WHAT THE SANITARIAN SHOULD KNOW ABOUT STAPHYLOCOCCI AND SALMONELLAE IN NON-DAIRY PRODUCTS. I. STAPHYLOCOCCI[1]

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ABSTRACT

After a review of the nature of staphylococci and staphylococcal enterotoxins including data on survival of these organisms under both natural and food processing conditions, the epidemiology of staphylococcal intoxications is discussed. The human nose is the main reservoir of *Staphylococcus aureus*. A number of circumstances must be fulfilled for foodborne staphylococcal intoxications to occur; these include: a reservoir for the infectious agent, a mode of dissemination of the organism, contamination of a food capable of supporting bacterial growth, enough time at a temperature which permits bacterial multiplication, and ingestion of sufficient amounts of staphylococcal enterotoxin by susceptible hosts. Control measures must be based on these circumstances. Principles of control, therefore, include limitation of contamination, inhibition of growth, and destruction of the organism. Control of staphylococcal intoxication must be emphasized at places foods are prepared (food processing plants, food-service establishments, and homes).

NATURE OF STAPHYLOCOCCI

Staphylococci are spherical cells occurring singly, in pairs, in tetrads, and in irregular clusters resembling bunches of grapes. These organisms are asporogenous, nonmotile, and Gram-positive. Colonies may appear white, yellow, or orange as a result of water-insoluble pigments. The species of concern to sanitarians is *Staphylococcus aureus*, pigmented and nonpigmented varieties. This species, distinguishable from the other species in the genus, S. *epidermidis*, by its ability to ferment mannitol and to coagulate citrated rabbit or human plasma (coagulase-positive), now includes organisms formerly known as *Staphylococcus pyogenes*, *Staphylococcus albus*, *Micrococcus pyogenes* var. *aureus* and var. *albus*, and *Staphylococcus citreus*, as well as S. *aureus*. Coagulase-Positive staphylococci produce a variety of toxins and enzymes, including a lethal toxin, dermonecrotoxin, hemotoxins, leucocidins, fibrinolysin, hyaluronidase, deoxyribonuclease, penicillinase, lipase, and enterotoxin.

S. *aureus* requires several amino acids, thiamin, and nicotinic acid, for growth. It is facultative and hence grows very well under anaerobic conditions if fermentable carbohydrates are present, but it grows even better under aerobic conditions. These organisms tolerate sodium chloride well, in fact, they grow vigorously in a 10% salt solution that would inhibit the growth of most bacteria. Under aerobic conditions S. *aureus* has a water activity (a_w) of 0.86 and can grow in solutions containing salt in concentrations as high as 22% (w/v); the rate of growth is substantially reduced when the water activity is less than 0.94 (10% salt solution), *(23, 35)*. Staphylococci also withstand drying at room temperatures and are able to survive in dust.

McDade and Hall *(25, 26)* examined the effects of temperature and relative humidity (RH) on the survival of staphylococci on various surfaces. The organisms were observed for a period of 7 days on glass, tile (asphalt, ceramic, rubber), silk sutures, and polished stainless steel. These contaminated surfaces were held at temperatures between 39.2 F and 98.6 F and at various relative humidities. In general, die-off was progressive as the temperature increased and became more pronounced at 77 F, and die-off increased at humidities toward the midrange (53-59% RH), except at 39.2-42.8 F. Minimal die-off and even some growth occurred at 95 to 98% RH. The best survival was at 11 and 33% RH.

Food poisoning strains of S. *aureus* grow well in custard at temperatures ranging from 44-114 F. Slightly less active multiplication occurred in chicken à la king and in ham salad *(1)*. S. *aureus* strains have also been reported to grow in chicken gravy at 50 F but not at 41 F *(16)*.

The natural flora in chicken, turkey and beef pies, and in raw crab meat may inhibit the growth of S. *aureus*. At 68 and 99 F staphylococci multiply but are outnumbered by competitors. Among the genera or species that inhibit or outgrow S. *aureus* are *Streptococcus*, *Lactobacillus*, *Micrococcus aurantiacus*, *Achromobacter*, *Escherichia coli*, *Bacillus cereus*, *Enterobacter*, *Pseudomonas*, and *Serratia (10, 15, 31,*

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33, 36). The bacterial flora, staphylococcus population, substrate, previous processing operations, and temperature of incubation influence the outcome.

McCroan et al. *(24)* found only a few coagulase-positive staphylococci in commercially prepared, wrapped sandwiches. No significant increase in the number of these organisms was observed after a period of room temperature storage. In the laboratory, these investigators inoculated sandwiches with staphylococci, and increased levels of the organism were observed after 48 hr incubation at room temperature. Particularly high levels of growth were found when the inoculation was made between the ham and cheese as opposed to on the ham which was placed next to a mayonnaise spread.

Crisley et al. *(7)* and McKinley and Clarke *(27)* found that at room temperature synthetic cream fillings support growth of *S. aureus*. When only water was added to the filling, staphylococci did not increase in numbers, but they remained viable. At the interface between pie crust and cream filling, staphylococci grew profusely. When other nutrients were added, the fillings readily supported staphylococcal growth. In England, Hobbs and Smith *(19)* studied synthetic cream and found that fillings limited to cooking fat, emulsifying agents, sugar, and salt (without protein) did not support the growth of coagulase-positive staphylococci, *Salmonella paratyphi* B, or *E. coli*. These organisms tended to die out in a few days. Synthetic cream with butter or margarine (without milk, eggs, or sugar) supported the growth of *E. coli* and *S. paratyphi* B, although much better growth was observed after milk, eggs, or sugar were added.

Some strains of staphylococci produce enterotoxin, a substance capable of irritating the gastrointestinal tract. Enterotoxin, an exotoxin, is a protein formed at the surface of cells. Crude enterotoxin is thermostable and can be boiled for 30 min without destroying its ability to cause vomiting, but purified toxins have not shown as much heat resistance. At least four antigenically distinct types of enterotoxin are known. Their classification (A, B, C_1, C_2, D) is based on their reactions with specific antibodies *(5)*. Type A causes most outbreaks of food poisoning. Type B is rarely involved in food poisoning but causes pseudomembranous enterocolitis. Types C and D have been defined only recently, and there is little information on their relative importance, although Type C has caused food poisoning on several occasions. Type A has been purified and described quite recently. Chu et al. *(8)* compare its properties to those of the previously purified Type B.

EPIDEMIOLOGY

A person who has eaten food contaminated with sufficient quantities of enterotoxin will usually manifest symptoms in 2 to 4 hr—although, in some instances, as little as 0.5 hr and as many as 7 hr may elapse. The incubation period depends on the amount of toxin consumed and the susceptibility of the host. Onset of illness is rapid. Salivation is followed by nausea, vomiting, retching, abdominal cramps and, frequently diarrhea. Prostration, cramps, headache, sweating, and dehydration accompany severe attacks; blood and mucus are sometimes seen in the stools and vomitus, and there may be shock (subnormal temperature and blood pressure). Reports on temperature are inconsistent, but subnormal temperatures are frequently noted. Dolman *(11)* gives a classic description of the intoxication.

The severity of staphylococcal intoxication has a good inverse correlation with the incubation period and a direct correlation with the dose. Duration of illness is usually 1-3 days. Mortality is low, but there are some fatal cases on record *(13, 28)*.

Susceptibility to staphylococcal enterotoxin varies. Ingestion of as little as 0.5 ml of crude filtrate produced vomiting in human volunteers, but as much as 13 ml of the same filtrate produced no reaction in another volunteer *(9)*. Several outbreaks are known in which all persons at risk became ill. In epidemics it is difficult to determine relative susceptibility because patients eat varying amounts of the contaminated food, and the distribution of toxin in the food may vary. Evaluation of incriminated foods usually discloses counts in excess of 500,000 staphylococci per g *(18)*. Counts, however, are not necessarily reliable measures of the toxicity of a food, because inadequate chilling between serving and sampling can permit further multiplication. At room or at warm holding temperatures, organisms initially present may multiply to large populations, or competitive organisms may have multiplied and restricted the growth of staphylococci—even to the point where die-off occurred. Enterotoxin can maintain its potency in a time-temperature situation that will destroy staphylococci; thus, foods that have a history of being cooked or heat processed might have low or even negative staphylococcal counts but contain enterotoxin.

According to current epidemiological information, the main reservoir of *S. aureus* is man. The principal site of multiplication is the nose. Colonization of *S. aureus* in the nose begins in infancy, and within the first 7-10 days of life as many as 90% of newborn children become nasal carriers. This rate drops to about 20% during the first 2 years of life. At 4-6 years of age the rate approaches the adult rate of

30-50% *(39)*. Hospitalized adults have a higher rate ranging up to 60% *(30)*.

There are 3 kinds of nasal carriers; those that yield the same phage type of S. *aureus* at all examinations for months and years; those that rarely harbor the organism; and the intermittent carrier, who harbors the organism for a few weeks, becomes free of it, and once again becomes a carrier—but usually of a different phage type. Some people may harbor more than one phage type. Staphylococci are more common in the nasopharynx than in the pharynx; they are often found in the nasal accessory sinuses and are frequently associated with sinusitis. Surveys have shown that the throat is positive about 4-60% of the time *(39)*. Pathological conditions in the throat are often associated with coagulase-positive staphylococci *(4)*; the same organisms are abundant in post-nasal drips associated with colds.

The incidence of S. *aureus* on human skin is estimated to be between 5 and 40%. Kallander *(21)* found these organisms on the skin of 30% of food handlers. According to Williams *(38)*, the self-sterilizing capacity (as a result of the acid reaction, the action of sweat, and drying) imputed to human skin does not seem to inhibit the growth of staphylococci. Sweat was reported to be a good medium for growth of staphylococci *(37)*. Coagulase-positive staphylococci found on hands may be of two categories: those that are superficial transients being repeatedly replenished from the nose or other sites, and those that are resident flora and multiply in the depths of the skin. Sweating can bring the entrenched resident flora to the surface.

S. *aureus* is a usual constituent of human fecal flora. In one study, this organism appeared in the stools of 73% of children less than 1 year of age and in the stools of 18% of adults who were examined *(3)*.

Staphylococci are widely distributed in the air. Incidence of the organism is at its highest in bedrooms and Turkish baths, lowest in open air, and increases with activity *(13)*. One study *(12)* reported that 2 staphylococcus cells were found per ft^3 of still air in an empty room. The presence of a person in the room increased the count to 9 cells per ft^3. After some slight activity, on the part of the occupant, the count went to 129, and after vigorous activity, to 837. Dressing and undressing increased the number to 1,672. Roundtree and Barbour *(34)* observed that sweeping and dusting increased S. *aureus* counts in air. Dust particles from clothing appear to be as important in contaminating air as droplet nuclei produced by sneezing. Handerchiefs and clothing harbor staphylococci for at least a month *(13)*.

S. *aureus* is often found in suppurative wounds. It may attack any tissue in the body; it is a common cause of pyemia. S. *aureus* often causes secondary infections. Burns, particularly if extensive, are easily invaded by staphylococci. Pimples, carbuncles, boils, sores, and superficial lesions frequently harbor staphylococci. Animals may be asymtomatic carriers. S. *aureus* is a frequent cause of mastitis in cows and ewes and is a cause of abscesses in fowls.

S. *aureus* passes from person to person and so remains endemic. Staphylococci can multiply freely in the saline nasal secretions. Droplets from the nose charge the skin, the clothing, and the air. Touching the nose and handling disposable tissues and handerchiefs that have absorbed nasal secretions transfers staphylococci to hands. Hands contaminate food during preparation. The flapping or shaking of clothing liberates dust particles laden with organisms; these particles together with droplet nuclei from coughing and sneezing account for the presence of staphylococci in the air. Aerial transmission leads to colonization of the nose, and the cycle that maintains endemicity is completed *(13)*. The ability of staphylococci to withstand drying aids in maintenance of this cycle.

For a staphylococcal intoxication to occur, *(a)* there must be a reservoir for the enterotoxigenic strain of S. *aureus* (the nose or hands of food handlers); *(b)* a mode of dissemination of the organism (handling of food); *(c)* contamination of a food that is capable of supporting the growth of the organism (food must not be too acid and must be relatively free of competing organisms); *(d)* a temperature level for a length of time sufficient to permit adequate multiplication of the organism and toxin production; and *(e)* consumption of a sufficient amount of enterotoxin by a susceptible person. Successful control measures must eliminate one or more of these factors. At present, little can be done about the first and last factors, and only limited control can be imposed on the second and third factors. The most practical control measures prevent growth of staphylococci by adequate chilling or hot holding of foods.

Any foods that are touched by hands are subject to contamination by infected persons. Foods most likely to be involved in staphylococcal intoxications are cooked products contaminated by handling and allowed to remain at room or inadequate refrigeration temperatures for periods of 8 hr or longer, usually overnight.

Hodge *(17)* reported that 99% of the staphylococcal outbreaks that he surveyed were caused by cooked, high protein foods. In such foods, staphylococci grow without check from their normal competition and may build up to extremely high numbers. Leftover foods were responsible for 94% of the outbreaks, and foods containing a mixture of two or more ingredients were responsible for 67% of staphylococcal outbreaks. High levels of salt and sugar in foods inhibit many organisms, but not staphylococci. In

the United States, foods incriminated in outbreaks of staphylococcal intoxication include ham and other meat products, poultry and poultry dressing, sauces and gravies, potato salad, custard-filled pastries, bread pudding, hollandaise sauce, cheese, and milk.

Most of the staphylococcal outbreaks reported in England and Wales are associated with processed meat handled after cooking *(32)*. Pressed jellied meat, frequently involved in outbreaks, is often cooked and then trimmed by hand while still warm, placed in a large mold with gravy or gelatin, and allowed to stand at room temperature for hours. Meat pies are often contaminated, after baking, during the addition of gelatin. Tongues are skinned by hand after boiling and often not reheated. Cooked meats such as ham and tongue are often contaminated during slicing or during the preparation of sandwiches. Jay *(20)* isolated coagulase-positive staphylococci from approximately 39% of market meat samples and in all of 11 chicken samples.

Canned foods have been associated with outbreaks reported in England and Wales. Most of these cans have become contaminated after they were opened, but sometimes the contamination entered through minute defects in the seam when the cans were handled while still wet after cooling or during labeling.

DETECTION OF STAPHYLOCOCCI AND THEIR TOXIN

Plating is generally used to determine the total number of staphylococci per g of foods. Tellurite, tellurite-polymyxin B, and staphylococcus 110 agars are frequently used to plate homogenates of foods and their dilutions *(6)*. Egg yolk is often added to each of these media because there is an excellent correlation between egg yolk reaction (a zone of opacity around each colony caused by a lipase) and production of coagulase. When small numbers of staphylococci are expected in foods, as when monitoring for quality control or detecting staphylococci in the environment of a food establishment, enrichment is useful. Cooked meat broth containing 10% sodium chloride is usually used. When using this procedure, the most probable number of organisms can be calculated.

There is no precise or practicable method of differentiating strains of S. *aureus* serologically, so bacteriophage typing is used. Because of the ubiquity of S. *aureus*, the finding of coagulase-positive staphylococci in food, and isolating them from the nares or on the skin of food handlers does not fix either source as the cause of an outbreak, even if these organisms are found in vomitus or stools of the victim. Phage types of strains from all 3 sources (carrier, food, and patient) must correlate to prove an association between carrier and an outbreak. In phage typing, a basic set of 21 phages is used. These phages are divided into 4 groups and 2 miscellaneous phages *(2)*. The organism being tested is often lysed by more than 1 phage from the same group and occasionally by phages from another group; thus, a phage pattern results. Most enteropathogenic strains are lysed by group III phages, although phages in groups II and IV also occasionally lyse enterotoxinogenic strains.

The essential requirement to incriminate a food is demonstration of enterotoxin in the food. The presence of enterotoxin in foods can be detected through the reactions of human volunteers who ingest the suspected food, by animal assays, and by serological precipitations. In human and animal testing the organism is grown in pure broth culture; the broth filtered to remove cells; and other toxins are deactivated by formaldehyde treatment, by boiling for 15-30 minutes, or with hemolysin antisera. Treated filtrates may then be injected into the veins of test animals (cats or monkeys) and the animals observed for emesis. Monkeys may be given 50 g of crude, treated filtrate through a stomach tube. Humans, cats, and monkeys can also be fed the suspected foods. Enterotoxigenic strains of staphylococci will cause vomiting. The expense and other difficulties associated with human volunteer and animal assays has led to the development of a laboratory method for detecting enterotoxin. Antiserum is made from purified enterotoxins, and a gel-diffusion procedure is used to detect the toxin.

In the single gel-diffusion test, a food extract is layered over an agar column containing antitoxin, and the enterotoxin diffuses through the agar and forms a band of precipitate. The band moves down the agar column at a rate corresponding to the concentration of the enterotoxin and the concentration of the antitoxin. The double gel-diffusion tube test has a neutral zone of agar between the enterotoxin and the antitoxin. As the 2 reagents diffuse through the neutral zone, they form a band of precipitate where the 2 substances meet in optimum proportion. Another method of performing the double gel-diffusion test uses a petri dish containing agar or a microscopic slide covered with a thin film of agar. Antitoxin is put in a central well in the agar and suspect toxin is added to peripheral wells. Both reagents diffuse through the agar and form lines of precipitation where they combine in optimal proportion.

CONTROL

The principles of preventing staphylococcal intoxication are: limitation of contamination, inhibition of growth, and destruction of organisms.

Limitation of contamination

For practical purposes, the main reservoir of staphylococci is man, and there is no practical way to remove S. *aureus* from its widespread and usually benign association with man. So, it is extremely difficult to prevent contamination of foods with staphylococci. Both nasal and skin reservoirs of S. *aureus* can be temporarily eliminated by topical application of antibiotics to the anterior nares; however, organisms of the original phage type often reappear in a few weeks after treatment is stopped. Antibiotic resistant strains may also develop as a result of such treatment.

Washing and desquamation of outer layers of skin are the main ways of removing staphylococci, but washing does not necessarily free hands from staphylococci. Soaps, in dilutions comparable to their concentration in lather, show little, if any, bactericidal action on S. *aureus (29)*. Their action, primarily, depends on mechanical removal of microorganisms from the washed surface by emulsification of the lipids on the skin. It is doubtful that germicidal soaps contact organisms long enough to have much effect *(13)*.

Contamination can be minimized by strict practices of personal hygiene and sanitary food preparation. Persons who have open lesions, recurrent boils, or sinus infections should not prepare foods. Hands, even when washed, should be deemed contaminated and should not be allowed to touch foods which are served without being cooked subsequently. Utensils that prevent hand contact with foods should be used wherever possible.

Day-by-day supervision of food operations is the key to preventing staphylococcal intoxication. The public health sanitarian, however, cannot provide this daily supervision; it is the responsibility of food service or food processing managers. These supervisors must know, and accept, the principles of sanitation before they will advocate and demand safe food preparation techniques and storage practices. Food workers at all levels must be trained, but before managers can train or supervise others to follow basic procedures of sanitation, they must thoroughly understand these procedures. It is axiomatic in industrial training that the training of managers should always precede the training of workers. Sanitarians should employ all means at their disposal (consultation, training courses, and seminars) to train food establishment managers.

Inhibition of growth

Contemporary practices of personal hygiene and environmental sanitation do not insure that the ubiquitous S. *aureus* will be kept out of foods. Therefore, foods must be treated as if they are contaminated, and precautions must be taken to keep pathogens from multiplying. Potentially hazardous foods, if stored for only a short time (3 days or less), should be kept at temperatures of 45 F or below; if stored for longer periods of time, lower temperatures should be used.

Storage temperature, considered alone, can be misleading. The rate of cooling a food (and hence the interval during which the food remains at temperatures suitable for the growth of staphylococci) is influenced by the characteristics of the food container. The larger the container—and consequently the greater the volume of its contents—the farther heat must travel from the center of the food to the wall where it is taken up by the cooling medium. The internal temperature of food cooled in shallow pans falls faster than that of foods cooled in large, deep containers. Cooling rate is also affected by the material of which the container is made. Good conductors of heat, such as stainless steel, cool faster than crockery, glass, or plastic. Movement of air around containers takes heat away faster than still air. Free air circulation around foods and forced air circulation speeds cooling.

Prompt cooling of leftover foods is essential for preventing staphylococcal outbreaks. Hodge *(17)* reported that during a 2-year period 79 of 83 staphylococcal outbreaks were attributed to foods that were inadequately cooled. Foods should never be permitted to cool to room temperature before being put in a refrigerator; they should be refrigerated while they are still hot (140 F). Techniques of rapid cooling, such as putting containers in freezer compartments, packing them in ice, immersing them in running water, or mechanical stirring of contents are of particular importance in insuring against staphylococcal growth.

The rate at which foods cool is influenced by their composition, state, geometry, bulk, and viscosity. Solid foods cool by conduction, which is slow cooling. Liquid foods cool by convection; convection currents are sluggish in highly viscous foods. Sandwiches, potato salad, stuffing, bread pudding, custards, croquettes, and meat loaf—all of which are potentially hazardous foods—cool slowly.

Processing should be carried out during the bacterial lag period (the period before rapid geometric growth); thus, foods should not be held between 45-145 F for more than 3 cumulative hr, including chilling time.

In reviewing the effect of foods as substrates for staphylococci, Longree *(22)* concluded that all foods containing much protein are potentially hazardous unless the pH of the mixture is less than 4.5. In most dishes this pH value is hard to attain, requiring a relatively large proportion of acid ingredients, and

that the ingredients be finely divided to allow penetration of acid.

Destruction of the organism

The effectiveness of thermal processing in controlling staphylococcal intoxications depends on when the process is applied and how the food is held after the process has been applied, as well as time and temperature employed. Heat must be applied to destroy contaminating organisms before they have an opportunity to produce enterotoxin. A temperature of 165 F is lethal to staphylococci, but enterotoxin remaining in food is not destroyed by conventional methods of cooking or heat processing of foods. In custard and chicken à la king, more than 10 million cells of food poisoning strains of S. *aureus* were reduced to nondetectable levels at temperatures of 140 F in 53 min and at 150 F in 6 min *(1)*.

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REPORT ON THE INTERSTATE MILK SHIPPERS CONFERENCE[1]

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Since 1950, when representatives of industry, state departments of health, and state departments of agriculture from 26 states attended the first meeting of the National Conference on Interstate Milk Shipments, this Conference has grown in stature and in attendance with representatives from all 48 contiguous states. The program has grown and is now of considerable significance both to health authorities and to the dairy industry. Therefore, it is appropriate and important at this time to review the objectives and purposes of the National Conference, the accomplishments, and more specifically, the Eleventh National Conference and some of its implications.

DEVELOPMENT OF CONFERENCE

The National Conference on Interstate Milk Shipments was a natural outgrowth of circumstances and necessity. The sanitary quality of milk shipped interstate as well as intrastate varied considerably and was of concern to health authorities in many areas. Efforts had been made to reconcile differences between areas, but lack of a uniform approach to the problem made it difficult to accomplish much on a nationwide basis. This lack of a uniform approach resulted in the creation and continuance of a confusing situation. Consequently, officials in receiving areas insisted on making inspections of milk plants and dairy farms in the producing areas. This system of supervision created a multiplicity of inspections and the application of a wide variety of requirements, which in turn led to more confusion and misunderstanding. Industrialization and urbanization during and following World War II and advances in dairy science and technology further complicated the already existing problems. It became obvious, therefore, that a proper solution to the problem was necessary to afford an acceptable quality of milk for all, to permit greater utilization of milk supplies available throughout the country, and to increase materially the incentive for greater production of high-quality milk. The more economical administration of interstate shipments of milk would reduce the actual cost of supervision and the complexity of maintaining satisfactory supervision of interstate milk supplies; this would give the confidence necessary to provide a good working agreement between States.

Recognizing these conditions and the need for a proper solution, several organizations requested the U. S. Public Health Service to devise a plan that would facilitate the certification of interstate milk supplies, and to assist the States in developing working agreements to implement such a plan. As a result, the first meeting of the National Conference on Interstate Milk Shipments was held in 1950. From this Conference came the voluntary State-Public Health Service program for the certification of interstate milk shipments. Although technological advances and new knowledge in the area of milk control have necessitated some changes, the fundamentals of the agreements developed by this Conference remain basically the same.

OBJECTIVES OF CONFERENCE

The principal objectives of the Conference can be summarized as follows: (*a*) the use of uniform ordinances, regulations, or standards that will permit and assure a free flow of high quality milk between states; (*b*) elimination of interstate barriers, intentional or unintentional, through cooperation, better understanding, and effective supervision; (*c*) to provide industry and regulatory personnel with information that will lead to mutual understanding and respect for each other's problems; and (*d*) to provide states and municipalities with reliable information concern-

[1]Presented at the 54th Annual Meeting of the International Association of Milk, Food, and Environmental Sanitarians, Inc., Miami Beach, Florida, August 14-17, 1967.