Certain staphylococci can produce clinical manifestations in man and animals. However, there is only limited knowledge of the interrelationships between human and animal staphylococci. A review of known data is presented and the need for further investigation is stressed.

ANIMAL STAPHYLOCOCCAL INFECTIONS AND THEIR PUBLIC HEALTH SIGNIFICANCE

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INTEREST in staphylococcal disease as a community problem has increased during the past decade. One of the most important reasons for this is our inability to effectively combat the infections with the antibiotics presently at our disposal. Efforts are continually being made to find new antibiotics and chemotherapeutic drugs to treat the infections as they occur; however, the ultimate goal should be the prevention of staphylococcal infections. To accomplish this, it will be necessary to know more about the epidemiology of the disease and to clarify the environmental circumstances that permit the organisms to exist and spread to and among human beings.

Staphylococci are ubiquitous organisms which are found most frequently within tissues, on skin surfaces, or in foods contaminated by exposure to infected human beings or animals. Knowledge concerning the relationship of human and animal pathogenic staphylococci is not extensive; consequently, the public health importance of animal staphylococci is difficult to ascertain. This paper reviews and brings together current information on type and distribution of animal infections and presents evidence that may relate human staphylococcal infections or intoxications to animal origin.

The importance of staphylococcal infection in man needs no further elaboration, for it remains one of the more serious bacterial diseases confronting medical science. It has held its ground and seemingly increased in incidence during the period when many life-saving antibiotic drugs have come into use. Certainly the antibiotic-resistant strains have increased as trouble makers. The significance of this disease requires that we look at the whole animal environment, not only for the purpose of deducing the relationships regarding the transmissibility of the agent between lower animals and man but, also, for information that will help us understand the host-parasite relationships and the many ramifications of immunity, resistance, and therapy. Obvious questions that need clarification are whether there are true animal strains which have the ability to produce disease in man or whether animals, when infected by human beings, serve merely as a medium to support the organisms while they await the opportunity to return to another human host.

Staphylococci are not uncommon in animals, but they are found more frequently in man. When they are found in diseased animals they are often associated with other microorganisms, and their specific role is not defined as clearly as it is in man.

Staphylococci in Animals

Staphylococci are associated with skin diseases in horses, but their classification into distinct entities in this species is difficult.[1] The organisms are found in acne and furunculosis which occur on areas of the skin frequently irritated or damaged by the friction of a saddle or harness. Moisture predisposes to these diseases which are more common in hot moist climates.

Botryomycosis is a granulomatous staphylococcal disease which occurs in horses. When named, it was thought to be caused by a fungus. It usually begins in lacerations and abrasions on the skin or, following castration of the animal, in the stump of the spermatic cord where small pockets of pus are found and there is a mass of newly formed tissues. Small granules resembling those of actinomycosis are found in the pus; and, when the granules are crushed, they yield masses of staphylococci which are imbedded in a capsular material.[2] Sometimes the disease generalizes, and, when this occurs, it is usually fatal.

Often staphylococci are found in suppurative processes in cattle and frequently, but not always, are associated with other organisms. In poorly kept animals, acne occurs usually on the lower part of the tail.

Furuncles often are seen on the udder along the hair line at the base of the teats or between them. Zinn, Anderson, and Skaggs[3] studied furunculosis in a dairy herd on a farm where the condition had persisted for two and one-half years. During this period, one entire herd of dairy cattle had been replaced by a second herd; furunculosis became persistent in the new herd three months after the animals had arrived on the farm. Coagulase-positive, phage-type 80/81 staphylococci were isolated repeatedly from the lesions and milk of these cows.

Mastitis associated with staphylococci is quite common in dairy herds. It is a stubborn form of the disease and is a problem in countries that have the most advanced technical dairying practices as well as in countries with the most primitive dairying practices.[4] Figures on the incidence of mastitis in the United States differ, but they seem to indicate that about 70 per cent of all dairy herds have staphylococcal mastitis. In some problem herds, it has been impossible to eliminate. The disease will vary greatly in severity from mild and chronic to severe and highly acute; in the latter form, gangrene of the udder may end in death of the cow.

Botryomycosis of the bovine udder is caused occasionally by staphylococci. This is a granulomatous lesion which may be mistaken for actinomycosis.

Three conditions in swine—impetigo, acne, and botryomycosis—are associated with staphylococcal infection. These are not common diseases in swine, and botryomycosis may be mistaken for actinomycosis of the mammary glands.[2]

Staphylococcal infections in sheep are found on the lips and the skin of the neck where the wool is replaced with ulcerations. In mastitis, or blue bag, staphylococci are found, usually in company with other organisms, and are associated with some mechanical injury. Debility and death in lambs resulting from staphylococcal infections in tick bite wounds have been described. Botryomycosis in sheep has been described but is rare.[1]

Avian staphylococcosis has been reported in turkeys, geese, ducks, and chickens.[5] The infection causes an acute septicemia or chronic arthritis. In the acute disease, the bird may die within two to three days; the chronic disease may last several weeks with the joints showing signs of inflammation. Omphalitis, or infections of the umbilicus, in turkey poults has been attributed to staphylococci.[6]

The most common site of staphylococcal infections in dogs is on the skin where infection follows some form of injury.[7] Infection, with pustules, may follow advanced cases of demodetic mange. The ear canal and inside of the ear flap is a frequent location of staphylococci in the dog. Acne and furunculosis occurs around the muzzle, and chailitis is usually the result of the infection's being transferred from some other part of the body to the lips. Staphylococci have been isolated from cases of bronchiectasis and enteritis. Also, the anal sacs may become infected.

Other animals, such as birds and cats which are in less frequent contact with man than the livestock and pets mentioned above, may also have staphylococcal infections. However, for the purpose of this paper, a discussion of the disease in these species is not necessary.

Perhaps of more epidemiological importance than active animal staphylococcal disease are the asymptomatic animal carriers; certainly these animals provide a reservoir which may not be an obvious source of pathogens. Several surveys have pointed up the types of staphylococci found in dogs, cattle, swine, horses, chickens, and cats.[8-11,21]

In an examination of 32 dogs seen at the Kansas State Veterinary Hospital, 93 per cent yielded staphylococci and 88 per cent were found to be harboring coagulase-positive staphylococci. Swabs were taken from several parts of the body and the frequency of coagulase-positive isolations from the various sites were as follows: throat, 39 per cent; nose, 70 per cent; skin of the axilla, 21 per cent; and skin of the groin, 39 per cent.[8] The health status of these dogs was not mentioned nor were they classified as outpatients or kennel patients. In England, 61 dogs, either hospitalized or brought to a veterinary school clinic for treatment, were examined for the presence of staphylococci in the nares.[9] Coagulase-positive staphylococci were found in 11.5 per cent of these dogs. In the same study, no staphylococci were isolated from the nares of 106 cattle, some of which were affected with staphylococcal mastitis. Coagulase-positive staphylococci were isolated from the nose of 1 out of 17 horses, but none were found in 15 rabbits and 50 sheep. Sixty per cent of 60 guinea pigs and 52.9 per cent of 17 monkeys yielded the organisms from the nares. Smith and Crabb[10] examined the nares and skin of 320 pigs in 32 herds and 200 chickens in 20 flocks; and, by picking multiple colonies, they made 869 isolations of coagulase-positive staphylococci from the swine and 738 isolations from the chickens.

Surveys in the veterinary college at Ft. Collins, Colo., revealed that 89 per cent of all animals examined (canine, feline, bovine, and equine) were harboring coagulase-positive staphylococci. A higher percentage of coagulase-positive organisms were found in the canines and felines than in the other species; also, a higher proportion of the nasal isolates were coagulase-positive than were the skin isolates.[11]

Characteristics of Staphylococci

Evaluation of the animal reservoirs of staphylococci as a source of human infection would be enhanced if there were some practical method of identifying the organisms as to human or animal types or strains. Unfortunately, there are no such reliable methods. Some minor differences have been noted in isolates from man and those from animals, but these differences are not sufficient to separate staphylococci into animal and human strains.

It is generally accepted that coagulation of plasma by staphylococci is a property of pathogenic strains only. While some of the other tests are of considerable value, the only criterion of pathogenicity is the production of coagulase by staphylococci isolated from man or animals. The appearance of beta hemolysis on sheep blood agar, liquefaction of coagulated serum, production of fibrinolysin, hematoxin, dermotoxin, and lethaltoxin are properties exhibited only by pathogenic strains, but they are not exhibited by all pathogenic strains. Consequently, the possession of any of these properties may be accepted as evidence of a pathogenic staphylococcus, although its absence is without significance.[12]

One author[1] has stated that the strains of staphylococci obtained from animals differ in three known respects from those obtained from man: (a) Most animal pathogenic strains produce beta hemolysin, while alpha hemolysin is a characteristic of human strains; (b) animal strains are predominantly of the 42D phage-type, which is uncommon in man; and (c) most pathogenic animal strains produce no opacity in egg-yolk broth, whereas, most human strains are egg-yolk-positive, producing opacity in this medium.

These criteria must be considered very carefully when they are used in an attempt to separate staphylococcal isolates. In a study of 110 pathogenic staphylococci taken from active lesions of dogs, cattle, sheep, poultry, goats, and horses, Smith[12] found seven strains which were nonhemolytic. He expressed the opinion that neither the presence of hemolysis on blood agar, other than the darkened beta type, nor the absence of hemolysis on this medium is of value in the classification of animal staphylococci and that the expression "hemolytic staphylococcus" might as well be dropped as it is of no significance. In his study, 68 per cent of the pathogenic strains showed alpha-beta type hemolysis, while only 23.6 per cent produced beta type. Two strains from goats produced only alpha hemolysis.

Edwards and Rippon[13] examined the characteristics of 395 strains of staphylococci isolated from milk or abnormal secretion from three herds in isolation units and from 19 other herds. They tested 64 strains for hemolysin production and found that 54 strains produced alpha, beta, and gamma hemolysins, and 10 strains produced only the alpha type. They also found a variation in the widths of the hemolytic zones, and this width variation was not typical of the hemolysin type.

In a recent study by Galton, et al.,[14] of staphylococci in milk from 262 cows during a six-month period, a total of 801 isolates were tested with phage; 551 (68 per cent) were typable at 10X the routine test dilution (RTD). Phage-type 80/81 was recovered from 15 per cent of the cows, and phage-type 77/44A (or a similar pattern) was recovered from 48 per cent. The remaining typable strains were a variety of other patterns. Type 42D was not found alone and was rarely detected in combination with other phages.[14] While studying staphylococcal infections in meat handlers (slaughterers, butchers, and so forth), Ravenholt[15] typed 25 coagulase-positive staphylococci isolated from lesions in cattle, swine, poultry, and sheep. He found seven phage-types in the 11 isolates which were typable, but none of these were the 42D type. Seto and Wilson[16] in typing 102 cultures from bovine milk in Wisconsin found phage-type 44A in 36 per cent.

Alder, Gillespie, and Herdan[17] tested groups of coagulase-positive staphylococci, from animal and human sources, for their ability to produce opacity in egg-yolk broth. In 118 strains from animals, they found 15.3 per cent to be EY-positive; in 560 strains from human beings, they found 15.9 per cent to be EY-negative. There was a significant difference in the reaction between the human isolates from abscesses and those from nasal swabs. In the latter group, 23 per cent were EY-negative.

The Effect of Antibiotics on Staphylococci

The nature of staphylococcal resistance to antibiotics is a major clinical problem in staphylococcal infections. Likewise, the resistance of staphylococci to the antibiotics increases the importance of these organisms as a public health problem. Antibiotic resistant strains of staphylococci are widely distributed in animals and their prevalence probably is associated with the use of antibiotics either as a feed supplement or in the treatment of clinical illnesses.

Comparative studies of the staphylococcal flora were made by Smith and Crabb[10] on swine and chickens. They examined for sensitivity 498 strains from 160 pigs in 16 tetracycline-fed herds and 380 strains from the same number of pigs and herds which had not been fed antibiotics; 92.6 per cent of the 498 strains isolated from the nose and skin of the tetracycline-fed pigs were resistant to this drug compared to 4.5 per cent of 380 strains in the control group. No penicillin-resistant strains were found in either group.

One hundred and nine strains were isolated from four herds which were being fed rations containing penicillin. Penicillin-resistant Staphylococcus aureus was present in all four herds. The organisms were found in 37 per cent of the 38 pigs examined. Sensistive strains were found in only 50 per cent of the pigs, and no staphylococci were found in 13 per cent. Of the 109 strains examined, 30.3 per cent were penicillin-resistant.

Studies were also made on 10 flocks of chickens being fed a ration containing tetracycline and penicillin and 10 flocks receiving no antibiotics in the feed. Sensitivity tests on 492 strains from antibiotic-fed chickens showed 69.9 per cent to be resistant in contrast to only 6.8 per cent from the controls.

Rajulu[8] tested the antibiotic sensitivity of 19 typable strains of staphylococci isolated from 32 dogs. Fourteen of these strains were resistant to several of the more commonly used drugs. Although there was variable sensitivity to bacitracin, furadantin, chloromycetin, and kanamycin, none showed real resistance to these drugs. Mann[18] made one isolation of coagulase-positive staphylococci from each of 32 dogs and found the following antibiotic sensitivity pattern: 17 strains were susceptible to streptomycin, 19 to tetracycline, 21 to oxytetracycline, 25 to penicillin, 29 to bacitracin, 30 to oleandomycin-tetracycline, and 31 to a combination of penicillin and streptomycin.

In the six-month study of the staphylococcal flora in the milk of a large dairy herd by Galton, et al.,[14] coagulase-positive staphylococci were found in the milk of 55 to 100 per cent of the cows that were treated for mastitis during this period. Seventy-two per cent of the cultures from the 62 treated animals were resistant to one or more of five commonly used antibiotics. The greatest number of organisms were resistant to penicillin and streptomycin, which were two of the drugs used in treatment. There was an actual increase in the strains resistant to dihydrostreptomycin, with the percentage of resistant strains increasing from about 30 per cent to 70 per cent during the third month and remaining high through the sixth month.

Interchange of Staphylococci Between Animals and Human Beings

Rather extensive research has yielded a seemingly small number of accounts of proved transmission of pathogenic staphylococci from animals to human beings or from human beings to animals. Perhaps proof of such transfers is so difficult that many have occurred without the source being identified. However, information at hand strongly supports the assumption that pathogenic staphylococci are passed between animals and human beings.

While studying the phage patterns of staphylococci of animal origin, Smith[19] used one strain of staphylococci which had been incriminated in a food-poisoning outbreak following consumption of ice cream, one strain which had been isolated from a case of human mastitis, and one strain from a case of bovine mastitis. These strains were shown by cross-absorption tests to be antigenically identical. All were susceptible to phage 42D. Each of these strains was injected into separate quarters of the udder of a cow one month after her first calving. Prior to injection, her milk was examined for staphylococci with negative results for two weeks. Apart from pyrexia, slight swelling of the udder, and clots in the milk from the three infected quarters, no other signs of infection were noted. These subsided completely in 48 hours, but staphylococci were regularly recovered from each infected quarter for six weeks after which the experiment was terminated. Smith concluded that his findings supported the view that some strains of staphylococci may be capable of infecting man as well as cows and that milk may be the source of human staphylococcal infection.

Wallace and others[20] in Hawaii found phage-type 80/81 antibiotic-resistant staphylococci in a dairy herd which had previously undergone intramammary infusions with a wide variety of antibiotics. Staphylococci of the same phage-type and antibiotic resistance pattern were isolated from one of the dairy workers who first noticed lesions three months after he began work with this herd. The investigators recognized the possibility that the human infection was acquired from the cows.

Conditions in the Kentucky dairy herds, reported by Zinn, et al.,[3] indicated transmission of phage-type 80/81 staphylococci from a human being to cattle. At the time the first herd developed furunculosis, one of the attendants was undergoing treatment for sinusitis and otitis externa in a local clinic where hospital-acquired staphylococcal infection was known to be a problem in the community. No phage-typing was done to identify the problem specifically. Three months after disposal of this herd, a new barn was built, and a new group of cattle brought in. All were free of furunculosis or other signs of infections. After three months contact with personnel on the farm, some cows began to develop furuncles on their udders.

In studying antibiotic-resistant staphylococci in swine and chickens, Smith[10] found, by application of phage-typing and other tests, that the antibiotic-resistant staphylococci isolated from animal attendants were usually identical with those isolated from their animals. It was apparent to the investigators that veterinarians should anticipate that antibiotic resistance will be more likely to complicate therapy of bacterial diseases of animals on farms where diets supplemented by antibiotics are used than on others. Physicians, also, should be aware of the implications when treating persons who care for antibiotic-fed animals.

The possible and probable transmission of S. aureus from man to turkey poults was investigated by Williams and Daines.[6] Although they were not entirely satisfied with the validity of the results, they found that sore throats and impetigo were endemic among workmen in a hatchery, that there was an unusually high death rate from omphalitis among poults within 72 hours after hatching, and that a turkey herder in a remote rarely visited area developed staphylococcal impetigo shortly after receiving a shipment of poults from the hatchery. When changes were made to minimize contact of men with incubating eggs and to keep those with lesions out of the incubators, death rates of the newly hatched poults dropped appreciably; and no losses due to omphalitis were seen.

Senior veterinary students and faculty members in close clinical contact with animals have been shown to have a much higher carrier rate and incidence of staphylococcal infection than other veterinary students. Pagano and others[21] isolated antibiotic-resistant staphylococci of phage-type 80/81 from the external nares of asymptomatic domestic animals and from human contacts during an epidemic of staphylococcal disease in veterinary students. Some of the infections could be attributed to person-to-person contact, and spread could also be explained by transmission of staphylococci from animals.

Staphylococcal Enterotoxin Food Poisoning

No doubt staphylococci are a very important cause of food-borne disease. Indeed, 40 per cent of the outbreaks reported to the National Office of Vital Statistics in the United States are caused by staphylococcal enterotoxin.[22] In the epidemiology of staphylococcal food poisoning, the two important sources of contamination are (a) the human carrier or individual with lesions, and (b) food products of animal origin, such as raw milk or raw milk products from infected animals. Some strains of staphylococci produce enterotoxin; and, when contaminated foods are allowed to stand unrefrigerated, the organisms multiply, producing powerful enterotoxin which is comparatively heat-resistant. Many types of food have been involved in staphylococcal outbreaks; some examples are ham, prepared ready-to-eat meats, dried beef, sausage, milk, cheese, prepared salads, and particularly cream- or custard-filled bakery products.[22]

Two rather large outbreaks of staphylococcal intoxication involving cheese occurred in 1958. In one outbreak reported by Allen and Stovall,[23] more than 60 cases occurred in Indiana and Michigan. Cheese of the Colby type produced from raw milk in one cheese factory in Wisconsin was involved. Coagulase-positive staphylococci of the same phage-type were isolated from the cheese, the plant, and milk from herds supplying the factory.

The other outbreak, reported by Hendricks, et al.,[24] and Hausler, et al.,[25] occurred among 200 persons in a state institution. All ill persons had eaten cheddar cheese made in a local factory from raw milk. Coagulase-positive staphylococci were isolated from cheese and from two of eight herds supplying milk to the factory.

Eight explosive outbreaks of food poisoning involving nearly 1,200 persons were investigated by Anderson and Stone.[26] The illnesses were traced to consumption of foods made from spray dried milk powder which contained a high content of S. aureus of a phage pattern often associated with food poisoning. In the seven outbreaks where phage-typing was done, only type 42E/53W was identified; it was found in all seven. In one of the outbreaks, a cook was found to have a septic finger from which a 42E/53W staphylococcus was isolated. The isolation of staphylococci of similar phage pattern from freshly opened tins of dried milk suggested that the finger might have become infected from the milk powder.

Due to selective bacteriostatic activity of curing salts, there is a quite different bacterial flora in cured and fresh meats. According to Lechowick, Evans, and Niven,[27] staphylococci, including the food-poisoning variety, are not inhibited by palatable salt concentrations. They inoculated ground muscle and hams, added curing salts, and smoked the hams. They found that staphylococci, when incubated at 86° F, grew vigorously in ground muscle containing any combination of curing ingredients that is permissible and palatable. Staphylococci died rather rapidly in curing pickle, but were protected by added meat juices. Most, if not all, staphylococci injected into hams with the pump pickle survived the pickling process until the hams were heated to an internal temperature of 137° F during smoking. On the other hand, heating in the smoke house at 120° F for 48 hours slowly killed the staphylococci in the interior of the ham, but the staphylococci on the surface occasionally multiplied.

Discussion

It is well established that certain staphylococci are capable of producing a variety of clinical manifestations in both man and animals, but, as yet, there are no entirely clear-cut criteria to determine whether host-adapted strains of human or animal origin exist. Reference is made frequently to such terms as "bovine staphylococci," "human strains," and "animal types." It is true that phage-type 42D is found frequently in the mastitic bovine udder and in normal udders. However, this type has been incriminated in outbreaks of food poisoning in man and in human mastitis. Similarly, type 80/81 is considered usually as the most common type in human infections. Ravenholt[15] suggests, however, that this type may possess unique mammopathic qualities because of its apparent ability to cause bovine and human mastitis.

During a six-month survey of the staphylococcal flora in the milk from a dairy herd of 262 cows, phage-type 80/81 was isolated from 40 (15 per cent). A total of 151 samples were obtained from these animals and type 80/81 was isolated from 58 (38 per cent). Thus, this type was obtained on repeated sampling from nine animals for periods of two to five months. However, in spite of the virulent nature of type 80/81, only eight of these cows were treated during the same month that this type was isolated, and visible evidence of mastitis was observed in eight. In addition, 68 (73 per cent) of the remaining 93 samples from these cows contained coagulase-positive staphylococci that were other phage-types or nonreactive, and only seven had visible evidence of mastitis the month the positive sample was obtained.

Even though coagulase-positive staphylococci were isolated from 713 (71 per cent) of 1,010 milk samples during the study period, the prevalence of clinical mastitis was not considered above normal. In previous years, a slight increase in mastitis had been observed during the winter. During the survey, this increase was again apparent during January, February, and March and was accompanied by an increase in coagulase-positive staphylococcal isolations.

Summary

Are the coagulase-positive staphylococci that occur in both man and animals put there by human beings, or do they gain access from other sources and await the opportunity to invade tissue damaged by trauma or other infectious agents?

Although other characteristics such as the type of hematoxin production and

precipitation of egg-yolk medium usually differ between human and animal pathogenic strains, similarities do occur; and we must consider that evidence on host-adaption is not conclusive. At present, coagulase production is the only single laboratory criteria upon which staphylococcal pathogenicity in man and animals may be based.

It is not surprising to find that many antibiotic-resistant strains of staphylococci occur in both human and animal infections and in carriers. The widespread use of antibiotics in therapy is perhaps exceeded only by the now common practice of adding small amounts of these drugs to animal feed. This usage enhances the development of resistant strains where no disease is occurring and thus provides an unsuspected source of human and animal infections with types most difficult to handle clinically.

Our knowledge concerning the interrelationships of staphylococci found in man and animals is somewhat limited and differences in available data make it difficult to evaluate. For this reason, only continued concerted effort by interested investigators will provide the answers to the many puzzling questions involved. When reported, the answers to these questions may lead to firm conclusions about the amount of interchange of staphylococcal infections and enterotoxin between animals and man. Only then can we determine the hazard to human beings of staphylococci in their animals or the reverse. Meanwhile, our current knowledge of the characteristics of staphylococci is of some value in epidemiological investigations when used with all other available information about the source of infections.

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A Meeting of Minds in Health and Welfare

What happens when a public welfare and a public health department are joined in a mutual administrative effort? In providing medical care for the indigent, one state, Kentucky, encountered numerous hurdles which "repeatedly and unnecessarily obstructed and delayed progression toward program objectives." This was reported by Dr. William H. McBeath, acting director of Kentucky's Health Department's Division of Medical Care at the Annual National Conference of the American Society for Public Administration held in April at Detroit. In Dr. McBeath's view, the joint welfare-health administrative difficulties derived from fundamental differences in viewpoint, orientation, and philosophy between the two fields.

Some of these differences are clearly shown in: their approach to receiver of service—welfare serves the individual, public health, the community; the nature of their service—the traditional public health focus is on prevention, welfare service is remedial, after dependency has been established; the form of their service—welfare furnishes financial support, public health gives direct health services; their control of service—welfare maintains quantity control with fiscal vigilance, public health attends to quality control by emphasizing standards of adequacy; their administrative pattern—welfare regulations promote greater stability and uniformity of program, health statutes permit more freedom in experimentation and research.

"In making administrative decisions in a medical care program," Dr. McBeath said, "the public welfare worker tends to think primarily in quantitative terms of correcting a manifest deficiency in individuals. In making the same decisions, the public health worker tends to think primarily in terms of avoiding deficiencies which threaten the community's well-being." Hope for success in aligning public welfare and public health "will be greatest," he added, "when we can direct our attention. . . to our common goal of solving the basic problem." In Kentucky, he said, most areas of the program are better administered "by a compromise bringing together the best of each viewpoint."