THE BACTERIA RESPONSIBLE FOR APOCRINE ODOR*

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Shelley, et al. (1), have shown that apocrine sweat is non-odorous when delivered to the surface but develops odor only when acted upon by the cutaneous microflora. The correctness of this view is demonstrated by the effectiveness of locally applied antibacterial agents in suppressing the pungent axillary odor (2, 3). In undertaking the present study we wished particularly to determine whether odor production was a unique function of a particular organism adapted to this region or whether different species had this capacity more or less nonspecifically.

METHODS AND RESULTS

The experimental subjects were all adult males, inmates of the Philadelphia County Prison. Cultures were taken by swabbing the axillae of individuals who had not used deodorants for some time previously. Apocrine sweat was obtained following local stimulation according to the methods of Shelley and Hurley (4); their technic was also followed in collecting the specimens under aseptic conditions (1). Negro subjects were used for the actual collection of apocrine sweat for in general this group appears to have a more abundant outpouring of apocrine sweat. We chose for study particularly those who, on the appropriate stimulation, produced visible droplets of apocrine sweat.

A. Bacterial flora of the axilla

The composition of the bacterial flora of the axilla is somewhat less constant than most other non-specialized regions of the glabrous skin. This is rather characteristic of intertriginous sites where increased moisture favors bacterial colonization and multiplication (5). Cultures of the axillae of 29 different individuals yielded the organisms shown in Table I. Only aerobic cultures were made because the bacterial decomposition of the apocrine sweat, which takes place after the extrusion of the sweat from the apo-pilosebaceous unit, must be an aerobic phenomenon. Coagulase negative micrococci and diphtheroids were found in roughly ninety and sixty per cent of the cases respectively. These are normal skin residents, while the other organisms, found in isolated cases only, are for the most part transients and, accordingly, are found inconstantly. It has been our general experience that gram negative rods are more commonly found in the axilla than on the normal glabrous skin, but they are not the dominant species in this region. The great preponderance of resident organisms implicates these

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as the chief source of the odor which is always present to some degree in everyone with functioning apocrine glands, though individuals differ markedly in this respect.

B. Inoculation experiments

Two methods of studying odor were employed. In the first, a capillary tube of apocrine sweat, collected under aseptic conditions, was added to 0.25 cc. of sterile saline in a microculture tube. In the second method, apocrine sweat, collected under the same conditions, was left in the capillary tubes, and small amounts of culture suspensions in saline were allowed to flow into the tubes. All tubes were incubated at 37°C. With the first method, it was possible to control the experiments throughout by removing small portions of the solution at intervals for culture and tests for odor. Before inoculation, the tubes were incubated for twenty-four hours or longer, following which those with odor, or found on culturing to be contaminated, were discarded. As a matter of fact, contaminated tubes were rarely found. After inoculation and incubation for twenty-four hours or longer, a final culture, at the termination of the experiment, showed whether the test organism had grown in pure culture. Indeed, apocrine sweat proved to be a good milieu for bacterial growth, doubtlessly because of its protein content, for it should be noted that no additional nutrient material was added to the tubes. It was generally difficult to detect the odor by smelling the outlet of the tube unless it was marked, but when the contents were inverted onto a piece of cotton, the odor was usually apparent. On the other hand, the capillary tubes which contained undiluted apocrine sweat yielded stronger odors and were more sensitive in this regard, but fewer manipulations were possible since the tubes had to be broken to detect the odor, terminating the test. We made no attempt to quantitate the strength of the odor, recording even a faint odor as a positive test. Much variation was evident in this respect but, in view of our limited objective, we did not feel it worth while to refine the test procedures further, though they are admittedly crude.

Using both methods, we have reconfirmed the fact that sterile apocrine sweat remains odorless, even upon incubation for several days. However, the introduction of the appropriate bacteria will produce the typical odor within twenty-four hours, and it does not appreciably increase after this time. Our results on odor
production with the inoculation technique are summarized in Table II. All of the organisms, with the exception of the beta-hemolytic streptococcus were recovered from the axilla. It is at once evident that a variety of species may produce odor; moreover, the odor produced by different species was identical. Many of the isolates of the resident organisms, mainly the diphtheroids and to a lesser extent the coagulase negative micrococci, generated odor. Perhaps strains of the same species differ in this respect as suggested by the apparent lack of this ability in some isolates, but we did not completely exclude the possibility that this was due to technical limitations of the method. Presumably, the apocrine sweat from different glands of the same and different individuals is equally suitable for odor production, for when a known odor producer, a coagulase positive micrococcus, was inoculated into twelve tubes prepared from three different individuals, odor was evident in all tubes. E. coli, Proteus vulgaris and Aerobacter aerogenes are other odor producing organisms, but our tests are obviously too limited to estimate whether strains of these species characteristically have this property. Such organisms are probably insignificant so far as odor is concerned for they are not generally present in great numbers in the normal axilla. We are inclined to think that odor producing organisms will be found among many species which we did not test and which do not occur in the axilla. Our single test, each with a Sarcina species and a beta hemolytic streptococcus, does not warrant the generalization that these species cannot produce odor.

The apocrine gland, unlike the eccrine gland, empties into the pilosebaceous apparatus, and samples collected at the surface may be contaminated with minute amounts of sebum. Therefore, we examined the possibility that sebum might be contributing to the odor. Sebum collected from another source (scalp hair) was added in appropriate dilutions to sterile saline, and the specimens were inoculated with a known odor producer. No odor resulted in any of these tubes. Viable organisms were recovered on the termination of the experiment ruling out a bacteriostatic effect of the sebum.

COMMENT

The factors which might account for the marked variation in the intensity of axillary odor among individuals are: 1) variation in the kinds and numbers
of organisms present, and 2) qualitative and quantitative differences in the apocrine sweat.

We have presented limited evidence against qualitative variations in the apocrine sweat since odor was readily produced by a single organism acting on sweat collected from different glands of the same and different individuals. That there are quantitative differences in the amount of apocrine sweat between individuals is obvious following appropriate stimulation, some persons consistently producing good droplets, while others produce almost imperceptible amounts. Whether there is a correlation between the intensity of the odor and the amount of sweat is not known at present; we are impressed that a prodigious odor can be generated in a droplet of apocrine sweat.

Differences in the odor-producing qualities of different organisms do not appear to be of decisive significance for organisms capable of generating strong odors can be isolated from any axilla. Still, our in vitro tests did show variations among isolates of the same species, and the possibility cannot be excluded. It is a reasonable conjecture that the quantity of organisms present could be a factor, stronger odors resulting when the bacterial population is large. It must be remembered that the apocrine sweat comprises only a tiny fraction of the fluid sweat of the axilla, the eccrine glands accounting for practically all of what is seen. In this respect eccrine sweat may be an influential factor, for moisture directly enhances the multiplication of organisms on the skin's surface.

It is obviously impossible to control apocrine odor by preventing apocrine sweating; there is no practical way to induce apocrine anhidrosis. For the reasons given, reduction in eccrine sweating could only indirectly influence the odor, and probably not appreciably in every case, for the axilla will tend to be moist even when eccrine sweating is minimal. In this connection, the benefit of proprieties containing aluminum salts should not be misinterpreted as being due to suppression of eccrine sweating for these agents are also antibacterial and when used daily in high concentrations may reduce the bacterial population greatly.

From the standpoint of choosing agents to suppress bacterial multiplication, the significant finding of the present study is the diversity of species which have the capacity to produce odor. While the dominant species are gram positive residents, wide spectrum antibiotics appear to have the greatest advantage since gram negative organisms may also produce odor. The reported clinical evaluations of antibiotic creams support this opinion (2, 3).

**SUMMARY**

A variety of bacterial species have been shown to be capable of producing the typical pungent odor in sterile apocrine sweat.

**REFERENCES**