11.4	15.6	12.8	11.6	19.9	10	17.4

[a] This value is for both wax and cholesterol esters.

[b] The differences between these and other values listed are more than likely caused by individual differences in the degree of lipolysis of triglycerides by bacterial lipases.

[c] This value includes cholesterol esters.

[d] This value includes a minor contribution from diglycerides.

acid content can change with time in the same individual. One study found that certain fatty acids from the same donor taken once a week for 7 weeks showed significant variation in concentration with time.[73] The study also reported significant differences between male and female fatty acid composition. In addition, minor differences were observed between fatty acids isolated from wax esters and cholesterol esters. However, it is difficult to draw conclusions from this data since only two subjects were involved in the study.

Approximately 50% of the fatty acids in sweat are saturated, with straight chain C_{16} and C_{14} being the dominant acids.[74] Monoenes typically constitute 48% of fatty acids, with straight chain C_{16} and C_{18} being the most prominent. The structures of unsaturated fatty acids have been reported to vary with age and sex.[75] The amounts of Δ9-type unsaturated fatty acids (in triglycerides, wax esters, and sterol esters) were always higher in females than in males. The amount of Δ9-type unsaturated fatty acids reaches a maximal value during the prepubertal years, decreases to a minimum from adolescence to middle age, and then begins to increase again with advancing age. In nature, Δ9-type monounsaturated compounds are the most common and Δ6-type are relatively rare. Interestingly, the presence of Δ6-type fatty acids in humans appears to be virtually unique among species studied.[76] Also, Δ6-type unsaturated fatty acids are almost exclusively derived from sebaceous glands, whereas Δ9-type acids appear to be primarily of epidermal origin. Dienoic fatty acids comprise about 2 to 3% of samples, with major isomers being 18:Δ5,8 and 18:Δ9,12.[77] Increased levels of the 18:Δ5,8 diene have been reported in acne patients.[78]

Several branched chain fatty acids have been detected in humans. The largest variation occurred with iso-even fatty acids. One study found significant variations (10- to 20-fold) in the amounts of iso-branched acids having an even number of carbons.[74] Odd-carbon iso- and anteiso-branched acids showed only a threefold variation among individuals tested. Another study examined the possibility that genetics controls the proportions of iso-even fatty acids by analyzing the sebaceous wax esters of twins.[79] While the general population has large variations in the proportions of iso-even fatty acids, intrapair differences in 13 pairs of identical twins were found to be very small. It has been suggested that slight differences in the overall composition of the sebaceous fatty acid mixture could lead to unique, individual odors in humans.[76] Another study found that certain short chain fatty acids, such as iso-valeric acid (iso C_5), are responsible for "offensive" human odors.[80]

Phospholipids

Phospholipids, which are present in the membranes of sebaceous cells, are typically not found in surface sebum. Although epidermal cells have phospholipids, the stratum corneum is virtually devoid of them. This is most

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Wax Esters

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likely due to their degradation in the granular layer, a process that allows for re-absorption of essential nutrients, such as phosphorus and choline. In the epidermis, fatty acids liberated by this degradation process remain in the keratinizing cells and become partly esterified with cholesterol. A similar mechanism has been proposed for fatty acids liberated in the sebaceous glands. However, the lack of cholesterol diminishes the probability of fatty acid esterification. Likewise, a lack of glycerol limits the formation of triglycerides. The study concluded that these fatty acids are most likely reduced to fatty alcohols and then esterified to form wax esters.

Wax Esters

On average, wax esters comprise approximately 20 to 25% of adult skin surface lipids. Wax esters are compounds that contain a fatty acid esterified with a fatty alcohol. Free fatty alcohols have not been found in human skin surface lipids, possibly due to the inability of bacterial or epidermal lipases to hydrolyze wax esters.[80] A study of the fatty alcohol profile derived from wax esters reported a considerable variety of compounds, ranging from C_{18} to C_{27}, with the C_{20} chain being the most abundant.[81] Both iso- and anteiso-branched chain fatty alcohols were also found. In adult wax esters, the most common positional isomer was the Δ6-type, comprising 98.28% of detected mono-unsaturated acids.[82] Since wax esters are known to be of sebaceous origin, this evidence would indicate that fatty acids with the Δ6 double bond position are also of sebaceous gland-origin, whereas those with Δ9-type are of epidermal origin. It was also reported that 26.7% of adult wax ester fatty acids were of a branched chain type. It is rare to find a wax ester that contains two fully saturated straight chain fatty acid components. One possible reason would be that the presence of unsaturation or branching makes it more likely that the resulting wax ester would be liquid at skin temperature.

Sterols

Sterol esters comprise approximately 2 to 3% of adult skin surface lipids. It has been proposed that sterol esters are not synthesized directly but rather are secondary products.[82] Two strains of bacteria, staphylococci and propionibacteria (minimally), have been found to esterify cholesterol.[83] There is also evidence that a major proportion of cholesterol esterification occurs on the skin surface.[84] Two to three times more sterols were found esterified on the skin surface than in the epidermis or in isolated stratum corneum. Free sterols are built up in the living portion of the epidermis and are then esterified primarily with sebum fatty acids, but also with some acids released from the epidermis during the late stages of keratinization. The fact that a high percentage of sterol esters (88.92%) have fatty acids with Δ6-type unsaturation lends support to the hypothesis that the fatty acids comprising them

are of sebaceous origin.[82] Approximately 20% of the fatty acids in sterol esters were reported to be of a branched chain type. Also, the levels of sterols and sterol esters have been reported to be higher in women than men. Cholesterol, cholest-5-en-3β-ol, is approximately 1 to 2% of adult surface lipids. Cholesterol, which is the most abundant steroid in animal tissues, is not believed to be synthesized in the sebaceous glands. It may be incorporated into sebum from the body's circulation (e.g., blood, plasma, etc.).

Squalene

Squalene comprises approximately 11 to 12% of adult lipids. Squalene, 2,6,10,15,19,23-hexamethyl-2,6,10,14,18,22-tetracosahexaene, cyclizes readily to form steroids in the body, including the steroid alcohols lanosterol and cholesterol. Squalene levels have been reported to be elevated in acne patients.[85,86] Patients with acne were reported to have a mean squalene content of 19.9%.[71] Squalene production in sebaceous glands has been found to vary depending on the gland size, with larger glands producing greater amounts of the lipid.

Miscellaneous Organic Compounds

A recent study of sweat collected from glass beads using cryofocusing GC/MS revealed a considerable number of trace organic compounds.[5] Most of the ketones detected were between butanone and decanone. However, trace amounts of the following were also found: 2-nonen-4-one, 2-decanone, 2-methoxy-2-octen-4-one, 6,10-dimethyl-5,9-undecadien-2-one, and possibly 3-hydroxyandrostan-11,17-dione. Numerous aldehydes were also detected, with the most prevalent being in the series between propanal and nonanal. Alkanes and alkenes below decane were not detected because of high volatility and amounts below instrument detection limits. Few amides were reported. However, a series of tertiary amines was detected ranging from *N,N*-dimethyl-1-dodecanamine to *N,N*-dimethyl-1-octadecanamine. Several heterocyclic compounds were detected, including substituted pyrroles, pyridines, piperidines, pyrazines, and furans. Nicotine was also detected in some samples. A number of haloalkanes were reported, including an incomplete series from chlorohexane to chlorohexadecane. Carbon disulfide and dimethyl sulfide and a few mercaptans, including thiomethane and 2-thiopropane, were also present. The chemical composition of secretions from the sebaceous gland is summarized in Table 3.6.

Apocrine Glands

The apocrine glands are another class of secretory glands. These glands are large coiled structures that are located close to hair follicles and their associated sebaceous glands (except where noted, the information in this section was

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Variation of Sebum

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lipid composition by age

Table 3.6 A Summary of the Composition of Sebaceous Secretions

Organic (major)		Organic (trace)
Triglycerides	30–40%	Aldehydes
Free fatty acids	15–25%	Ketones
saturated	50%	Amines
monounsaturated	48%	Amides
polyunsaturated	2%	Alkanes
Wax esters	20–25%	Alkenes
Squalene	10–12%	Alcohols
Cholesterol	1–3%	Phospholipids
Cholesterol esters	2–3%	Pyrroles
		Pyridines
		Piperidines
		Pyrazines
		Furans
		Haloalkanes
		Mercaptans
		Sulfides

obtained from Robertshaw[87]). They are localized primarily in the axillary and perineal areas. The excretory portion of these glands takes the form of a huge intertwined coil that can extend well into the sub-dermal fatty layer.[2] The duct leaving the coil takes a more or less vertical path parallel to an adjacent hair follicle into which it opens at a point above the hair's sebaceous gland.

Few studies have been made to analyze the secretions emanating from the apocrine glands. Detailed analysis of apocrine secretions is complicated by contamination from eccrine and sebaceous glands. One of the few studies done on human apocrine secretions found a substance that was milky in appearance and dried to a plastic-like solid.[88] This material fluoresced and had a variable odor. One source reported several substances isolated from apocrine secretions, including proteins, carbohydrates, cholesterol, and iron.[89] C_{19}-steroid sulfates and Δ16-steroids (e.g., 5α-androst-16-en-3α-ol and 5α-androst-16-en-3-one) have also been reported.[90,91]

Variation of Sebum Composition with Age of Donor

It has been well established that the chemical content of sweat changes from birth to puberty and up through old age. Rates of sebum excretion, amounts of certain fatty acids, the ratio of wax esters to cholesterol, and cholesterol esters have been found to change.[92] Some components do not show any significant difference with age. Table 3.7 compares the variation in surface lipid composition by age group.[93]

Table 3.7 Changes in Surface Lipid Composition with Age

Age	Free Fatty Acids	Triglycerides	Wax Esters	Cholesterol	Cholesterol Esters	Squalene
5 days	1.5	51.9	26.7	2.5	6.1	9.9
1 month–2 years	20.8	38.4	17.6	3.7	10.3	9.4
2–4 years	22.9	49.6	8.0	4.2	8.9	6.2
4–8 years	15.9	45.6	6.9	7.2	14.6	7.7
8–10 years	17.8	47.4	17.8	3.2	5.7	8.3
10–15 years	18.8	42.9	23.6	1.8	4.2	8.4
18–45 years	16.4	41.0	25.0	1.4	2.1	12.0

Source: Ramasastry, P., Downing, D. T., Pochi, P. E., and Strauss. J. S., Chemical composition of human skin surface lipids from birth to puberty, *J. Invest. Dermatol.*, 54(2), 143, 1970. With permission.

Newborns

One study[94] of neonatal skin surface lipids (from vernix caseosa, a grayish-white substance that covers the skin of the fetus and newborn) reported the following lipid classes and amounts: sterol esters, 35%; triglycerides, 26%; wax esters, 12%; squalene, 9%; free sterols, 9%; diesters, 7%; and miscellaneous lipids, 4%. These values show more similarity to adult sebum profiles than to young children. The composition of wax ester fatty acids (with regard to the chain type and the amount of saturated and unsaturated) in vernix caseosa has been found to be quite similar to that of adults.[82] However, the amount of sterol esters showed considerable difference. In adults, sterol esters constituted approximately 2.81% of the skin surface lipids, whereas vernix caseosa contained 25.4%. The large percentage of high molecular weight sterol esters in vernix caseosa probably helps to provide a waxy coating of low water solubility that prevents excessive wetting of fetal skin. Fatty acids in vernix caseosa were predominantly saturated (65%) while adult samples were primarily mono-unsaturated (54%). A study of vernix caseosa by Miettienen and Lukkäinen found at least eight additional sterols besides cholesterol, including lanosterol.[95]

The vernix caseosa of male fetuses contained much more sebum than those of female fetuses, which had a higher proportion of epidermal lipids.[75] The differences were significant enough to be able to distinguish the sex of the fetus based on the thin layer chromatogram of lipids extracted from the vernix caseosa. Although androgen levels are high in newborns, the level of hormones (as well as sebum production) drops rapidly during subsequent months.[96] This leads to a dramatic change in the amount and composition of excreted lipids.

Young Children

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Adolescents

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Age

	Cholesterol Esters	Squalene
	6.1	9.9
	10.3	9.4
	8.9	6.2
	14.6	7.7
	5.7	8.3
	4.2	8.4
	2.1	12.0

Chemical composition of human
2): 143. 1970. With permission.

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Young Children

The sebum composition of children aged 2 to 8 years old is dominated by epidermal lipids (e.g., cholesterol and its esters).[93] Typically, the amounts of wax esters and squalene in young children were measured to be approximately one third and one half of adult levels, respectively. By the ages of 8 to 10 years, the levels rose to about two thirds of adult levels. Adult levels were reached between the ages of 10 to 15 years. Median wax ester secretion rates were found to be between 10 to 50 μg/10 cm^2 per 3-hr collection period.[97] Levels of cholesterol and cholesterol ester secretion were found to vary little between children, with the average amount secreted being 11.0 ± 4.5 and 16.6 ± 8.7 μg/10 cm^2 per 3-hr period. Another study measured the total sebum production rate in children.[43] The rates varied in females from 0.60 mg lipid (per 10 cm^2 area per 3-hr period) for 7 year olds to 1.29 mg for 14 year olds. For males the values varied between 0.58 mg for 9 year olds to 2.17 mg for 16 year olds. However, in late childhood, as sebum secretion begins to increase, the amount of wax esters (which are of sebaceous origin) increased relative to cholesterol and cholesterol esters (which are of epidermal origin).

There are significant changes in the relative concentrations of the major fatty acids constituting the triglyceride and wax ester fraction of children's sebum.[98] The levels of C_{15} fatty acids increased from 3% in the pre-pubertal group to 9% in the pubertal group. The levels of $C_{16:1}$ fatty acids increased to between 20 to 40% while C_{18}, $C_{18:1}$, and $C_{18:2}$ sharply declined. Also, the ratio of wax esters to cholesterol and cholesterol esters begins to increase between the ages of 7 and 8 and reaches a more "adult" profile by age 10 or 11.[99] Sebum secretion rates continue to increase during adolescence until about age 17 or 18, when a relatively stable phase is achieved.[100] Once maturity has been achieved, it appears that little changes occur in sebaceous gland activity until middle age.

Adolescents

At the onset of puberty, hormone-mediated sebaceous gland enlargement occurs and sebum production increases significantly. It has been suggested that during puberty the proportion of endogenously synthesized sebaceous lipids (characterized by squalene, wax esters, and Δ6 fatty acids) increases while the proportion of exogenous-type (characterized by cholesterol, Δ9 fatty acids, and linoleic acid) decreases. This may be explained by the fact that sebaceous cells have a relatively constant amount of exogenous-type lipid, but they synthesize variable amounts of the endogenously synthesized sebaceous-type lipids. The amount synthesized is directly related to the

gland's activity. The more active the gland, the more dilute the exogenous lipids become due to excess production of sebaceous-type lipids.

In one study, the levels of sebum production (measured as milligrams of lipid collected on a 10-cm^2 patch of skin over 3 hr) of subjects of varying age were measured.[43] The values obtained for adolescent males and females were 2.35 mg and 2.17 mg (per 10-cm^2 area per 3-hr period), respectively. The largest jump in sebum production occurred between the ages of 12 and 13 in both males and females. The mean sebum levels differed significantly in certain age cohorts for subjects with and without acne. In addition, patients suffering from acne vulgaris were found to possess a greater amount of lipolytic agents than those patients without acne, which might explain the reported elevated fatty acid levels.[101] The difference between subject males aged 15 to 19 with and without acne was 2.80 mg and 1.73 mg. For females 15 to 19, the values were 2.64 mg and 1.85 mg. In the 20 to 29 age cohort, the values for males were 2.87 mg and 2.37 mg; for females the values were 2.58 mg and 1.77 mg.

Post-Adolescence

Sebum production continues with age, peaking during the mid thirties and then begins to decline in middle age. In old age, levels of sebum may drop to near pre-puberty levels. One study of sebaceous wax esters found that secretions decreased about 23% per decade in men and about 32% in women.[102] This is in contrast to some findings that show the rates remain somewhat stable.[43,103] Overall, it appears that no significant changes occur in sebum composition until much later in life. A study of sebum secretion rates of four adult males by gravimetry found a range of rates from 2.15 to 4.47 mg/10 cm^2 per 3-hr period.[104] In another study, the sebum production rates for males and females were reported for 20 to 29 year olds, 2.48 mg and 2.03 mg; 30 to 39 year olds, 2.52 mg and 2.04 mg; 40 to 49 year olds, 2.39 mg and 1.86 mg; and 50 to 69 year olds, 2.42 mg and 1.10 mg.[43]

The principal cause for the decrease in sebum gland activity with age is diminished hormonal stimulation.[100] Testosterone levels in men begin to decrease significantly between the age of 50 to 60 years. Sebaceous gland activity typically does not decrease until a decade or so later. In women, the decrease is observed a decade or so sooner than in men. The sebum production rates for males and females have been reported to be 1.69 mg and 0.85 mg, respectively, for subjects over age 70.[43] Interestingly, some studies have shown that, although sebaceous gland activity decreases with age, the glands themselves become larger rather than smaller.[105] It also appears that with advancing age, the proportion of Δ9-type unsaturated fatty acids increases.[75]

The Composition of

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The Composition of Latent Print Residue

Although considerable research has been conducted on sweat samples, comparatively little data are available on the content of latent print residues. The components of sweat transferred to different surfaces may differ from that found on the surface of skin. The law enforcement community began to examine latent print residues critically and scientifically in the late 1960s. The foundation work in this area was sponsored by the United Kingdom Home Office and conducted by the Atomic Weapons Research Establishment (AWRE) and the Atomic Energy Research Establishment (AERE). These research efforts concentrated on analyzing the chemical components of latent print deposits.

United Kingdom Home Office

The United Kingdom Home Office sponsored a considerable number of research projects over the past 35 years. These projects were carried out in cooperation with the Central Research Establishment (CRE) and the Police Scientific Development Branch (PSDB), which was formerly known as the Scientific Research and Development Branch (SRDB). These efforts represented, in many cases, the first attempt to perform a detailed study and analysis of visualization methods as well as the composition of print residue.

During the mid to late 1960s, a series of projects were done to investigate the organic and inorganic substances present in a latent print. One study examined the water soluble components and reported the following substances (and approximate amounts): chloride, 1 to 15 μg; calcium, 0.03 to 0.3 μg; sulfur, 0.02 to 0.2 μg; urea, 0.4 to 1.8 μg; lactic acid, 9 to 10 μg; amino acids, 1 μg; phenol, 0.06 to 0.25 μg; sodium, 0.2 to 6.9 μg; potassium, 0.2 to 5.0 μg; and ammonia, 0.2 to 0.3 μg.[106] A subsequent study examined the change in chloride content in fingerprints as a factor of the donor's age.[107] Results indicated that the chloride content decreased with advancing age. In addition, the donor's occupation also appeared to be a factor. Office workers were found to have the highest amounts of chloride followed by laboratory workers and persons employed in workshops. Differences were also found between left and right hands as well for individual fingers. Statistically, digits on left hands were found to have a higher chloride content, presumably because most of the donors were right handed. Thumbs had the lowest amount of chloride while little fingers had the highest. However, because of a significant variation present within the same individual, these trends were not always observed.

The Home Office also conducted detailed studies of the lipid (or water-insoluble) portion of latent prints, with an emphasis on the free fatty acid

content.[108] Palmitic acid was found to be the most abundant fatty acid. In general, the most abundant acids were $C_{18}/C_{18:1}$ + squalene followed by $C_{16}/C_{16:1}$, $C_{14}/C_{14:1}$, C_{15}, and $C_{12}/C_{12:1}$. Another study confirmed that palmitic, stearic, and palmitoleic acids were the most abundant fatty acids.[72] This study also addressed the contribution of cosmetics present in samples from female volunteers. They found that the presence of cosmetics might introduce peaks in the early portion of the chromatogram (e.g., decanoic acid). The mean values obtained for the amounts of the various lipid classes found in forehead samples are reported in Table 3.5.[72] Those values can be compared with the following average values obtained from fingers: squalene, 14.6%; cholesterol, 3.8%; free fatty acids, 37.6%; wax esters (with diglycerides), 25%; and triglycerides (with monoglycerides and cholesterol esters), 21%. Although some differences are to be found in the free fatty acid and glyceride values, these discrepancies can be attributed to individual variations in bacterial lipase activity. Additional studies, using gas-liquid chromatography, detected over 40 different organic constituents in sebaceous secretions. The results, expressed as general lipid classes, are reported in Table 3.5.[3] The report stressed that the sebaceous secretions are very important with regard to fingerprint visualization because they are more stable to water than the principal components of eccrine sweat.

Oak Ridge National Laboratory

A 1993 child abduction case in Tennessee inspired a local police criminalist and a chemist from the Oak Ridge National Laboratory (ORNL) to team up and analyze fingerprint residues.[109-111] Knoxville Police criminalist Art Bohanan observed that children's fingerprints left on nonporous surfaces (such as a vinyl car seat) did not seem to last for more than a day or two. Subsequent analyses performed by Buchanan et al. at ORNL indicated a significant difference in the chemical composition of children's and adults' print residues.[112,113] Children's prints contained more volatile components that would not remain in the deposit for more than a couple of days (depending on the environmental conditions). In both children and adults, fatty acids (as methyl esters) in the C_{12} to C_{24} range were detected. Although cholesterol was found in prints from children and adults, the amount was significantly higher in children. There were differences detected between samples from male and female children, although these compounds were not identified. The most abundant compound detected in the isopropyl alcohol extracted material of adults was squalene. In addition, several long chain fatty acid esters were identified, including pentadecanoic acid dodecyl ester, and the undecyl, tridecyl, pentadecyl, heptadecyl, and octadecyl esters of hexadecanoic acid.

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Subsequent studies conducted at the ORNL yielded some unusual results.[114] Nicotine was detected in some of the adult samples. Although initially dismissed as environmental contamination caused by handling tobacco products or exposure to second-hand tobacco smoke, a subsequent analysis of a sample obtained from an individual who had quit smoking several weeks before (but had been chewing nicotine gum) showed traces of nicotine. While unexpected, this result is not unprecedented. Robinson et al. reported the presence of nicotine, as well as morphine and alcohol, in sweat in 1954.[115] Traces of steroids were also observed in some of the fingerprint samples. ORNL plans to direct future efforts toward the ability to detect trace amounts of special target compounds (e.g., illegal drugs and their metabolites) in latent print residues to provide investigative leads for law enforcement purposes. If successful, such noninvasive methods could potentially eliminate the need to obtain biologically hazardous samples such as blood or urine.

Pacific Northwest National Laboratory

With funding obtained from the Technical Support Working Group (TSWG is an interagency working group that funds counter terrorism projects), the U.S. Secret Service (USSS) teamed up with the Pacific Northwest National Laboratory (PNNL) to conduct a research project to investigate the composition of latent print residue. The most critical aspect of this project was to investigate how latent print residue changes over a period of time. Fingerprint samples from 79 volunteers, ranging in age from 3 to 60 years old, were analyzed. Volunteers placed fingerprints on filter paper samples. The samples were then stored at ambient conditions before being extracted. After derivatization, the samples were analyzed by gas chromatography/mass spectrometry.

The results of this study were in agreement with the data obtained at the ORNL.[116] Several samples analyzed appeared to be contaminated by external sources of lipids, such as hand lotions, cosmetics, and soaps. Removing all traces of these contaminants proved difficult. The data obtained from the aging of fingerprint residues were also reported. As expected, most of the unsaturated lipids (e.g., squalene and fatty acids such as oleic and palmitoleic) tended to diminish substantially within the 30-day period, with significant losses during the first week noted. Since lipids like squalene and oleic acid are liquid at room temperature, they provide an environment suitable for partitioning of certain lipid-specific visualization reagents. Once they have been modified and the majority of the water content of the print has evaporated, the print dries out and is no longer amenable to lipid partitioning reagents. For example, reagents like Nile red, which partition into the lipid layer, are generally ineffective on prints more than a few days old.

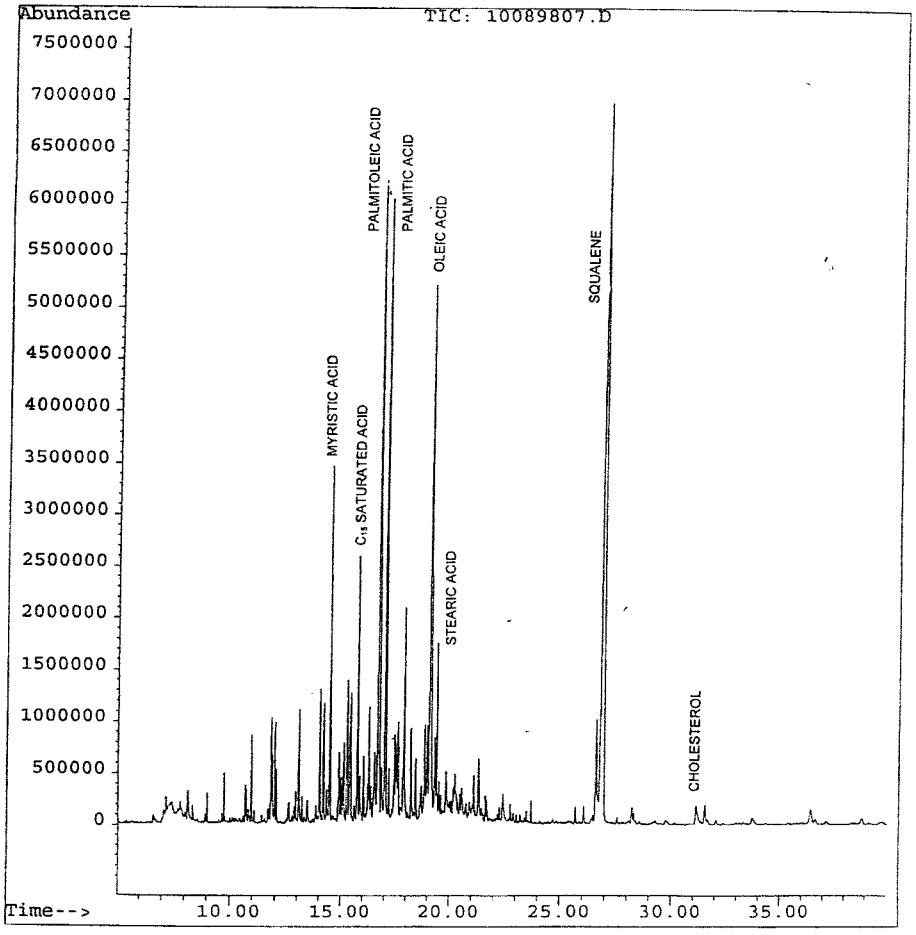

Figure 3.4a A chromatogram of a fingerprint deposit extracted and analyzed shortly after deposition.

In contrast, saturated compounds (e.g., palmitic and stearic acids) remained relatively unchanged during the same time period. Wax esters also remained relatively stable. Overall, as the sample fingerprint aged, compounds in the low molecular weight range began to form. These compounds would be consistent with lighter molecular weight saturated acids (e.g., nonanoic acid) and diacids (e.g., nonandioic acid). Figures 3.4a and 3.4b are chromatograms of samples taken from the same donor and analyzed initially and 60 days later. Overall, the results of the study indicate that saturated compounds dominate aged samples. Unfortunately, these compounds do not make good targets for chemical reagents.

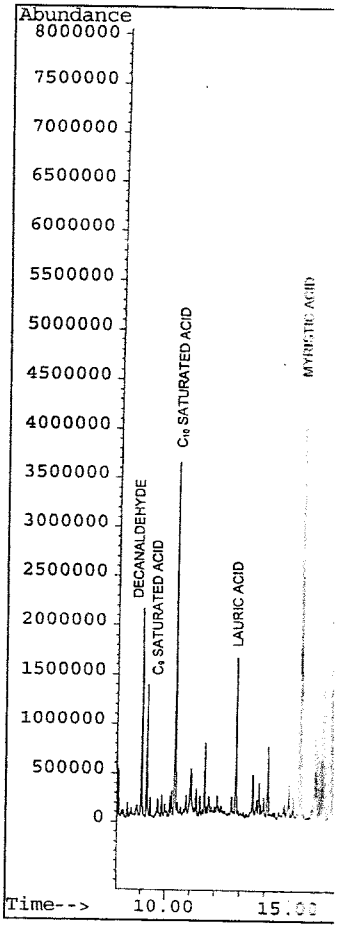

Figure 3.4b A chromatogram lyzed 60 days after deposition

Savannah River Technic

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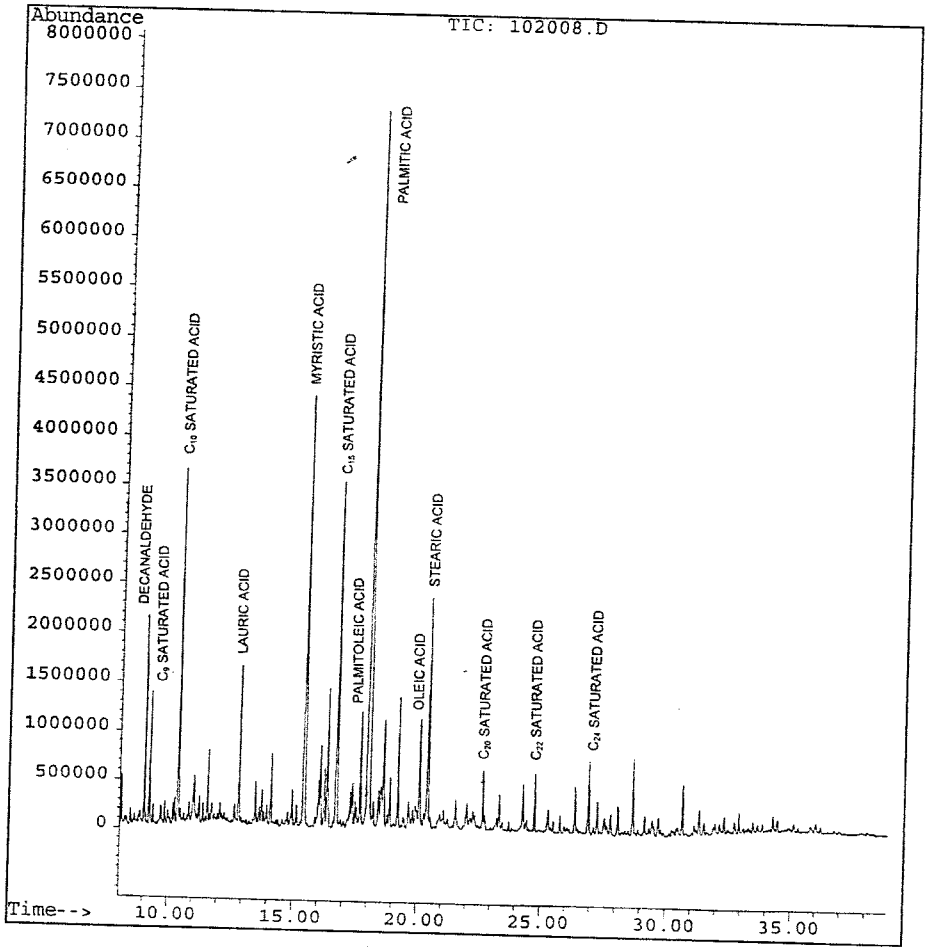

Figure 3.4b A chromatogram of the same fingerprint deposit extracted and analyzed 60 days after deposition.

Savannah River Technical Center Research

Another project was recently begun at the Savannah River Technical Center (SRTC) in cooperation with the USSS to analyze latent print residue and how it changes with time. With funding from both the TSWG and Department of Energy, the SRTC is looking into characterizing the degradation products formed as the latent print residue ages to determine if any of these compounds may be suitable for chemical visualization reagents. The SRTC is focusing on the formation of hydroperoxides, one class of breakdown products formed as lipids oxidize. A series of standard lipids representative of the

various lipid classes found in a latent print was used. These included compounds typically found in print residue, including cholesterol, triglycerides, fatty acids, wax esters, cholesterol esters, and catalyze the reaction between triplet a sensitizer (protoporphyrin IX dimethyl ester, 0.01% of the overall mixture). The sensitizer was added to oxygen and light to form singlet oxygen (a highly reactive species). These compounds were placed on a glass slide and aged in various conditions (e.g., light/no light and/or indoors/outdoors). Like PNNL, the SRTC found that unsaturated compounds are rapidly depleted from samples even in cool, dark storage conditions. An experiment involving the aging of squalene on a glass slide found that after one month of exposure to ambient conditions, 10% of the sample was composed of hydroperoxides. The SRTC is looking into chemiluminescent methods for visualizing the hydroperoxides formed as fingerprints age.

Forensic Science Service

Recent work done at the Home Office Forensic Science Service (FSS), Metropolitan Laboratory, London, England, involved the use of thin layer chromatography (TLC) to directly separate sebum-rich fingerprints from five donors left on TLC plates.[117] The FSS has recently updated this work.[118,119] Although the use of TLC to analyze latent print residues is not new,[120,121] the direct separation and characterization of a deposited print was unique. The ultimate goal of these experiments was to react the separated classes of latent print residue with different chemical reagents. Additional studies are being planned in cooperation with the Police Science and Criminology Institute, University of Lausanne, Switzerland. In addition, the FSS has been working on trying to identify the compound(s) responsible for inherent luminescence observed in some latent prints. Efforts using TLC, GC/MS, and Raman spectroscopy have not provided a definitive answer, but one leading candidate is bilirubin. The FSS suggested that bacteria, present on the skin, might be involved. Bacteria are known to produce porphyrins (intermediates in the synthesis of heme), which fluoresce in the visible region. The most likely candidate for inherent luminescence, bilirubin, is the breakdown product of heme.

Currently, a collaborative effort, funded by the TSWG, is underway between the USSS and FSS to investigate the effect of light conditions on the aging of print residues. The project will analyze samples from five male donors, aged 24 to 34, at a sampling interval of 0 (shortly after deposition), 3, 7, 9, 10, 15, and 20 days. The samples will also be cut in half and then subjected to different lighting conditions while at constant temperature and humidity. Although the study is not complete, some of the initial results are consistent with data generated by PNNL. There appear to be significant differences in decomposition rates for samples in the different lighting conditions. It

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would be of interest, if future funding is available, to evaluate the impact of other environmental conditions on latent print decomposition products and rate.

DNA From Latent Prints

Another important component of latent print residue is deoxyribonucleic acid (DNA). It is not surprising that a significant amount of DNA is often present in visible blood prints. However, it can also be deposited in non-blood latent print residue from the epidermal cells that are continuously sloughed off the skin surface through rubbing of the skin or through direct contact with a substrate. In the past, an examiner was often forced to decide what evidence is more important, the DNA or the ridge detail. Advances in DNA technology have made this decision easier since fewer latent print visualization processes inhibit sample analyses. The use of polymerase chain reaction (PCR) analysis has allowed subnanogram quantities of DNA to be detected, amplified, and analyzed. In addition, "lab on a chip" technology will soon allow for extremely fast analysis and identification at the crime scene.[122-125] Examiners are now also able to extract DNA in situations previously considered improbable. Sweet et al. reported that identifiable DNA was obtained from a bite mark on skin from a victim who had been drowned.[126] Such advances will begin to highlight the need to rapidly and reliably extract, analyze, and identify DNA recovered from latent prints.

DNA From Blood Prints and Stains

The recovery of DNA from visible blood prints and latent blood prints developed by chemical reagents has been well documented. Most studies found that only a few visualization reagents inhibit DNA analysis. A study of envelopes, stamps, and cigarette butts by Presley et al. using Chelex extraction and PCR HLA DQ alpha typing found negative DNA results after processing with PD.[127] A subsequent study by Walls also found that physical developer adversely affected DNA analysis.[128] However, it was reported that the problem with PD could be overcome by using organic extraction rather than Chelex. Stein et al. studied the effect of black powder, ninhydrin, cyanoacrylate fuming, and gentian violet on 1-, 14-, and 56-day-old bloodstains and saliva samples. They found that none of the latent print visualization treatments adversely affected DNA extraction, quality, or typing using restriction fragment length polymorphism (RFLP) or PCR-short tandem repeat (STR).[129] Another study examined the effects of cyanoacrylate (CA) fuming and forensic light sources on bloodstains with subsequent analysis

of DNA using RFLP.[130] No adverse effects were reported. Newall et al. investigated the effect of CA fuming on blood prints and also found no inhibition.[131] Another light source study was conducted by Andersen and Bramble.[132] They found that exposure of DNA to 255-nm shortwave UV radiation (1 mW/cm^2 at a distance of 25 to 35 cm) for as little as 30 sec could drastically reduce the chances of recovering and identifying DNA using PCR-STR analysis.

A study of the effect of seven different blood reagents (amido black, DFO, ninhydrin, Hungarian Red, Crowle's Double Stain, luminol, and Leucomalachite Green) on DNA recovered from diluted blood prints on several porous and nonporous substrates and analyzed using the PCR-STR/Profiler Plus multiplex system found no adverse results.[133] Miller reported success with these reagents with a blood dilution factor of up to 1:10,000.[134] The report also mentioned that as the length of exposure to the reagents and the extent of dilution of the blood sample increased (beyond 1:10,000), the possibility of recovering DNA diminished significantly. A similar result for luminol was reported by Gross et al.[135] Champod reported on PCR-STR analysis work done by Brignoli and Coquoz that found difficulties with LMG and *o*-tolidine, but not with MMD.[136] Hochmeister et al. reported a similar result for LMG and *o*-tolidine using RFLP analysis.[137] Roux et al. also looked at the effect of visualization reagents on blood prints.[138] MMD, magnetic fingerprint powder, and UV radiation were found to interfere with PCR DNA analysis. The study also found that DFO, Sticky-side powder, ninhydrin with secondary metal salt treatment, amido black, diaminobenzidine, luminol, CA with rhodamine 6G, and black powder could adversely affect recovery and analysis of DNA using the D1S80 system primers. Most of these problems were resolved by using CTT system primers. Their study also indicated problems with the blood reagent benzidine dissolved in glacial acetic acid.

DNA From Developed Latent Prints

Very few studies have been published that examine the possibility of recovering DNA from treated latent prints (rather than treated bloodstains or blood prints). Recently, Zamir et al. investigated the effect of DFO treatment of latent prints on DNA analysis and found that it had no adverse effect.[139] Another related issue involves the possibility of recovering DNA from undeveloped fingerprints left on commonly handled objects. This issue was highlighted by van Oorschot and Jones in the journal *Nature* in 1997.[140] The quantity of DNA recovered from objects like a car key, briefcase handle, and a telephone handset was found to be sufficient to identify the person who had handled the item. In some cases DNA transferred from another source (a secondary transfer) was detected and identified. However, a similar study

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done by the Royal Canadian Mounted Police (RCMP) found that such secondary transfers can occur but are rare.[141] Another study by Ladd et al. found that primary transfer was not always detected and that no secondary transfer occurred with their samples.[142]

A subsequent letter in *Nature* reported success in using PCR-STR to obtain profiles from single cells using six forensic STR markers.[143] DNA was successfully amplified in 91% of the cells tested and a full DNA profile was obtained in 50% of those cases. This sort of success ultimately leads to the question of whether DNA could be recovered from a smeared or partial, developed print that was not of identification value. Two issues are critical. Do latent prints contain a sufficient number of cells and what effect do all of the latent print visualization techniques have on DNA analysis? Two projects funded by the TSWG in cooperation with the USSS are currently underway to begin exploring both concerns. Dr. Mark Batzer, from The Louisiana State University Medical Center (LSUMC), New Orleans, LA, is working on quantifying the amount of cellular material present in a latent print as well as using nuclear DNA methods to analyze and identify it. Dr. Robert Bever, of the Bode Technology Group, Springfield, VA, is working on optimizing mitochondrial DNA (mtDNA) techniques for partial latent prints. Since there are inherently several orders of magnitude more copies of mtDNA present in a cell, the likelihood of finding it is better in very small or degraded samples.

DNA is also capable of yielding more than just a strict identification or elimination of a suspect. The sex and geographic origin of the individual can now be determined from DNA.[144-146] DNA markers that can yield information about hair color, height, and other morphological characteristics are also being explored. This was evident at the recent Millennium Conference on Forensic Human Identification sponsored by the Forensic Science Service, which was held in London in October 1999.[147-150] Interestingly, this technology is likely to be involved in settling a controversy surrounding Beethoven's origin.[151]

Miscellaneous Compounds and Contaminants

Many environmental contaminants have been detected both in analyses of sweat and fingerprint residues. Caution must be exercised in determining whether such compounds might indeed be contaminants or as compounds derived from an endogenous source. There may be some overlap between compounds present in the contaminant and ones from an endogenous source, which could lead to overestimates of the quantity of such compounds. Bernier et al. reported a significant amount of glycerol in one sample.[5] This

was later found to be caused by the use of hair gel by one of the volunteers. Benzene, toluene, styrene, and alkyl substituted benzenes were also detected but considered as exogenous contaminants. A number of siloxanes, believed to be related to the column stationary phase, and phthalates were also detected. Hexamethylcyclotrisiloxane and octamethylcyclotetrasiloxane were the two primary siloxane compounds. In addition, 1,1-difluoroethane was one of the most intense peaks detected. This compound is a component of Dust-Off, a product used to cool the glass injection port liner between runs.

The study by PNNL also detected several exogenous contaminants, including acetaminophen and n-butylphenylsulfonamide, a detergent found in gasoline. A number of hydrocarbons and glycerol esters were detected and attributed to contamination by cosmetics or other personal hygiene products. Typical examples of contaminant hydrocarbons include a series from tricosane to nonacosane, eitriacontane, and dotriacontane. Examples of esters include the 3,4-methoxyphenyl-2-ethylhexyl ester of propenoic acid and glyceryl trioctyl ester.

Conclusions

Latent print residue is a complex mixture of many different types of substances. Derived primarily from the three major secretory glands, sweat is deposited on virtually every surface touched by hands. Future efforts must continue to focus on determining how latent print residue adheres to, interacts with, and changes with time on different surfaces. This information is critical to understanding not only how reagents used to visualize latent prints work, but also to provide better guidance in modifying existing reagents and developing new ones.

Interestingly, there have been efforts in this past decade by several laboratories to produce "artificial sweat." Both the German Bundeskriminalamt (BKA) and the FSS have worked on creating a way of reproducibly creating a standard latent print. The applications for such a "standard latent print" are numerous. With the advent of laboratory accreditation guidelines established by organizations such as the American Society of Crime Laboratory Directors (ASCLD-LAB) and the International Organization for Standardization (ISO), the use of a "standard latent print" becomes critical in evaluating the effectiveness of visualization reagents that are routinely used in the evidence processing laboratory, as well as in the area of comparative testing and evaluation of new reagents worldwide. In the near future, the TSWG will be providing funding to build upon the groundwork established by the BKA and FSS. This project will also take advantage of the knowledge gained by the recent research efforts that have examined the chemical composition of recent and aged latent print residues.

References

1. Odland, G. F., *Biochemistry*, University Pre
2. Vincent, P. G. 11(41), 8-12,
3. Goode, G. C. composition a
4. Bernier, U. R., nations by ga of attractants glass beads. *A*
5. Bernier, U. R., Analysis of hu etry. 2. Identifi the yellow feve
6. Quinton, P. M.
7. Sato, K. and D of the human
8. Berglund, L. G effects of dis 127-138, 197
9. Jensen, O., N Physical and *(Stockholm)*, 5
10. Cage, G. W., water intake o *J. Appl. Physio*
11. Brusilow, S. *Physiol.*, 214,
12. Mitchell, H. H environmenta particular refe
13. Sato, K., The sweat gland. *R*
14. Bayford, E., S
15. Olsen, R. D., 53(10), 4, 197
16. Sato, K., Feibl pharmacologi *Invest. Derma*

References

1. Odland, G. F., Structure of the skin. In Goldsmith, L. A., Ed. *Physiology, Biochemistry, and Molecular Biology of the Skin. 2nd ed.* New York: Oxford University Press, 1991.
2. Vincent, P. G., Skin. A brief look under the surface. *Fingerprint Whorld,* 11(41), 8-12, 1985.
3. Goode, G. C. and Morris, J. R., Latent fingerprints: a review of their origin, composition and methods for detection. *AWRE Report No. 022/83,* 1983.
4. Bernier, U. R., Booth, M. M., and Yost, R. A., Analysis of human skin emanations by gas chromatography/mass spectrometry. 1. Thermal desorption of attractants for the yellow fever mosquito (*Aedes aegypti*) from handled glass beads. *Anal. Chem.*, 71, 1-7, 1999.
5. Bernier, U. R., Kline, D. L., Barnard, D. R., Schreck, C. E., and Yost, R. A., Analysis of human skin emanations by gas chromatography/mass spectrometry. 2. Identification of volatile compounds that are candidate attractants for the yellow fever mosquito (*Aedes aegypti*). *Anal. Chem.*, 72(4), 747-756, 2000.
6. Quinton, P. M., Sweating and its disorders. *Annu. Rev. Med.*, 34, 429-452, 1983.
7. Sato, K. and Dobson, R. L., Regional and individual variations in the function of the human eccrine sweat gland. *J. Invest. Dermatol.*, 54, 443-449, 1970.
8. Berglund, L. G., Gallagher, R. R., and McNall, P. E., Simulation of the thermal effects of dissolved materials in human sweat. *Comput. Biomed. Res.,* 6, 127-138, 1973.
9. Jensen, O., Nielsen, E., Rusters. The corrosive action of palmar sweat. II. Physical and chemical factors in palmar hyperhidrosis. *Acta Dermatovener (Stockholm),* 59, 139-143, 1979.
10. Cage, G. W., Wolfe, S. M., Thompson, R. H., and Gordon, R. S., Effects of water intake on composition of thermal sweat in normal human volunteers. *J. Appl. Physiol.*, 29, 687-690, 1970.
11. Brusilow, S. W. and Gordes, E. H., Ammonia secretion in sweat. *Amer. J. Physiol.*, 214, 513-517, 1967.
12. Mitchell, H. H. and Hamilton, T. S., The dermal excretion under controlled environmental conditions of nitrogen and minerals in human subjects, with particular reference to calcium and iron. *J. Biol. Chem.*, 178, 360, 1949.
13. Sato, K., The physiology, pharmacology, and biochemistry of the eccrine sweat gland. *Rev. Physiol. Biochem. Pharmacol.*, 79, 52-131, 1979.
14. Bayford, F., Sweat. *Fingerprint Whorld,* 1, 42-43, 1976.
15. Olsen, R. D., The chemical composition of palmar sweat. *Fing. Ident. Mag.*, 53(10), 4, 1972.
16. Sato, K., Feibleman, C., and Dobson, R. L., The electrolyte composition of pharmacologically and thermally stimulated sweat: a comparative study. *J. Invest. Dermatol.*, 55, 433-438, 1970.

17. Seutter, E., Goedhart-De Groot, N., Sutorius, H. M., and Urselmann, E. J. M., The quantitative analysis of some constituents of crude sweat. *Dermatologica*, 141, 226-233, 1970.
18. Schultz, I. J., Micropuncture studies of the sweat formation in cystic fibrosis patients. *J. Clin. Invest.*, 48, 1470–1477, 1969.
19. Verde, T., Shephard, R. J., Corey, P., and Moore, R., Sweat composition in exercise and in heat. *J. Appl. Phys. Respir. Environ. Exerc. Phys.*, 53, 1540–1545, 1982.
20. Kaiser, D. and Drack, E., Diminished excretion of bicarbonate from the single sweat gland of patients with cystic fibrosis of the pancreas. *Eur. J. Clin. Invest.*, 4, 261-265, 1974.
21. Miklaszewska, M., Free amino acids of eccrine sweat. Method. *Pol. Med. J.*, 7, 617-623, 1968.
22. Miklaszewska, M., Comparative studies of free amino acids of eccrine sweat and plasma. *Pol. Med. J.*, 7, 1313–1318, 1968.
23. Liappis, N., Kelderbacher, S. D., Kesseler, K., and Bantzer, P., Quantitative study of free amino acids in human eccrine sweat excreted from the forearms of healthy trained and untrained men during exercise. *Eur. J. Appl. Physiol.*, 42, 227-234, 1979.
24. Gitlitz, P. H., Sunderman, F. W., and Hohnadel, D. C., Ion-exchange chromatography of amino acids in sweat collected from healthy subjects during sauna bathing. *Clin. Chem.*, 20, 1305–1312, 1974.
25. Coltman, C. A., Rowe, N. J., and Atwell, R. J., The amino acid content of sweat in normal adults. *Am. J. Clin. Nutr.*, 18, 373-378, 1966.
26. Jenkinson, D., Mabon, R. M., and Manson, W., Sweat proteins. *Br. J. Dermatol.*, 90, 175-181, 1974.
27. Hadorn, B., Hanimann, F., Anders, P., Curtius, H-Ch, and Halverson, R., Free amino acids in human sweat from different parts of the body. *Nature*, 215, 416-417, 1967.
28. Hamilton, P. B., Amino-acids on hands. *Nature*, 205, 284-285, 1965.
29. Oro, J. and Skewes, H. B., Free amino-acids on human fingers: the question of contamination in microanalysis. *Nature*, 207, 1042–1045, 1965.
30. Liappis, N. and Hungerland, H., The trace amino acid pattern in human eccrine sweat. *Clin. Chim. Acta*, 48, 233-236, 1973.
31. Marshall, T., Analysis of human sweat proteins by two-dimensional electrophoresis and ultrasensitive silver staining. *Anal. Biochem.*, 139, 506-509, 1984.
32. Nakayashiki, N., Sweat protein components tested by SDS-polyacrylamide gel electrophoresis followed by immunoblotting. *J. Exp. Med.*, 161, 25-31, 1990.
33. Uyttendaele, M., De Groote, M., Blaton, V., and Peeters, H., Analysis of the proteins in sweat and urine by agarose-gel isotachophoresis. *J. Chromatogr.*, 132, 261-266, 1977.

34. Takemura, T., Wertz, eccrine sweat. *Br. J. I*
35. Boysen, T. C., Yana method of sweat coll
36. Lobitz, W. C., and M of studies on chlorid atinine. *Arch. Derma*
37. Förström, L., Goldyn sweat. *J. Invest. Derm*
38. Johnson, H. L. and M *J. Invest. Dermatol.*, 3
39. Vree, T. B., Muskens, amines in human swe
40. Naitoh, K., Inai, Y., an ping system and its us vapor released from h
41. Strauss, J. S., Downin glands. In Goldsmith *of the Skin, 2nd ed.* Ne
42. Nikkari, T., Compara 257-267, 1974.
43. Pochi, P. E. and Strauss activity of the human s
44. Greene, R. S., Downin variation in the amou *Invest. Dermatol.*, 54, 2
45. Shuster, S. and Thody tion. *J. Invest. Dermatol*
46. Ebling, F. J., Hormonal activity. *J. Invest. Derm*
47. Hamilton, J. B. and M oophorectomized wom
48. Pochi, P. E., Strauss, J fractional 17-ketosteroi castrates. *J. Invest. Derm*
49. Jarrett, A., The effects of of acne vulgaris. *Br. J. D*
50. Strauss, J. S. and Pochi, steroidal hormones and *vivo. Recent Progr. Horm.*
51. Oertel, G. W. and Treibe roids by human skin. *En*

34. Takemura, T., Wertz, P. W., and Sato, K., Free fatty acids and sterols in human eccrine sweat. *Br. J. Dermatol.*, 120, 43-47, 1989.
35. Boysen, T. C., Yanagawa, S., Sato, F., and Sato, K., A modified anaerobic method of sweat collection. *J. Appl. Physiol.*, 56, 1302–1307, 1984.
36. Lobitz, W. C., and Mason, H. L., Chemistry of palmar sweat. VII. Discussion of studies on chloride, urea, glucose, uric acid, ammonia nitrogen, and creatinine. *Arch. Dermatol. Syph.*, 57, 908, 1948.
37. Förström, L., Goldyne, M. E., and Winkelmann, R. K., IgE in human eccrine sweat. *J. Invest. Dermatol.*, 64, 156-157, 1975.
38. Johnson, H. L. and Maibach, H. I., Drug excretion in human eccrine sweat. *J. Invest. Dermatol.*, 56, 182-188, 1971.
39. Vree, T. B., Muskens, A. T. J. M., and van Rossum, J. M., Excretion of amphetamines in human sweat. *Arch. Int. Pharmacodyn.*, 199, 311-317, 1972.
40. Naitoh, K., Inai, Y., and Hirabayashi, T., Direct temperature-controlled trapping system and its use for the gas chromatographic determination of organic vapor released from human skin. *Anal. Chem.*, 72(13), 2797–2801, 2000.
41. Strauss, J. S., Downing, D. T., Ebling, F. J., and Stewart, M. E., Sebaceous glands. In Goldsmith LA, Ed. *Physiology, Biochemistry, and Molecular Biology of the Skin, 2nd ed.* New York: Oxford University Press, 1991.
42. Nikkari, T., Comparative chemistry of sebum, *J. Invest. Dermatol.*, 62, 257-267, 1974.
43. Pochi, P. E. and Strauss, J. S., Endocrinologic control of the development and activity of the human sebaceous gland. *J. Invest. Dermatol.*, 62, 191-201, 1974.
44. Greene, R. S., Downing, D. T., Pochi, P. E., and Strauss, J. S., Anatomical variation in the amount and composition of human skin surface lipid. *J. Invest. Dermatol.*, 54, 246, 1970.
45. Shuster, S. and Thody, A. J., The control and measurement of sebum secretion. *J. Invest. Dermatol.*, 62, 172-190, 1974.
46. Ebling, F. J., Hormonal control and methods of measuring sebaceous gland activity. *J. Invest. Dermatol.*, 62, 161-171, 1974.
47. Hamilton, J. B. and Mestler, G. E., Low values for sebum in eunuchs and oophorectomized women. *Proc. Soc. Exp. Biol. Med.*, 112, 374-378, 1963.
48. Pochi, P. E., Strauss, J. S., and Mescon, H., Sebum excretion and urinary fractional 17-ketosteroid and total 17-hydroxycorticoid excretion in male castrates. *J. Invest. Dermatol.*, 39, 475-483, 1962.
49. Jarrett, A., The effects of progesterone and testosterone on the surface sebum of acne vulgaris. *Br. J. Dermatol.*, 67, 102-116, 1959.
50. Strauss, J. S. and Pochi, P. E., The human sebaceous gland: its regulation by steroidal hormones and its use as an end organ for assaying androgenicity *in vivo. Recent Progr. Horm. Res.*, 19, 385-444, 1963.
51. Oertel, G. W. and Treiber, L., Metabolism and excretion of C_{19}- and C_{18}-steroids by human skin. *Eur. J. Biochem.*, 7, 234-238, 1969.

52. Downing, D. T., Strauss, J. S., Norton, L. A., Pochi, P. E., and Stewart, M. E., The time course of lipid formation in human sebaceous glands. *J. Invest. Dermatol.*, 69, 407-412, 1977.

53. Stewart, M. E., Steele, W. A., and Downing, D. T., Changes in the relative amounts of endogenous and exogenous fatty acids in sebaceous lipids during early adolescence. *J. Invest. Dermatol.*, 92, 371-378, 1989.

54. Downing, D. T., Lipolysis by human skin surface debris in organic solvents. *J. Invest. Dermatol.*, 54, 395-398, 1970.

55. Reisner, R. M., Silver, D. Z., Puuhvel, M., and Sternberg, T. H., Lipolytic activity of *Corynebacterium* acnes. *J. Invest. Dermatol.*, 51, 190-196, 1968.

56. Marples, R. R., Downing, D. T., and Kligman, A. M., Control of free fatty acids in human surface lipids by *Corynebacterium* acnes. *J. Invest. Dermatol.*, 56, 127-131, 1971.

57. Holt, R. J., The esterase and lipase activity of aerobic skin bacteria. *Br. J. Dermatol.*, 85, 18-23, 1971.

58. Weary, P., Comedogenic potential of the lipid extract of *Pityrosporum ovale*. *Arch. Dermatol.*, 102, 84-91, 1970.

59. Puhvel, S. M., Reisner, R. M., and Sakamoto, M., Analysis of lipid composition of isolated human sebaceous gland homogenates after incubation with cutaneous bacteria. Thin layer chromatography. *J. Invest. Dermatol.*, 64, 406-411, 1975.

60. Cove, J. H., Holland, K. T., and Cunliffe, W. J., An analysis of sebum excretion rate, bacterial population and the production rate of free fatty acids on human skin. *Br. J. Dermatol.*, 103, 383-386, 1980.

61. Marples, R. R. and Kligman, A. M., Ecological effects of oral antibiotics on the microflora of human skin. *Arch. Dermatol.*, 103, 148-153, 1971.

62. Cunliffe, W. J., Coterill, J. A., and Williamson, B., The effect of clindamycin in acne: a clinical and laboratory investigation. *Br. J. Dermatol.*, 87, 37-41, 1972.

63. Shalita, A. R., Wheatley, V. R., and Brind, J., Clinical and laboratory evaluation of antibacterial agents in the treatment of acne vulgaris (abstract). *J. Invest. Dermatol.*, 60, 250, 1973.

64. Marples, R. R., Kligman, A. M., Lantis, L. R., and Downing, D. T., The role of the aerobic microflora in the genesis of fatty acids in human surface lipids. *J. Invest. Dermatol.*, 55, 173-178, 1970.

65. Mykytowycz, R. and Goodrich, B. S., Skin glands as organs of communication in mammals. *J. Invest. Dermatol.*, 62, 124-131, 1974.

66. Downing, D. T., Strauss, J. S., and Pochi, P. E., Variability in the chemical composition of human skin surface lipids. *J. Invest. Dermatol.*, 53, 322-327, 1969.

67. Lewis, C. A. and Hayward, B., Human skin surface lipids. In Borrie P, Ed. *Modern Trends in Dermatology. Vol. 4*. London: Butterworths, 1971.

68. Haahti, E., Ma
erence to gas ch
59), 1961.

69. Nicolaides, N. a
Soc., 33, 404-40

70. Felger, C. B., Th
puberty. *J. Soc.*

71. Nordstrom, K.
terization of wa
Invest. Dermatol.

72. Darke, D. J. and
palmar and fore

73. Boniforti, L., Pas
Identification an
uid chromatogra

74. Green, S. C., Stew
composition amc

75. Nazzaro-Porro,
fatty acids in ski

76. Nicolaides, N., Si
1974.

77. Nicolaides, N. an
surface lipid. *Lip*

78. Krakow, R., Dow
a fatty acid in hu
J. Invest. Dermat

79. Stewart, M. E.,
control of the pro
wax esters. *J. Inve*

80. Kanda, F., Yagi,
Elucidation of ch
Dermatol., 122, 7

81. Nicolaides, N., Th
lipid and their rel

82. Nicolaides, N., Fu
of wax esters and
surface lipid. *Lipi*

83. Puhvel, S. M., Este
phylococcus epider
ulosum). *J. Invest.*

84. Freinkel, R. K. and
Dermatol., 52, 148

68. Haahti, E., Major lipid constituents of human skin surface with special reference to gas chromatographic methods. *Scan. J. Clin. Lab. Invest.*, 13(Suppl. 59), 1961.
69. Nicolaides, N. and Foster, R. C., Esters in human hair fat. *J. Am. Oil Chem. Soc.*, 33, 404-409, 1956.
70. Felger, C. B., The etiology of acne. I. Composition of sebum before and after puberty. *J. Soc. Cosmet. Chem.*, 20, 565, 1969.
71. Nordstrom, K. M., Labows, J. N., McGinley, K. J., and Leyden, J. J., Characterization of wax esters, triglycerides, and free fatty acids of folicular casts. *J. Invest. Dermatol.*, 86, 700-705, 1986.
72. Darke, D. J. and Wilson, J. D., The total analysis by gas chromatography of palmar and forehead lipids. *AERE Report No. G 1528*, 1979.
73. Boniforti, L., Passi, S., Caprilli, F., and Nazzaro-Porro, M., Skin surface lipids. Identification and determination by thin-layer chromatography and gas-liquid chromatography. *Clin. Chem. Acta*, 47, 223-231, 1973.
74. Green, S. C., Stewart, M. E., and Downing, D. T., Variation in sebum fatty acid composition among adult humans. *J. Invest. Dermatol.*, 83, 114-117, 1984.
75. Nazzaro-Porro, M., Passi, S., Boniforti, L., and Belsito, F., Effects of aging on fatty acids in skin surface lipids. *J. Invest. Dermatol.*, 73, 112-117, 1979.
76. Nicolaides, N., Skin lipids: their biochemical uniqueness. *Science*, 186, 19-26, 1974.
77. Nicolaides, N. and Ansari, M. N. A., The dienoic fatty acids of human skin surface lipid. *Lipids*, 4, 79-81, 1968.
78. Krakow, R., Downing, D. T., Strauss, J. S., and Pochi, P. E., Identification of a fatty acid in human surface lipids apparently associated with acne vulgaris. *J. Invest. Dermatol.*, 61, 286-289, 1973.
79. Stewart, M. E., McDonnell, M. W., and Downing, D. T., Possible genetic control of the proportions of branched-chain fatty acids in human sebaceous wax esters. *J. Invest. Dermatol.*, 86, 706-708, 1986.
80. Kanda, F., Yagi, E., Fukuda, M., Nakajima, K., Ohta, T., and Nakata, O., Elucidation of chemical compounds responsible for foot malodour. *Br. J. Dermatol.*, 122, 771-776, 1990.
81. Nicolaides, N., The monoene and other wax alcohols of human skin surface lipid and their relation to the fatty acids of this lipid. *Lipids*, 2, 266-275, 1966.
82. Nicolaides, N., Fu, H. C., Ansari, M. N. A., and Rice, G. R., The fatty acids of wax esters and sterol esters from vernix caseosa and from human skin surface lipid. *Lipids*, 7, 506-517, 1972.
83. Puhvel, S. M., Esterification of [4-^{14}C]cholesterol by cutaneous bacteria (*Staphylococcus epidermis, Propionibacterium acnes,* and *Propionibacterium granulosum*). *J. Invest. Dermatol.*, 64, 397-400, 1975.
84. Freinkel, R. K. and Aso, K., Esterification of cholesterol in the skin. *J. Invest. Dermatol.*, 52, 148-154, 1969.

85. Summerly, R., Yardley, H. J., Raymond, M., Tabiowo, A., and Ilderton, E., The lipid composition of sebaceous glands as a reflection of gland size. *Br. J. Dermatol.*, 94, 45-53, 1976.

86. Cunliffe, W. J., Cotterill, J. A., and Williamson, B., Skin surface lipids in acne. *Br. J. Dermatol.*, 85, 496, 1971.

87. Robertshaw, D., Apocrine sweat glands. In Goldsmith LA, Ed. *Physiology, Biochemistry, and Molecular Biology of the Skin. 2nd Ed.* New York: Oxford University Press, 1991.

88. Shelley, W. B., Apocrine sweat. *J. Invest. Dermatol.*, 17, 255, 1951.

89. Knowles, A. M., Aspects of physiochemical methods for the detection of latent fingerprints. *J. Phys. E. Sci. Instrum.*, 11, 713-721, 1978.

90. Toth, I. and Faredin, I., Steroid excreted by human skin. II. C_{19}-steroid sulfates in human axillary sweat. *Acta Med. Hung.*, 42, 21-28, 1985.

91. Labows, J. N., Preti, G., Hoelzle, E., Leyden, J., and Kligman, A., Steroid analysis of human apocrine secretion. *Steroids*, 34, 249-258, 1979.

92. Yamamoto, A., Serizawa, S., Ito, M., and Sato, Y., Effect of aging on sebaceous gland activity and on the fatty acid composition of wax esters. *J. Invest. Dermatol.*, 89, 507-512, 1987.

93. Ramasastry, P., Downing, D. T., Pochi, P. E., and Strauss, J. S., Chemical composition of human surface lipids from birth to puberty. *J. Invest. Dermatol.*, 54, 139-144, 1970.

94. Kärkkäinen, J., Nikkari, T., Ruponen, S., and Haahti, E., Lipids of *vernix caseosa*. *J. Invest. Dermatol.*, 44, 333-338, 1965.

95. Miettienen, T. A. and Lukkäinen, T., Gas-liquid chromatographic and mass spectroscopic studies on sterols in vernix caseosa, amniotic fluid and meconium. *Acta Chem. Scand.*, 22, 2603–2612, 1968.

96. Forest, G. M. and Bertrand, J., Sexual steroids in the neonatal period. *Steroid Biochem.*, 6, 24-26, 1975.

97. Stewart, M. E. and Downing, D. T., Measurement of sebum secretion rates in young children. *J. Invest. Dermatol.*, 84, 59-61, 1985.

98. Sansone-Bazzano, G., Cummings, B., Seeler, A. K., and Reisner, R. M., Differences in the lipid constituents of sebum from pre-pubertal and pubertal subjects. *Br. J. Dermatol.*, 103, 131-137, 1980.

99. Pochi, P. E., Strauss, J. S., and Downing, D. T., Skin surface lipid composition, acne, pubertal development, and urinary excretion of testosterone and 17-ketosteroids in children. *J. Invest. Dermatol.*, 69, 485-489, 1977.

100. Pochi, P. E., Strauss, J. S., and Downing, D. T., Age related changes in sebaceous gland activity. *J. Invest. Dermatol.*, 73, 108-111, 1979.

101. Kellum, R. E., Strangfeld, K., and Ray, L. F., Acne vulgaris. Studies in pathogenesis: triglycerides hydrolysis by *C. acnes in vitro*. *Arch. Dermatol.*, 101, 41-47, 1970.

102. Jacob
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103. Cunli
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104. Strau
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105. Plewi
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106. Cuthb
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107. Cuthb
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108. Wilso
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109. Noble
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110. Witze
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111. Noble

112. Fletch
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113. Schult
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114. Bucha
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89-95.

115. Robin
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116. Mong
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117. Bramb
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118. Jones
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119. Davies
excited
eous el

102. Jacobsen, E., Billings, J. K., Frantz, R. A., Kinney, C. K., Stewart, M. E., and Downing, D. T., Age-related changes in sebaceous wax ester secretion rates in men and women. *J. Invest. Dermatol.*, 85, 483-485, 1985.

103. Cunliffe, W. J. and Schuster, S., Pathogenesis of acne. *Lancet*, 1 (7597), 685-687, 1969.

104. Strauss, J. S. and Pochi, P. E., The quantitative gravimetric determination of sebum production. *J. Invest. Dermatol.*, 36, 293-298, 1961.

105. Plewig, G. and Kligman, A. M., Proliferative activity of the sebaceous glands of the aged. *J. Invest. Dermatol.*, 70, 314-317, 1978.

106. Cuthbertson, F., The chemistry of fingerprints. *AWRE Report, SSCD Memorandum SAC/8/65*, 1965.

107. Cuthbertson, F., The chemistry of fingerprints. *AWRE Report No. 013/69*, 1969.

108. Wilson, J. D. and Darke, D. J., The results of analyses of the mixtures of fatty acids on the skin. Part 1. commentary. *AERE Report No. G 1154*, ca. 1978.

109. Noble, D., Vanished into thin air: the search for children's fingerprints. *Anal. Chem.*, 67, 435A-438A, 1995.

110. Witze, A., Scientists do detective work on kid's fingerprints. *Dallas Morning News*. 21 April 1997, pp. 8D, 10D.

111. Noble, D., The disappearing fingerprints. *Chem Matters*. February, 9-12, 1997.

112. Fletcher, J. A., Gas Chromatography-Mass Spectrometry Identification of the Chemical Composition of Fingerprints, unpublished report, 1994.

113. Schultz, C. S., Determining the Chemical Composition of Children and Adult's Fingerprints Using Gas Chromatography-Mass Spectrometry, unpublished report, 1994.

114. Buchanan, M. V., Asano, K., and Bohanon, A., Chemical characterization of fingerprints from adults and children. *SPIE Photonics East Conf. Proc.*, 2941, 89-95, 1996.

115. Robinson, S. and Robinson, A. H., Chemical composition of sweat. *Physiol. Rev.*, 34, 215, 1954.

116. Mong, G. M., Petersen, C. E., and Clauss, T. R. W., Advanced fingerprint analysis project. Fingerprint constituents. *PNNL Report 13019*, 1999.

117. Bramble, S. K., Separation of latent fingermark residue by thin-layer chromatography. *J. Forensic Sci.*, 40, 969-975, 1995.

118. Jones, N. E., Davies, L. M., Brennan, J. S., and Bramble, S. K., Separation of visibly-excited fluorescent components in fingerprint residue by thin-layer chromatography. *J. Forensic Sci.*, 45, 1286–1293, 2000.

119. Davies, L. M., Jones, N. E., Brenna J. S., and Bramble, S. K., A new visibly-excited fluorescent component in latent fingerprint residue induced by gaseous electrical discharge. *J. Forensic Sci.*, 45, 1294–1298, 2000.

120. Duff, J. M. and Menzel, E. R., Laser-assisted thin-layer chromatography and luminescence of fingerprints: an approach to fingerprint age determination. *J. Forensic Sci.*, 23, 129-134, 1978.

121. Dikshitsu, Y. S., Prasad, L., Pal, J. N., and Rao, C. V. N., Aging studies of fingerprint residues using thin-layer and high performance liquid chromatography. *Forensic Sci. Int.*, 31, 261-266, 1986.

122. Lloyd, R., Lab on a chip may turn police into DNA detectives. *Washington Post*, 1 March 1999, A9.

123. Wu, C., Device eliminates wait for DNA results. *Sci. News*, 27 March 1999, 155, 199.

124. Bredemeier, K., In Virginia, fréedom from fear for crime victims, relief for families. *Washington Post*, 7 July 1999, A14.

125. Morrison, R. D., E-gels allow DNA results in 35 minutes. *Law Enforcement Tech*, 1999 August, 88-89.

126. Sweet, D. and Shutler, G. G., Analysis of salivary DNA evidence from a bite mark on a body submerged in water. *J. Forensic Sci.*, 44(5), 1069–1072, 1999.

127. Presley, L. A., Baumstark, A. L., and Dixon, A., Thé effects of specific latent fingerprint and questioned document examinations on the amplification and typing of the HLA DQ alpha gene region in forensic casework. *J. Forensic Sci.*, 38(5), 1028–1036, 1993.

128. Walls, C., Effects of latent print technology on PCR DNA analysis. *CBDIAI Examiner.* Fall, 17-18, 1997.

129. Stein, C., Kyeck, S. H., and Henssge, C., DNA typing of fingerprint reagent treated biological stains. *J. Forensic Sci.*, 41(6), 1012–1017, 1996.

130. Shipp, E., Roelofs, R., Togneri, E., Wright, R., Atkinson, D., and Henry, B., Effects of argon laser light, alternate source light, and cyanoacrylate fuming on DNA typing of human bloodstains. *J. Forensic Sci.*, 38(1), 184-191, 1993.

131. Newall, P. J., Richard, M. L., Kafarowski, E., Donnelly, W. J., Meloche, G. E., and Newman, J. C., Homicide case report: successful amplification and STR typing of bloodstains subjected to fingerprint treatment by cyanoacrylate fuming. *Can. Soc. Forensic. Sci. J.*, 29(1), 1-5, 1996.

132. Andersen, J. and Bramble, S., The effects of fingermark enhancement light sources on subsequent PCR-STR DNA analysis of fresh bloodstains. *J. Forensic Sci.*, 42(2), 303-306, 1997.

133. Fregeau, C. J., Germain, O., and Fourney, R. M., Fingerprint enhancement revisited and the effects of blood enhancement chemicals on subsequent profiler plus fluorescent short tandem repeat DNA analysis of fresh and aged bloody fingerprints. *J. Forensic Sci.*, 45(2), 354-380, 2000.

134. Miller, K., Blood reagents — their use and their effect on DNA. *FIRRS Bulletin No. 42*, November 1998.

135. Gross, A. M., Harris, K. A., and Kaldun, G. L., The effect of luminol on presumptive tests and DNA analysis using the polymerase chain reaction. *J. Forensic Sci.*, 44(4), 837-840, 1999.

136. Brignoli, C. and Coquoz, R., DNA compatibility with fingerprint detection techniques and blood reagents, paper presented at the International Fingerprint Research Group Meeting, 25-28 May 1999.

137. Hochmeister, M. N., Budowle, B., and Baechtel, F. S., Effects of presumptive test reagents on the ability to obtain restriction fragment length polymorphism (RFLP) patterns from human blood and semen stains. *J. Forensic Sci.*, 36(3), 656-661, 1991.

138. Roux, C., Gill, K., Sutton, J., and Lennard, C., A further study to investigate the effect of fingerprint enhancement techniques on the DNA analysis of bloodstains. *J. Forensic Ident.*, 49(4), 357-376, 1999.

139. Zamir, A., Oz, C., and Geller, B., Threat mail and forensic science: DNA profiling from items of evidence after treatment with DFO. *J. Forensic Sci.*, 45(2), 445-446, 2000.

140. Van Oorschot, R. A. H. and Jones, M. K., DNA fingerprints from fingerprints. *Nature*, 387, 767, 1997.

141. Bellefeuille, J., Bowen, K., Wilkinson, D., and Yamishita, B., Crime scene protocols for DNA evidence. *FIRRS Bulletin No. 45*, April 1999.

142. Ladd, C., Adamowicz, M. S., Bourke, M. T., Scherczinger, C. A., and Lee, H. C., A systematic analysis of secondary DNA transfer. *J. Forensic Sci.*, 44(6), 1270–1272, 1999.

143. Findlay, I., Taylor, A., Quirke, P., Frazier, R., and Urquhart, A., DNA fingerprinting from single cells. *Nature*, 389, 555-556, 1997.

144. Batzer, M. A., Arcot, S. S., Phinney, J. W., Alegria-Hartman, M., Kass, D. H., Milligan, S. M., Kimpton, C., Gill, P., Hochmeister, M., Ioannou, P. A., Herrera, R. J., Boudreau, D. A., Scheer, W. D., Keats, B. J. B., Deininger, P. L., and Stoneking, M., Genetic variation of recent alu insertions in human populations. *J. Mol. Evol.*, 42, 22-29, 1996.

145. Harpending, H. C., Batzer, M. A., Gurven, M., Jorde, L. B., Rogers, A. R., and Sherry, S. T., Genetic traces of ancient demography. *Proc. Natl. Acad. Sci.*, 95, 1961–1967, 1998.

146. Saferstein, R., DNA: a new forensic science tool. In *Criminalistics — An Introduction to Forensic Science, 7th edition.* Upper Saddle River: Prentice-Hall, 2000.

147. Lowe, A., DNA based predictions of physical characteristics. First International Conference on Forensic Human Identification in the Millenium, 1999, http://www.forensic.gov.uk/conference/papers_list.htm.

148. Jobling, M. A., The Y chromosome as a forensic tool: progress and prospects for the new millennium. First International Conference on Forensic Human Identification in the Millenium, 1999, http://www.forensic.gov.uk/conference/papers_list.htm.

149. Kloosterman, A., Application of Y-chromosome specific STR-typing in forensic stains. First International Conference on Forensic Human Identification in the Millenium, 1999, http://www.forensic.gov.uk/conference/papers_list.htm.

150. Van Oorschot, R. A. H., Szepietowska, I., Scott, D. L., Weston, R. K., and Jones, M. K., Retrieval of genetic profiles from touched objects. First International Conference on Forensic Human Identification in the Millenium, 1999, http://www.forensic.gov.uk/conference/papers_list.htm.

151. Claiborne, W., Beethoven: a life undone by heavy metal? *Washington Post*, 18 October 2000, A3.