HUMAN FOOD POISONING DUE TO GROWTH OF CLOSTRIDUM PERFRINGENS (C. WELCHII) IN FRESHLY COOKED CHICKEN: PRELIMINARY NOTE

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Although our studies are as yet incomplete, it seems desirable to summarize here our observations relating to certain outbreaks of food poisoning in which Clostridium perfringens has been the only organism of significance isolated. This and other facts including the illness of a human volunteer, who showed typical symptoms following the eating of a sample known to be contaminated by this organism and to be free from all other species commonly regarded as food-poisoning agents, suggest that C. perfringens may at times produce potent enterotoxin(s?) for humans. Although such a conclusion does not appear strange when compared with other information concerning the species (lamb dysentery by type B cultures—see McCoy and McClung, 1938; Oakley, 1943), we have been unable to find previous reports of cases similar to those we have encountered.

The majority of the cases which have come to our attention resulted from food eaten in a large public cafeteria. Outbreaks occurred in December, 1943, January, 1944, and January and February, 1945. One outbreak (confirmed bacteriologically) in February, 1945, occurred, however, in individuals who ate in a private dining club. Although evidence on the number of persons involved in the various outbreaks is scanty, at least twenty persons are known to have been involved at one time or another, and the evidence suggests a higher figure. Similarly, although little can be said about the proportion of individuals who were ill in relation to the total number consuming the contaminated dishes, there is reason to suspect individual susceptibility. Repeat attacks in the same person are known to have occurred. The symptoms include nausea, vomiting (rare), intestinal cramps, and (invariably) a pronounced diarrhea. In the majority of cases the symptoms began 8 to 12 hours after the meal, with the diarrhea continuing for about 12 hours. Although the affected individuals were weak on the day following the period of symptoms, most of those who were ill were able to continue normal activities. One individual was confined at home in bed for 48 hours.

In all instances for which we have adequate records, the cases trace to the eating of dishes prepared from chicken which was cooked one day and served on the following day. The chickens were cooked by steam under low pressure for a period of 3 hours; they were then removed from the broth, which was saved for later use, and boned. Creamed chicken, croquettes prepared from the precooked chicken and browned in deep fat, and other similar dishes have been implicated and confirmed by bacteriological evidence. It should be noted that it is a common institutional practice to precook fowl on the day previous to
serving providing refrigeration is available for the holding period. In the cafeteria adequate refrigeration was available, and inattention to this was not the responsible factor except that the pans of food may not have cooled to the refrigerator temperature during the holding period. The evidence points to a survival of the causal organism in the broth and subsequent growth during the storage period. The heat used in the final preparation of the dishes apparently was insufficient to destroy the toxin which was formed. It is believed that the broth was usually the agent carrying the contamination since beef dishes in which chicken broth was occasionally used were implicated only when chicken broth was used. It would appear that the contaminating organisms were present on the fowls following dressing and survived the cooking process by virtue of protection by the fat. This and other circumstances peculiar to the kitchen are believed to be the responsible factors since samples of washings from raw chickens from other kitchens not experiencing the trouble invariably were positive. The poisonings seem to have been eliminated by a rigid cleanup system in the cafeteria kitchen and by the institution of a routine of boiling the broth after the refrigeration period and before mixing with any other food materials.

In our samples the spoilage could be detected in many cases by the appearance of gas bubbles in liquid or semiliquid samples. In all samples of broth, but not necessarily with samples like croquettes, gram stains revealed heavy contamination by gram-positive rod forms, which upon isolation following enrichment in thioglycollate broth, litmus milk with reduced iron, or sulfite agar (Committee on Bacteriologic Technic, 1943, 1944) gave cultural and other reactions of C. perfringens. Attempts to demonstrate enterotoxin production in mice and guinea pigs by these cultures have yielded only negative results; other animals have not been tested, and likewise the toxin type of the new strains has not been established. A positive Nagler reaction is inhibited by type A antitoxin, but this is true, as would be expected, of types B, C, and D.

In view of these facts, it seems reasonable to suppose that C. perfringens may be involved in the toxic spoilage of foods other than chicken and may have been overlooked previously because of the fact that the routine bacteriological examination of food samples involved in food-poisoning outbreaks may not have included procedures which would reveal the presence of the organism. It is suggested that in the future those who are investigating outbreaks include the inoculation of suitable anaerobic media. It should perhaps be emphasized that, in view of the ubiquitous distribution of the organism, mere demonstration of the organism in a food may not necessarily mean that the food was contaminated sufficiently for this species to be the offending organism. By the time of examination the contamination in our samples was sufficient for the food to show many rod-shaped cells by microscopic smear and, in some instances, gas production. Lack of susceptible experimental animals has prevented direct demonstration of the toxin in filtrates of foods; it is possible also that the toxin is an endotoxin.
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