AXILLARY ODOR
Experimental Study of the Role of Bacteria, Apocrine Sweat, and Deodorants

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AS FAR back as history records, man has been aware of a distinctively malodorous scent that may emanate from his axilla. Since the axilla is remarkable anatomically for the presence of the apocrine gland, the unique body odor of this area has been popularly ascribed to this structure. Furthermore, racial differences in body odor have been related to differences in number of apocrine glands seen in the various races. The intense acridity of body odor sometimes noted under stress situations is thought to be the result of apocrine sweating.

As a result of these speculations the apocrine gland has been thought of as a "scent gland," responding to emotional stimuli and thus performing the function of such similar glands in the lower mammalia. In studying the physiology of the apocrine gland we have been impressed by the lack of appreciable odor in pure apocrine sweat as it initially appears on the skin surface. Moreover, it was noted that upon standing such sweat later developed a definite foul odor, which increased in intensity as time progressed. The realization of this fact prompted an investigation of axillary odor, with particular reference to the specific roles of bacteria, apocrine sweat, and deodorants.

1. THE ROLE OF BACTERIA

A. The Bacteriology of Apocrine Sweat.—The axilla, a moist, intertriginous skin site, contains maximal numbers of resident and transient skin micro-organisms. However, there is no knowledge of the bacteriologic state of apocrine sweat. As a prelude to the study of the role of bacteria in the production of axillary odor, it was necessary to determine what organisms, if any, might normally reside in the apocrine secretion.

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Method: Twenty healthy men with no abnormalities of the axillae served as subjects.

1. Preparation of the axillae: The evening before the collection of the specimens, the axillae were closely shaved and cleansed thoroughly with Ivory soap. Again in the morning on arising, the axillae were cleansed with Ivory soap. Three hours later, immediately prior to collection of sweat specimens, the axillae were cleansed for a third time with Ivory soap. After rinsing, the axillae were swabbed with a pad soaked in 70% ethyl alcohol. Finally a 4 by 4 in. (10 by 10 cm.) Steri-Pad saturated with 70% ethyl alcohol was applied to the axilla and left in place for five minutes.

2. Collection of specimens of sweat: Upon removal of the alcohol-saturated Steri-Pad, a thin film of alcohol was seen on the skin surface. This was allowed to evaporate, and immediately afterward 0.15 cc. of a 1:1000 solution of epinephrine was injected subcutaneously to stimulate apocrine sweating. It is significant that eccrine sweating was not seen in these subjects during the collection period.

As soon as the apocrine sweat appeared, it was collected in a sterile capillary tipped glass pipette approximately 8 in. (20 cm.) in length. The dilated distal portion of the collecting tube was plugged with cotton in order to insure sterility. After the pure apocrine sweat was collected, the narrow capillary portion of the tube was dipped in a tube containing sterile petrolatum, thus sealing this end of the collecting tube to reduce evaporation of the specimen. The entire collecting tube then was placed in a large sterile test tube, capped with cotton, and promptly transported to the laboratory for bacteriologic study.

3. Methods of bacteriologic study: The contents of the fine capillary pipette were placed in 1 ml. of brain-heart infusion broth. Cultures were incubated aerobically and/or anaerobically. Hanging drop and Gram stain preparations were made of the broth cultures, and subcultures were made to nutrient agar and blood (horse) agar. When Gram-negative bacilli were present in the cultures, subcultures were also made to eosin-methylene-blue agar and SS agar.

The Brewer jar method was used in the anaerobic studies. Microaerophilic conditions were established with use of the Brewer jar method also, but the method was modified in that hydrogen was added, and the jar was not completely exhausted of oxygen.

Results: The bacteriologic study showed the following:

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<td>24</td>
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<td>Number with bacterial growth</td>
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Of the four specimens which showed bacterial growth, two contained organisms of the genus Corynebacterium; one, of the genus Aerobacter, and one, of the genus Sarcina.

Comment: In spite of elaborate techniques and significant technical improvement, it is essentially true that complete and consistent sterilization of the human skin is practically impossible. Bacterial counts can be significantly and even rapidly reduced, but because of the difficulty in protecting such a vast exposed organ, absolute skin sterility is as yet an elusive goal.

The only organisms grown in this study were common skin aerobes. These were of the Corynebacterium group, a normal resident of the skin surface, and two commonly found organisms, Sarcina (skin, air, soil, dust, etc.) and aerobacter (gastro-intestinal tract, air soil, etc.), both of which may be found in the transient flora of the skin surface. We feel that their presence in the four specimens was the result

of accidental contamination of the apocrine sweat during collection. It is of interest that no anaerobes were found in apocrine sweat since sebum has been shown commonly to harbor such organisms (Propionibacterium).

Conclusion: Human apocrine sweat is normally a sterile fluid.

B. In Vitro Study of Axillary Odor.—With the knowledge that apocrine sweat was a sterile fluid as it initially appeared on the skin surface, an attempt was made to study axillary apocrine odors in vitro and to determine the role of the axillary flora in the production of this odor.

Method: Ten healthy Negro men were chosen for this study. No abnormalities were present in their axillae.

1. Preparation of the axillae: One axilla was prepared exactly as described above. The opposite axilla was unshaved and was not cleansed for at least 24 hours prior to collection.

2. Collection of the specimen: Collection of the specimen was identical with that described under bacteriologic study.

Ten microtubes (Tubes No. 1) were filled with pure apocrine sweat taken from the cleansed axilla, and 10 tubes (Tubes No. 2) were filled with pure apocrine sweat taken from the uncleansed axilla. In addition, another series of tubes (Tubes No. 3), especially prepared with a thin film of hexachlorophene in absolute alcohol lining the interior of the tube, were filled with pure apocrine sweat collected from the uncleansed axilla. All of these tubes were then allowed to incubate at room temperature and studied at varying intervals. Several similar specimens were kept refrigerated at 0°C.

3. Examination of the specimens: The specimens incubated at room temperature were then examined for odor in a careful, consistent manner at 1 hour, 6 hours, and 24 hours, then once daily up to the fifth day. Later examinations were made on the 8th, 11th, and 14th days. They were read in the same order at each examination, i.e., Tubes No. 1 (containing pure apocrine sweat from the prepared axilla); Tubes No. 2 (containing pure apocrine sweat from the unprepared axilla with hexachlorophene lining the interior of the tube); Tubes No. 3 (containing pure apocrine sweat from the unprepared axilla).

The tube was broken at the capillary end and then tested for odor by two examiners, and the results were recorded independently by each examiner, without knowledge of the other's reading.

Results (Fig. 1): No odor could be detected in any of the tubes at one hour after collection. In Tubes No. 1 no odor developed throughout the entire 14-day period. Tubes No. 2 had no perceptible odor until the final reading on the 14th day, when a faint odor was noted. Tubes No. 3 at six hours generated a strong odor which became very strong at 24 hours and remained at this intensity throughout the rest of the readings. In the refrigerated specimens no odor ever developed.

Comment: It should be emphasized that at one hour no odor was noticed in any of the tubes, as the graph indicates. This is to be expected, of course, if bacteria play any role in the production of the odor, since pure apocrine sweat was collected and the contaminant axillary bacteria would require an incubation period to produce this odor.

In the capillary tube, it may take longer for this odor to develop than in situ, since a smaller number of organisms may be present, or since in such a small tube the early odor changes might be below the threshold of our relatively insensitive olfactory organs.

The order in which the readings were made is significant, since it is possible that if Tubes No. 3 were studied first, the odor, which became intolerably strong on the second day, might have been followed temporarily by a period of anosmia.
It is interesting to note that on the 14th day there was some faint odor in Tubes No. 2, after all other readings of this tube had shown no odor. This would indicate that hexachlorophene had begun to lose its potency and that it was not entirely bactericidal but perhaps only bacteriostatic (at least for some organisms). Furthermore, it indicates that the axillary organisms were included in these specimens.

Of great significance is the fact that the odor developed in these tubes was, as far as we could tell, identical with the acrid axillary odor which we identify with the axilla of man. It is not surprising that the refrigerated samples failed to develop any odor, since the necessary bacterial activity would be absent under such conditions.

Summary: 1. Axillary odor was captured in the “test tube.” It was produced within six hours after collection of pure apocrine sweat from an uncleansed, unshaved axilla.

![Diagram](https://example.com/diagram.png)

**Fig. 1.—Effect of bacteria in development of odor in pure apocrine sweat.** Tube 1 represents the series of tubes containing pure apocrine sweat from a “sterile” axilla. Note the absence of any odor. Tube 2 represents the series of hexachlorophene-coated tubes containing pure apocrine sweat from an “unsterile” axilla. No odor developed in these tubes until the 14th day. Tube 3 represents the series of tubes containing apocrine sweat from the “unsterile” axilla.

2. No odor was noted in the samples of pure apocrine sweat collected from the prepared “sterile” axilla even after 14 days.

3. The addition of hexachlorophene to a tube containing pure apocrine sweat from a normal unprepared axilla prevented any odor from developing until the 14th day, when a faint yet typical axillary odor was recorded.

Conclusion: Bacterial action is necessary for the production of odor from apocrine sweat. This experimentally produced odor is indistinguishable from the classical axillary odor seen in man.

C. In Vivo Study of Axillary Odor.—Although in vitro studies clearly indicated that bacterial action upon apocrine sweat would result in the typical body odor and that such odor could be prevented by removal or destruction of these bacteria, it was necessary to test this principle in situ; therefore an in vivo study was made of the effects of the use of a popular “germicidal” detergent on axillary odor and bacterial flora.
Method: Twenty-five healthy Negro men were chosen for this study and were divided into two groups of 10 (Group I) and 15 (Group II) men each. No abnormalities were present in their axillae, and axillary hair growth was good in all subjects.

Group I: 1. Preparation of the axillae: No shaving of axillary hair was done in these subjects. One axilla was carefully washed for five minutes with pHisoHex 4 once daily for seven days. The opposite axilla was also carefully washed for five minutes with pHisoderm once daily for seven days. Thorough rinsing of the axilla with tap water was done in each case following each wash, but no other soap, deodorant, or alcohol was applied to these areas during this trial period.

2. Examination of the axillae: These men were tested for the presence of axillary odor at 1-, 2-, 8-, and 18-hour periods. Subjects were placed in a supine position with their hands beneath their head and elbows directed laterally. Two examiners unaware of what the previous treatment had been carefully appraised these axillae for odor, each independently stating their findings to a recorder. A rest period of 10 minutes was allowed between readings. Readings were either negative (no odor) or positive (presence of odor), and no attempt was made to grade the readings. The odor of hexachlorophene, which was in itself pleasant, very faint, and easily distinguished from the classic axillary body odor, was recorded as negative (no odor), unless an axillary odor coexisted, in which case a positive reading was made. Differences in readings, when they occurred, were recorded as positive.

Group II: 1. Preparation of the axillae: The axillary hair was unshaved in these men also. For three days a daily five-minute wash with pHisoHex (to one axilla) and pHisoderm (to the opposite axilla) was done. Thorough rinsing of the axillae with tap water followed the washing, and no other soap, deodorant, or alcohol was applied to the areas during this trial period.

2. Collection of specimens: Two and one-half hours after the last axillary wash, 10 of these men were seen and placed in a supine position with their hands beneath their heads and elbows directed laterally. With sterile scissors and forceps, axillary hairs were removed from the respective axillae and placed on blood agar plates. Only one hair was placed on each plate. They were then incubated at 37.5 C. for 48 hours.

In the other five men, axillary hairs were removed and cultured in the same manner 20 hours after the last axillary wash.

Results: Group I. As is seen in Figure 2, the pHisoHex-treated axillae showed a significantly greater odor protection than the control axilla.

Group II: Of the hairs collected two and one-half hours after the last wash, none from the pHisoHex-washed side showed growth, whereas all of the hairs from the pHisoderm side showed luxuriant growth (Fig. 3).

In the test 20 hours after the last axillary wash, only one of five axillary hairs from the pHisoHex-washed axillae showed bacterial growth, while four of five pHisoderm-washed axillary hairs showed excellent growth after incubation at 37.5 C. for 48 hours.

Comment: It has been shown that pHisoHex 4 greatly reduces skin flora when used exclusively and repeatedly for cleansing. The effect of the hexachlorophene was revealed by using the same detergent base, pHisoDerm, as a control.

4. pHisoHex is 18.4% hexachlorophene in pHisoderm, a water-miscible detergent preparation containing entsufon (sodium octylphenoxethoxyethyl ether sulfonate), wool fat, cholesterol, and petrolatum. Both pHisoHex and pHisoderm are manufactured by Winthrop-Stearns, Inc., 1450 Broadway, New York.


Fig. 2.—Effectiveness of an antibacterial-detergent preparation (pHisoderm) as an axillary wash in reducing axillary odor as compared with the detergent alone in the opposite axilla. The unshaved axillae of 10 men were washed once daily for seven days with the above preparations and examined for odor at the end of this time.

Fig. 3.—A, a hair which was taken from the axilla of a subject who had daily axillary washes with pHisoderm for three days. Note the luxuriant bacterial growth on the agar plate, after 48 hours' incubation at 37.5 C. B, a similar hair from the opposite pHisoHex-washed axilla, in which no such growth is present.
It should be stressed that mechanical cleansing of the axilla is most important, and large amounts of antiseptics will hardly suffice in the absence of such cleansing. It is difficult to sterilize any skin surface, especially intertriginous sites, and diligent, thorough washes were, therefore, insisted upon in this investigation.

Conclusion: The use of pHisoHex in a daily five-minute wash resulted in the abolition of odor in seven subjects for as long as 18 hours as compared with abolition in two subjects using pHisoderm preparation. Concomitantly there occurred striking reduction in axillary bacterial flora as revealed by the study of representative hairs. This observation parallels the in vitro study on apocrine sweat and offers in vivo evidence that bacterial action is necessary for the production of body odor from the axilla.

II. THE ROLE OF APOCRINE SWEAT

The typical odor of the human axilla is certainly unique among the odors emanating from various body sites. Familiar to all of us, it has been attributed to the presence of apocrine sweat, but no direct studies had previously been made. Recent investigation of the physiology of the apocrine gland ² have enabled us to examine apocrine sweat itself rather than mixed “axillary secretion” previously studied. Pharmacologic stimulation of the apocrine gland with epinephrine and immediate collection of the apocrine sweat droplets at their point of origin prevented any such contamination with other axillary secretions. In the earlier section of this paper it was demonstrated that normal apocrine sweat is initially sterile and odorless. Axillary organisms rapidly contaminate the sweat, and the classical axillary body odor developed in such unsterile specimens of pure apocrine sweat.

There is still further proof of the role of apocrine sweat in the production of axillary odor.

The endocrine system exerts a profound influence upon the apocrine sweat gland. The gland is undeveloped before the age of puberty and undergoes some degree of atrophy following the climacteric.⁷

It is known that children of the prepubertal age are without the classical axillary odor, and it has been shown that they do not produce apocrine sweat. ³ It is believed also that many of the postclimacteric group have little or no such distinct odor. A study was made to see if there was a correlation between absence of odor and absence of apocrine sweat in elderly individuals.

Method: Five healthy men, ranging in age from 65 to 70 years were chosen for examination. The axillae were unshaved and unwashed for at least 12 hours prior to the examination. The patients were placed in a supine position, hands beneath their heads, with elbows pointing laterally and axillae exposed. Each was examined for axillary odor and was then given a test dose of epinephrine to stimulate apocrine sweating.

Results: Four of the five subjects studied showed the usual axillary odor. Those four also presented true apocrine sweat after adrenergic stimulation. The fifth subject, a 69-year-old white man, did not show the typical axillary odor, but rather a flat, somewhat vague scent. This patient also produced no apocrine sweat after stimulation.

Comment: The apocrine sweat in these four men was carefully examined and was identified with certainty according to the criteria elaborated previously. ² These

criteria were: (1) sweat was turbid white on the surface and in a capillary tube; (2) it dried to form a glistening glue-like cap; (3) it was follicular in location; (4) it appeared after an adrenergic stimulus. An equally thorough search for this secretion was made in the fifth subject, but none was seen.

Aprocrine sweat may be seen in the older age groups, as we have previously stated. However, there is no doubt that the amount is diminished just as the number of functioning glands is diminished. Yet even with minimal quantities, the essential substrate, namely, apocrine sweat, is available and will markedly influence the resultant odor of the area.

Conclusions: Aprocrine sweat is the necessary substrate for the production of the classic axillary odor of man.

III. OTHER FACTORS INFLUENCING AXILLARY ODORS

A. Axillary Hair.—The presence of terminal hair in the axillae is commonly regarded as an additional factor incrementing axillary odor. It is felt that retained axillary secretion and debris on the hair shaft act as an excellent medium for the growth of micro-organisms. Moreover, cleansing is made more difficult by the presence of such hair, and bacteria cling tenaciously to it. An attempt was therefore made to determine the influence of such hair in the development of axillary odor.

Method: Ten healthy Negro men were chosen for study. No abnormalities of the axillae were present.

1. Preparation of the axillae: One axilla was shaved and carefully washed with Ivory soap for 10 minutes. The opposite axilla was unshaved and was also washed with Ivory soap for 10 minutes. Each axilla was thoroughly rinsed with water.

2. Examination of the axillae: Olfactometric observations were then made in the same manner as previously described.

Results: In Figure 4 the results are clearly illustrated. The unshaved axillae of 2 men had a definite odor at the one-hour reading, and at six hours 9 of the 10
subjects showed such odor. At 48 hours all 10 of this group had the usual axillary scent. Contrast these with the shaved axillae, where no odor was detectable at 6 hours, and even at 24 hours only one man showed axillary odor. However, at 48 hours the shaved axillae of 9 of the 10 subjects showed the usual odor.

Comment: The very significant reduction in axillary odor in the shaved axillae substantiates the concept that retention of axillary secretion and bacteria will prompt an increase in odor.

It is apparent that the effect of this shaving was remarkable and was similar to the results of using pHisoHex in the unshaved axillae. It is understandable, therefore, that in women who regularly shave their axillae there is a markedly diminished axillary odor.

Conclusion: Axillary hair acts as a collecting site for axillary secretion, keratin, and debris, and favors bacterial growth and decomposition of such retained material. The removal of this hair results in marked reduction or elimination of appreciable axillary odor for as long as 24 hours.

B. Clothing.—Various authors have stated that in odor to secure an odor-free area it is necessary to remove or reduce the surfaces upon which the secretions can cling. This is certainly true in the case of the axillary hair, as was demonstrated above. It also becomes apparent that clothing may play a similar role in odor production. Its influence in this process was investigated.

Method: Four healthy white men were chosen for this study.

1. Preparation of the axillae: The axillae were unshaved and were washed with pHisoHex for five minutes daily for one week. No special stimuli, emotional experience, or excessive exercise was encountered during this period, and no other soap, deodorant, alcohol, or other chemical was applied to the axillae. In all subjects eccrine sweating was normal and stained the shirt surface in the area. All wore clean white shirts during this study. Examination of the odor of the axillae and the shirts was then made at 1-, 3-, 6-, 12-, and 24-hour intervals.

Results: A great diminution of axillary odor was seen during this entire period.

It was noticed, however, that within two to three hours the definite body odor developed in the shirts at the site adjacent to the axillary skin. This zone was stained by axillary secretion.

Comment: The practical significance of this study is immediately appreciated. While we may with various techniques approach sterility in the axilla and thus remove odor of this area, it is true that bacteria normally reside on adjacent clothing and when the substrate, i. e., apocrine sweat, is present, the necessary decomposition will occur and odor will develop. Thus, even if the axilla is odorless, some odor can develop from the adjacent clothing. This is especially true if apocrine sweating is excessive, since obviously large amounts of the required odor substrate, apocrine sweat, will be available.

Conclusion: Clothing will definitely aid in the production of axillary odor, since organisms may cling to this clothing along with the axillary secretion and then result in bacterial decomposition and subsequent odor production.

C. Eccrine Sweat.—It has been said that eccrine sweat is a sterile, odorless fluid upon secretion. Furthermore, it has been claimed that after the sweat stands for several hours an odor will develop, presumably because of bacterial action. However, the collection techniques employed in these studies did not allow for an accurate study of pure eccrine sweat, since the samples were contaminated with keratin, debris, sebum, apocrine sweat, or hair. It is apparent that the study of the odors of pure, normal eccrine sweat depends upon a collection of uncontaminated specimens. In the present investigation appropriate techniques were employed to permit collection of such uncontaminated eccrine sweat.

Method: Profuse thermal sweating was induced in a normal healthy man in whom the axillae had been thoroughly cleansed and shaved. Uncontaminated eccrine sweat droplets were collected from the axillae in the small glass pipettes in the manner described earlier. It should be stressed that the secretion was absolutely clear and watery in consistency.

Two sets of five tubes each were collected, viz., Tubes A, containing pure eccrine sweat; Tubes B, containing pure eccrine sweat and with a thin film of hexachlorophene lining the interior of the tubes.

In addition, small vials of contaminated eccrine sweat were collected from the trunk, legs, and neck. A "scrapping" technique, that is, running the mouth of the vial along the skin of these sites, was used. The fluid in these vials was not clear, but was turbid white in appearance. These tubes were then incubated and were independently examined for odor at 2, 24, 48, 72, and 96 hours by two investigators.

Results: It was found that no odor whatsoever developed in any of the tubes of pure eccrine sweat that were collected from the axillae.

Appraisal of the small vials containing contaminated eccrine sweat from the trunks, legs, and necks revealed that a distinct odor was present at 48 hours. This odor was not pungent or at all offensive and was markedly different from the "axillary odor" previously developed in vitro. This odor increased slightly in intensity at the end of 96 hours. These specimens were cultured for bacteria, and all showed definite heavy growth both in brain-heart infusion broth and on blood agar plates in 48 hours.

Comment: It is almost impossible to properly evaluate the specific roles of body secretions in the production of odor unless one is able to selectively study the secretion itself. Such contaminants as keratin, sebum, and debris could be responsible for odor development per se and could, therefore, mask the primary odor.

It seems apparent that eccrine sweat is of no importance as a primary odor source. However, it would seem that a definite function might be ascribed to this secretion in the production of axillary odor. Aside from the obvious impetus to bacterial growth it affords, eccrine sweat might also aid in the volatilization of the odoriferous products of bacterial decomposition; thus, the apparent "flush" of odor at an emotional crisis, when emotional eccrine sweating occurs. Fresh apocrine sweat, while odorless when it initially appears on the skin surface, could also aid in the volatilization process.

Conclusion: Uncontaminated eccrine sweat has no perceptible odor, whether sterile or unsterile. The addition of bacteria and of contaminants such as keratin, sebum, debris, apocrine sweat, and hair to eccrine sweat is necessary for the development of any odor. Such odor is rather vague, flat, and indistinct, and totally unlike that generated from apocrine sweat.

**IV. THE ROLE OF ANTIPERSPIRANT-DEODORANT PREPARATIONS**

Investigation of axillary odor almost inevitably leads to a study of the effects of antiperspirant-deodorant preparations. Such substances are used regularly by a vast proportion of the American public, yet their mechanism of action is not entirely clear.

*A. Antiperspirant Activity of the Aluminum Salts.*—Sulzberger, Zak, and Herrmann 11 showed histologically in biopsy specimens from the axilla that aluminum chloride preparations may produce periductal inflammatory changes which result in eccrine duct occlusion. They implied that a resultant diminution of eccrine sweat is produced at the skin surface. It is admitted that not all of the sweat glands of the area are rendered functionless, and that only a partial anhidrosis would result. We have attempted to determine whether a significant reduction in the amount of eccrine sweating could be demonstrated physiologically.

Sulzberger and his associates 11 also stated that a degenerative change occurred in the apocrine tubular cell after the repeated use of aluminum chloride preparations. Again their studies were limited to the histologic approach.

From the clinical test standpoint our preliminary observations 12 suggested that aluminum chloride paste deodorant preparations might reduce or abolish apocrine sweating. This early work has not been confirmed in our later more carefully controlled studies detailed below.

**Methods:** Ten healthy men were chosen for this study. These men had eccrine sweating of equal amounts in each axilla, as determined by the Randall starch paper-iodine technique. The axillae were shaved at the beginning of the study and on the sixth day. This was done in order to facilitate the proper prints with the starch paper-iodine method. Five-minute daily applications of 25% aqueous aluminum chloride solution were made in the one axilla for seven days. The opposite control axilla was similarly treated with plain water. At the end of this period simultaneous sweat imprints were made of both axillae. Satisfactory sweat responses could be obtained in the absence of any direct emotional or pharmacologic stimuli.

Fifty healthy men were selected from approximately 100 subjects for a study of apocrine sweat. All of these men had abundant and symmetrically equal apocrine sweat responses. The axillae were unshaved, and washing was limited to once daily. Nothing was applied other than the product under study. The following preparations were studied in 10 men each: (a) 25% aluminum chloride in aqueous solution; (b) spray deodorant A (commercial); (c) spray deodorant B (commercial); (d) cream deodorant (commercial); (e) zinc oxide paste.

In the case of the aluminum chloride solution the application technique consisted of placing gauze pledgets (4 by 4 in. [10 by 10 cm.]]) saturated with the solution in the axilla for five minutes daily for seven days, as was done previously in these studies. All other test substances were applied in one axilla once a day for seven days. In all instances the opposite axilla served as a control and was untreated except for the usual washing. At the end of a week the


axillae were carefully cleansed with alcohol to remove all debris, accumulated cream, paste, or liquid deodorant, so that there would be no difficulty in identification of the apocrine sweat. These men were then given a standard test dose of epinephrine (0.15 cc. of 1:1000 solution) subcutaneously in each axilla. We then observed the amounts of apocrine sweat produced, and compared the control and test sites.

Results: In all 10 subjects no significant reduction in the amounts of eccrine sweating was noticed as shown by the prints with the Randall starch paper-iodine technique. Comparison of these prints on the seventh day with those taken before the experiments began revealed no appreciable change.

No significant decrease in the quantity of apocrine sweat could be detected on the treated or the untreated axillae. In three of the men who had used the aluminum chloride preparations miliaria vesicles were produced on the treated areas.

Comment: The lack of any significant reduction in axillary eccrine sweating following the application of aluminum chloride may seem paradoxical. Admittedly, on other nonflexural body sites, such as the back or abdomen, such treatment would definitely produce some degree of anhidrosis, which after repeated application of the aluminum salts would become more and more complete. However, a satisfactory explanation of this deviation may be that the axillary eccrine sweating constantly washes away the aluminum salts and thus prevents the usual effects of these substances elsewhere, viz., injury to the keratin with subsequent eccrine poral occlusion, and resultant anhidrosis. If this explanation is correct, it would be anticipated that
persons showing minimal sweating in the axillae would derive the greatest relative antiperspirant effect of these aluminum agents. None of the subjects studied by us fell into this hypohidrotic group.

Several vehicles were employed in the study of the effects of these preparations on apocrine sweating. Ointment and liquid commercial deodorants, which contain perfumes as well as aluminum salts, and a simple 25% aqueous aluminum chloride solution were used. All axillae were given maximal exposure to the test substances. The zinc oxide paste was employed to determine the possible mechanical blocking effect of such preparation. It, too, was ineffective in producing any reduction in apocrine sweating. This might be expected since these glands will secrete against an external pressure of 225 mm. of mercury. 2b

Conclusions: By qualitative test methods no significant reduction in the amount of apocrine or eccrine sweating could be demonstrated following the repeated use of aluminum chloride preparations.

B. Deodorant Activity of the Aluminum Salts.—Since it has been shown that the aluminum preparations as commonly used in the axilla do not possess appreciable antiperspirant activity, it would appear their chief role is that of a deodorant. Their continued use by so many persons obviously supports such an assumption. Studies were undertaken, therefore, to evaluate the magnitude and significance of this reputed action.

Method: The unshaved axillae of 10 healthy men were cleansed daily as usual. Saturated pledgets containing 25% aqueous aluminum chloride solution were applied daily for five minutes to one axilla for seven days. A similar application of water was used on the control axilla.

These men were then examined for axillary odor at 2 and 18 hours after the last application.

Results: At two hours, 6 of the 10 subjects showed absence of any axillary odor on the treated side. The remainder of these men showed an alteration of the typical axillary odor to a relatively inoffensive one. At 18 hours 2 of the 10 subjects showed abolition of the axillary odor on the treated side, and 2 showed alteration. The remaining six men had typical axillary odor. In all of the subjects the control axillae presented a pungent offensive odor.

Conclusion: Aluminum chloride preparations have deodorant activity. They may completely abolish axillary odor in some individuals for 12 to 18 hours or may render the usual axillary odor relatively inoffensive so that it will be disregarded.

C. Mechanism of Deodorant Action of Aluminum Salts.—The problem of mechanism of action of the deodorant preparations containing aluminum salts, was then explored. It was felt that the action might be antibacterial and/or chemical in nature. The following studies were done to investigate these possibilities.

Methods: Ten healthy men were again chosen for study. The axillae were unshaved and were cleansed daily as usual. On one axilla a saturated pledget of 25% aqueous aluminum chloride solution was applied daily for five minutes for three days. A similar application of water was used on the control axilla. Two and one-half hours after the last application, the men were examined, and axillary hairs were selected from the axillae and placed on blood agar plates. These hairs were collected with scissors and forceps which were kept sterile at all times. One hair was placed on each culture plate. The cultures were then incubated at 37.5 C. for 48 hours.

Pooled apocrine sweat was collected then from 20 normal healthy men and placed in a small vial. At the end of 48 hours a very strong odor developed in this vial. One-tenth cubic centi-
meter of this apocrine sweat was then added to 3 cc. of distilled water and to a 25% aqueous aluminum chloride solution, respectively. The specimens were then examined immediately for any odor change.

Results: Of the aluminum-chloride-treated axillary hairs from the 10 subjects, six showed no bacterial growth on the agar plates; four showed good growth. The axillary hairs of the control side all showed luxuriant bacterial growth at 48 hours. An example is illustrated in Figure 6.

![Figure 6](https://via.placeholder.com/150)

Fig. 6.—A, a hair which was taken from the axilla of a subject who had daily five-minute applications of tap water to one axilla for three days. Note the luxuriant bacterial growth on the agar plate after 48 hours' incubation at 37.5 C. B, a similar hair from the opposite aluminum chloride-treated axilla in which no such growth is present.

The specimen containing water plus apocrine sweat gave the typical acrid odor one might expect. However, an instantaneous odor change occurred in the solution containing the aluminum chloride and apocrine sweat. The resultant odor was decidedly different from the original odor and may be described as a somewhat sharp yet relatively inoffensive scent. Dilution of this solution of the apocrine sweat and aluminum chloride resulted in a gradual diminution of the odor to the point
where it became somewhat pleasant. This odor was similar to the altered odor noted in the axillae of the men treated with the aluminum chloride in the previous experiment.

Comment: Certain aluminum salts are antibacterial, and the experiment above supports this fact. The effect is limited, however, and was not as marked as was seen in the hairs from the hexachlorophene-washed axilla.

It is probable, therefore, that much of the deodorant effect of aluminum salts is antibacterial in nature.

Conclusion: The deodorant action of aluminum chloride preparations is twofold in mechanism. There is both an antibacterial effect, thus preventing odor development, and a chemical change, thus rendering the normal odoriferous products of bacterial decomposition inoffensive.

GENERAL COMMENT

Although scientific section discussion has been given, some features of this study deserve further clarification and amplification.

In the normal person we have noticed some variation in the type of axillary odor produced in the absence of any special soap, deodorant, antiperspirant, or perfume. We have arbitrarily divided these odors into three general categories: (1) typical acrid body odor, which is commonest and which requires little description; (2) a rancid odor, which is quite intolerable and reminds one of rancid butter; (3) a relatively vague, indistinct, and less offensive odor.

In addition, there are occasional odors which are unique. It should be stressed that all of these odors are quite different from other body odors and would not in any way interfere with the identification as axillary odor. They are all easily distinguished from the odor of contaminated eccrine sweat, for example, or that of sebum, urine, or feces.

The axilla, as an intertriginous surface with maximal amounts of secretion and optimal growth conditions, is responsible for a large number of bacterial colonies. Little is written regarding the normal bacteriology of the axilla, and an attempt was made by us to outline the normal range of flora in the axillae of our subjects. Swabs taken from the axillae of 20 healthy men were inoculated on blood (horse) infusion agar, and, in general, the following bacteria were found routinely: (1) Micrococcus pyogenes, var. aureus (coagulase-positive Staphylococcus); (2) Micrococcus pyogenes, var. albus (coagulase-negative Staphylococcus); (3) Cornebacteria; (4) Aerobacter aerogenes; (5) Sarcina lutea.

Speculation upon the relative effects of the various bacteria is interesting. One might assume that the presence of the coliform organism would allow for a fecal odor of some sort. However, this does not seem to be the case. As a matter of fact, in two instances in which apocrine sweat was collected from the unsterile axilla of a normal healthy Negro such sweat developed a rather pleasant odor after about 48 to 72 hours. This odor remained pleasant even at the end of seven days. Bacteriologic

examination of both specimens revealed pure cultures of Aerobacter aerogenes. This may indicate that individual variations in axillary odor may reflect either chemical differences in apocrine sweat or differences in resident bacteriologic flora or both.

It is interesting to note that in a young white man suffering from severe axillary eccrine hyperhidrosis relatively little axillary odor was encountered, in spite of definite apocrine gland activity. It is likely that the enormous amounts of eccrine sweat produced continually washed away all apocrine secretion and thereby the substrate necessary for the production of axillary odor.

Comment should be made concerning the fact that the odor of the axilla arises in a manner common to odors of diverse nature, namely, on the basis of bacterial activity. Hence the apocrine gland cannot be viewed as a specific scent gland, and the bacterial genesis of axillary odors seriously imperils some of the teleological theorizing as to the sexual significance of axillary odor.14

Recently chlorophyll has received a great deal of publicity regarding its role as a deodorant. We are unable to attribute any specific axillary deodorant action to chlorophyll after oral administration. In five subjects who took daily 600 mg. oral doses of chlorophyll for seven days there was no odor reduction as compared with the initial axillary odor before administration of the drug. These findings support those of Brocklehurst.15

Finally, we may glean from this work some practical recommendations regarding the most effective means of reducing or inhibiting axillary odor. There is no uniformly effective way of significantly diminishing apocrine or eccrine sweating of the axilla. We are forced to rely almost completely, therefore, on a deodorization process. Clearly, the most complete and prolonged means of preventing axillary odor includes shaving of the axillary hair, plus the topical application of a potent antibacterial substance, such as hexachlorophene. We used pHisoHex because of its proved bacteriostatic effect, ease of application, and low sensitizing index.

**SUMMARY AND CONCLUSIONS**

1. Apocrine sweat is odorless and sterile when it initially appears on the skin surface.
2. Axillary micro-organisms acting on the apocrine sweat in vitro will promote the development of the typical acrid axillary body odor within a few hours. Exclusion of these organisms (sterile apocrine sweat) or inhibition of their growth (addition of hexachlorophene to the sweat) will prevent any odor from developing.
3. The only substrate required for the production of this distinctive malodorous scent is apocrine sweat.
4. From a practical standpoint, the intensive use of hexachlorophene-detergent preparations will abolish the axillary odor for more than 18 hours in most persons.
5. The presence of hair greatly increases axillary odor, since it acts as a collecting site for axillary secretions, debris, keratin, and bacteria. Shaving and careful washing of the axilla eliminate odor for more than 24 hours.

6. Pure eccrine sweat, whether sterile or unsterile, neither has nor develops an odor. Eccrine sweat, contaminated with sebum, keratin, or debris, develops odor presumably as a result of bacterial action. This odor is mild and very definitely distinct from the odor developing in pure apocrine sweat. The role of the eccrine sweat in the production of axillary odor consists of an accentuation of bacterial growth and also the volatilization of the odoriferous compounds derived from apocrine sweat.

7. No significant reduction in eccrine or apocrine sweating was seen following the use of preparations containing aluminum salts.

8. Aluminum preparations have demonstrable deodorant activity. Daily applications of these substances will abolish or alter the usual axillary odor in many persons for 12 to 18 hours.

9. This deodorant activity in the case of aluminum chloride is based on antibacterial action and a chemical effect on the odoriferous products.