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Well, this morning we have with us on the panel, Dr. A. B. Stevenson, of the Canadian Department of Agriculture, Vineland Station, Ontario; and Dr. Carlton Herman, Protection Wildlife Research Center, FWS, Patuxent Wildlife Center, Laure1, Maryland.

We are going to change the program somewhat, because we have a varied program; and I intend to throw a lot in a hurry because I am a Maine-iac and I can throw a lot in a hurry. We are going to devote twenty minutes per segment of our various discussions in this unit, and five minutes or more will be devoted for the discussion. Then, you can throw the questions at the individual speakers at the end of the hour.

Well, this morning and yesterday afternoon, we discussed many aspects of research, which is my particular discussion, and maybe you have wondered exactly how does research fit into it, because you have had a lot of research thrown at you. Various field men like John Beck and Ochs and other field

men located throughout the region all see these various problems and they do something about it. They take a material from research and modify it or apply it to the field before researchers get to this point. Also they see a product about to be marketed and they see this tool fits somewhere, so they discuss this and set up a guideline for an investigation --I will call this investigations — and then conduct the programs.

Consequently we have basic research conducted by the Bureau of Wildlife Research and also field research conducted by John Beck and his cohorts, so yesterday and this morning, we discussed some of the things which research is actually doing, but a little bit behind the field application, so I will try not to duplicate what has been discussed. I am going to try to put myself in the wildlife researcher's framework.

First of all, there are three areas of investigation available in developing methods for solving bird problems: one, the biological; two, the physical; and, three, the chemical. Let's take these in order. Before I continue, rather than take you to Cloud 9, I will keep you on earth and cover very lightly the areas of research and give you details of research that show great promise.

One, the biological: This area of investigation had to do with habitat manipulation. It concerns such things as developing resistant species of grain, substituting crops not subject to damage, and changing feeding practices. The most recent findings in this area are that a simple over-head roof, erected over fish piers, will prevent herring and great black-backed gulls from feeding under such cover.

Two, the physical: This area of investigation has to do with the bird itself, in most cases, stress. To be effective, the birds must be subject to stress when they are in the area. If they overcome one stress, another must be applied, either separately or along with the overcome stress. The latest phase of research in this area is the development of air horns controlled by batteries. Distress calls have been used successfully on starlings, herring gulls, and red-winged blackbirds. However, in the case of scaring devices and distress calls, unless they are employed correctly, the birds, when scared from an area, may return. When they do, they will consume more food in a shorter period of time than normal.

Trapping has been employed successfully in urban areas as well as rural areas. Recently, in Syracuse, New York, 55,000 starlings were captured in decoy traps in one week. Two traps were located on dumps and one in an eleven-story building. The trap in the building was the most successful. Decoy birds, left in the traps, determine to a great extent the species caught. Young starlings normally feed on only soft fruit, thus they are difficult to bait. Therefore, trapping is the best means for control in cherry and blueberry crops.

Three, the chemical: Some oral poisons are fast-acting and please the public, however, they do create a spook factor in most birds. Slow-acting poisons

require twelve hours to seven days for death to occur, thus the birds die at roost sites. The Bureau is searching for toxic chemicals which are selective to the species, and we have found several. They have no secondary poisoning hazards, 500-1000 more toxic to the target species than to mammals, and some are more toxic to one species of bird than to another. For example, a chemical coded 1339 is more toxic to red-wings than to starlings. The birds demonstrate no aversion to accepting the treated baits.

Contact poisons are slow-acting and toxic to all species of birds, therefore, it is important to know the species present at the roost. Contact sprays have been applied to vegetation roosts after they have been found not to be phyto-toxic. It has been found that such are successful if applied to the roost when the birds are molting and the roost is not too dense. Methocel, a thickening agent, added to the spray, will decrease the run-off and thus obtain a more effective kill. Diesel oil has been found to be a more effective carrier than water. To date, it has been found that organo-phosphates are the most effective.

Some work has been done with three-eighth inch rope treated with the material. The ropes are strung in the roost, and the birds, arriving at- the roost, jockey for position; in doing so, land on the rope for a brief period before occupying the final roost position.

Roost toxicants have been employed successfully under some conditions, however, repellent effects have been observed with the organo-phosphates. Chlorinated hydrocarbons are also effective, and very little repellent effects have been observed. More on this technique later in the program.

Contact sprays have been employed successfully, using the experience gained from the use of the light trap. It was found that birds can be herded from a roost toward powerful lights. This technique has been adopted to the application of control sprays. The material is applied in a curtain behind the lights. The material is captured and recirculated. The birds fly toward the light and through the curtain of toxic material.

Drugs have been used successfully as drugged birds usually display or demonstrate the effect of the drug. Some drugs cause greater display than others. This principle is being used at airports, dumps, and on grain crops to scare the birds from the area. For example, several ears of corn per acre are stripped and the standing ears treated with the drug. The birds, upon ingesting the drugged corn, either fall into unnatural positions or flutter their wings and tumble in flight or on the ground; such displays spook the other birds from the area.

Wetting agents have been used under laboratory conditions in the field. Bird kills have been achieved with a two per sent solution of a wetting agent and water when air temperatures have been forty degrees Fahrenheit and the wind at five miles per hour. Death was obtained by exposure. Further tests revealed that only one of two seconds of wetting is required to produce death. The lower the air temperature and the greater the wind velocity, the more

effective the control and time until death. Death usually occurs in about twenty-seven minutes. The light trap principle is being employed with this technique. The material is not phyto-toxic, nor will it affect fish at the concentrations used.

Chemical sterilants have been used successfully to sterilize eggs in the bird. A black dye, used to identify the bird that consumed the treated bait, was found to be as effective as the chemical sterilants. The dye appears in the egg yolk. If the egg is hard-boiled, and the yolk sliced, black rings are readily observed. The depth of the dyed rings aid in determining at what stage in egg development the black dye was consumed.

You will note that I have devoted considerable time to control. This was meant to be, as the biggest per cent of research money today is being spent on reductional methods. I have a few minutes for questions. Are there any?

DR. SPEAR: In your opening sentence or paragraph, you mentioned the Cloud 9 aspect, and as I understood you, you threw out ultrasonics as a Cloud 9 approach. Did I read you correctly?

MODERATOR FAULKNER: You sure did. We have a lot to learn as far as ultrasonics are concerned. First of all, from the information which is being distributed from Germany and also from the United States in the hearing range of birds, why they certainly don't — aren't able to hear beyond our frequency. We get into decibles we cannot employ; because when you get over 110 decibles, this is detrimental to men. If you go up to 110 decibles, which you might need, you get into problems.

Again, Phil, there might be something, with the findings we are getting today, in ultrasonics, in the equipment we are getting — even fifty per cent of it is not standardized when they guarantee the frequencies. They are having a job to get fidelity with equipment. We had to go to the Bureau of Standards and measure these things out. But we are getting more finesse at this line, so we are finding out better how the birds are communicating.

It takes two years to talk each other's language when we talk to an electronics man. I personally believe we have a ways to go yet; and maybe in the next two years there will be more on this one, but actually there has been very little time devoted with this phase. We have been working with distress calls mostly.

Our next speaker this morning is Dr. A. B. Stevenson.

DR. STEVENSON: Probably I should point out right in the beginning that my inclusion in this program isn't to be taken as any indication that I am an expert in this field, but I accepted the offer to participate on the panel because it gives me my first opportunity to attend a conference of this nature and establish contact in the field in which I am a novice as yet. Furthermore, control in itself is not a major part of my program so far. I am in this field of bird damage to food crops, more as an accident of geography than

any other particular qualification. My first impression is that the field of vertebrate control poses more problems than the insects do. Then there are some crises where the two problems are not so far apart.

The topic I have been given this morning is called "Canadian Programs," and you have already heard about the Ontario blackbird program, so I will confine my remarks to the program carried out by federal agencies. I will mention briefly some of the projects going on throughout the country and then later deal with more of the problems in my home area, the Niagara Fruit Belt.

In the Canadian federal service the departments most, likely to be involved in bird control problems are the Canadian Wildlife Service of the Department of Northern Affairs and Natural Resources, which is concerned with all kinds of problems that involve wildlife as pests, and the Canada Department of Agriculture, concerned chiefly with crop damage by birds.

The Canadian Wildlife Service is active in three main fields of bird control: crop depredations, bird aircraft-encounters, and the fouling of reservoirs. In this work, they cooperate with municipal and provincial governments and the National Research Council.

In the field of crop depredations, the damaging of grain by ducks and sandhill cranes is important in the Canadian West. The Canadian Wildlife Service and Saskatchewan Department of Natural Resources have a project underway to devise a means of reducing the damage by sandhill cranes that will be less damaging to the whooping crane population than the practice of issuing shooting permits. Also extensive work has been done on the duck problem. W. J. D. Stephen of the Canadian Wildlife Service staff in Manitoba has been active in this work. In determining the extent of duck damage it was estimated that in 1959, nineteen per cent of the farms on the Canadian prairies were affected, with an average loss of about $280 per farm, and an estimated total loss of about twelve million dollars. Mallards and pintails are the species that do most of the damage. There is a public relations problem here, as many farmers think the ducks damaging their crops come from waterfowl management areas. The reaction of farmers to duck damage is therefore a limiting factor to the maintenance and increase of the continental duck supply.

Promising methods of controlling the damage by ducks, cranes, gulls and pheasants has been the use of electronically-timed acetylene exploders combined with the provision of alternate feeding areas or pastures, such as plantings of barley. In some cases ducks have been herded to feeding areas by aircraft. Other scaring devices such as flashing lights and reflectors for use by day or night have been devised.

The problem of birds at or near airports is being studied by Harris of the Canadian Wildlife Service. The work has included: one, aircraft protection; two, bird detection and provision of warning services; three, control by, (a) modification of environment; (b) repulsion of birds; (c) local extermination. While gulls, starlings and rock doves (pigeons) were the worst hazard, these studies showed that at least nine other species have been shown to

strike aircraft.

Food, cover, and water were determined to be of major importance in attracting birds to the vicinity of airports. In at least five major airports in Canada, garbage dumps appeared to be responsible. Also agricultural crops, mainly forage or cereal, near airports are attractants, especially during cultivation, and again at maturity of the crop. Some crops offer cover for certain species. Recommendations regarding the controlled use of such land are another step in control.

Earthworms and insects on airport runways are another attractant — chemical control of these is being investigated. Aquatic insects, grasshoppers, seeds and fruits of weeds and other wild plants are also of importance.

From the point of view of modifying available cover, attention has been given to modification of structural features of buildings to prevent their use by the birds. Removal of grass, trees and other plants from the runways or its vicinity is another step. Natural bodies of water within airport limits attract ducks. These may be drained, filled, or wires may be strung across such ponds to prevent the birds reaching them.

Repulsion methods include use of distress calls and trained falcons, specifically peregrine falcons, to control glaucous-winged gulls. Also some attention has been given to use of dogs to harass birds. There is also the local extermination of some relatively immobile species, *i-e.*, trapping doves, and permit shooting of pheasants.

Other projects being carried out by the Canadian Wildlife Service include a study of the effect on the number of returning salmon in one Nova Scotia river of shooting mergansers. Also some work is being done on the fouling of reservoirs. I have no further information on these projects.

In the Canada Department of Agriculture probably the first bird damage study, now nearing completion, was that of Bird and Smith in Winnipeg, Manitoba. Large flocks of red-wing blackbirds feeding in grain and sunflower fields in southern Manitoba were causing sufficient damage to arouse complaints. In 1960 a study of the nesting, flocking and feeding habits was begun. Bird and Smith found that red-wings in the area fed largely on waste grain and weed seeds in the spring, that insects formed a large part of the diet of both adults and young during June and July, and that vegetable matter again was the major source of food later in the summer. The damage to cereals occurred from the milk stage to ripeness and was of relatively short duration; even then the birds did not feed entirely on the crops, consuming large numbers of harmful insects. Their conclusions were that the red-wing as a whole does far more good than harm. They recommended preventing damage by early planting of crops, leaving harvested fields untilled to serve as feeding areas, avoiding the planting of susceptible crops near known roosts, and using scaring devices persistently.

In Nova Scotia, there is a recently initiated project on bird damage to small

fruits at a Canada Department of Agriculture research station; I have no further details on this one.

Our own work is concerned with bird damage to fruit crops in the Niagara Peninsula of Ontario. Damage has been increasing in recent years, and is of most concern on sweet cherries and grapes. The introduction of various new varieties of grapes that appear to be especially susceptible to bird damage has intensified the problem. In answer to requests of grower organizations and the area's large wine industry investigations into bird damage to grapes were begun in 1963. The project is to study the identity, importance and feeding behavior of bird species attacking grapes in the area and to devise or assess methods of damage control. The project may be expanded to include cherries and other crops.

A questionnaire was sent to most fruit growers in the area. About eleven per cent responded, almost all being persons having some damage- Most reports were of damage to cherries, mainly sweet cherries and to grapes. Fifty-three per cent reported damage to grapes, but many of these were to common American varieties usually not damaged to any great extent. The most seriously damaged varieties are French hybrids and a few new American varieties such as New York Muscat, Some of these varieties command a premium price about fifty per cent higher than the common kinds.

A surprising twenty five per cent reported damage to peaches, and apples, pears, strawberries and vegetables were mentioned in some cases. About twenty-two per cent of the reports were of what I would classify as significant damage to cherries, about one hundred dollars or more, and about the same to grapes, Five percent reported significant damage to other crops, What could be regarded as serious injury to at least one crop was reported in about one-third of the replies. Only two growers claimed more than $1,000, about five and one-half per cent were over $500; about one-fifth were in the $200-$500 range.

To assess the damage in individual vineyards several methods are used., To note the frequency of damaged vineyards, we simply walked through the various vineyards and noted whether damage was nil, slight, moderate, or severe. In other cases, one hundred vines per vineyard were observed while walking through, and injury was rated one, light; two, moderate; or three, severe. To record damage in more detail, a certain number of sites were chosen at random at which one fruit cluster at each of two or three levels was assessed. In this way, any difference in damage at different heights from the ground was accounted for. The clusters were either rated in categories from one to six, depending on the estimated per cent berries damaged, or the bunch was removed and the numbers of injured or missing berries were counted. In some cases the greater part of the injury consists of the removal of berries from the vine. In others, damage is chiefly in the form of pecked and punctured fruits that often are invaded by rot and fruit flies. The heaviest damage recorded of the former type has been about fifty per cent of the berries missing; of the latter type also about fifty per cent injury, most of punctured berries with a weight loss of thirty-two per cent. The actual loss was

probably greater since many clusters so injured were unmarketable.

Proposed behavior studies include the observation of susceptible vineyards to determine the abundance and identity of birds attacking the fruit, the behavior of individuals or flocks within the vineyard, movement to the crop from roosting or other feeding areas, and the importance of local versus wandering individuals or flocks in damage.

Factors affecting damage: Cover is one important factor. While damage is sometimes evenly distributed throughout large vineyards, it seems to be more frequently serious in specialized situations, such as near woodlots, hedgerows and orchards. In some larger plantings there may be severe injury near the cover, but the overall loss may not be serious. Sometimes small plantings of an attractive variety may be severely attacked, even when located among larger patches of other varieties that may remain practically unscathed. Young vines with less luxuriant growth are often relatively heavily damaged. Bunches of grapes that are exposed are more apt to be injured than those under cover of the foliage, and so losses on very vigorous vines may not be serious. More often than not there is more feeding injury near the top of the trellis, but in one or two cases I have observed the greater damage near the ground.

Damage may begin about a month before harvest, and naturally gets worse as the grapes mature. Often we will find that on an otherwise immature bunch birds may have pecked a few small grapes that ripened earlier.

Most of the older, standard American varieties that still make up the most of our vineyards are not usually damaged very much. These are mainly labrusca type grapes with the so-called foxy taste. A few of the earlier maturing older varieties, certain of the French hybrids that are increasing in numbers in our area, and a few new varieties developed in North America are the most susceptible.

Bird Species: In our survey, starlings were implicated by eighty-six per cent of the growers, robins by seventy-three per cent, grackles by thirty-eight per cent, and about one dozen other species were mentioned by a small percentage. Many growers reported damage by more than one species and I have not figured out the relation between species reported and crop. In some vineyards robins are active just about every time we visit at all times of day. All of the robins that we have taken in vineyards for stomach examination were feeding almost exclusively on grapes. On the other hand, there are damaged vineyards where we have not been able to catch the birds in the act; it may be that starlings visit these in flocks at various times. We would like to determine how the damage caused by a moderate number of robins feeding all day would compare to that caused by a large flock of starlings visiting a vineyard for only a short time.

Control: Our survey showed that slightly more than half of the growers use some sort of exploder. Fifty per cent spend some time shooting. Many go in for visual repellents — forty per cent — and with some this is the only approach. Only a few use traps, poison baits and other methods.

The cost of control varies. Only five per cent estimated that they spend more than $100 a year, but it was often pointed out that this was difficult to estimate since so much of the grower's own time was involved. Another problem with noise-making devices in a thickly populated area such as ours is the effect of the noise on the non-farming population.

In organizing our program, I felt that we needed to learn more about the nature of damage and the behavior of the damaging birds before venturing into experiments on control methods. We are testing a unit for broadcasting distress calls that has been developed as a prototype by a Toronto firm. This consists of a compact amp-tape unit powered by a 12-volt car battery, to which several loudspeakers can be added. The unit plays a small tape cartridge automatically, a 10-second call every fifteen minutes. A photo-electric cell activates the unit at dawn and turns it off at night.

Are there any questions?

MR. HOCKENYOS: I am interested in this German pistol, since I have put in a lot of time on pyrotechnics. Could you give me an idea how I could take steps to get one imported?

DR. STEVENSON: 1 will get your name and send you the name of the company* It is a chap named Paul Zing, a fireworks company. The one pistol that we have is just a small Derringer type or something that fires. The firecracker is about this long and it is activated by a blank shell, like the fire used for a starting pistol.

I have seen another grape grower in the area which imported one more like a six-shooter, and he could put six of the small cartridges in it, and, of course, had to pop in the firecracker each time, but I think the starting pistol they sent us must have cost about a dollar and a half in German money, but we can't get the permission to import them into Canada because of the firecracker.

MODERATOR FAULKNER: Thank you, Dr. Stevenson. (Incidentally they have another one that looks like a pen light, another German product.) The next one is Dr$_0$ Carlton Herman on "Disease Aspects."

DR. HERMAN: At the request of our moderator in correspondence a while back, the title that I put on this presentation is "Disease as a Factor in Bird Control." I have, expounded on this on numerous occasions in the past and have met with much interesting discussion, and on one occasion your moderator heard me do this and requested that I repeat somewhat the same sort of philosophy here.

Disease is an entity not too well understood by those not closely associated with it. Superficial considerations often lead to the suggestion that perhaps here is a tool with excellent possibilities for use in control of undesired species of animals. The problems of controlling depredations by blackbirds inspire investigation of this possibility. To discuss disease as a factor in

bird control we must first review what is known of natural occurrence and its impact on the population. In the first part of my presentation I will cite as examples primarily what is known of blackbirds and starlings. In order to understand the potential of disease as a control mechanism it is necessary to examine efforts with mammal control, for no such approach has as yet been attempted with wild birds. Certain prerequisite criteria are essential before a disease control project can be implemented and these are set forth at the end of this paper.

There is a tremendous volume of published data on the occurrence of potential disease-causing organisms in wild birds. Most of it relates to discovery of new parasites and their taxonomy. Case reports, histories of prevalence or frequency of specific diseases in limited areas or summaries of such reports are rare. Details of pathogenesis or pathology, or a clarification of life cycles, that tell of disease potential or mode of infection are even rarer.

Epizootics occasionally have been recognized in wild bird populations, but the causes often remain unidentified. In contrast, there are many case re-ports for single individuals; these only suggest a potential for losses and are not evidence of actual epizootics. For example, aspergillosis (a fungus disease of air sacs and lungs) has been reported from cowbirds and grackles (Clark, 1960), pseudotuberculosis, in icterids, has been reported as cause of death at least twice (Beaudette, 1940; Clark and Locke, 1962), and a case of tuberculosis has been reported from a cowbird (Hudson and Beaudette, 1929). There have been some detailed surveys; for example, parasites of starlings in New England (Boyd, 1951), and malaria in redwing blackbirds from Cape Cod (Herman, 1938). Similar parasites frequently have been reported from the same or related species of birds. Other surveys have emphasized the occurrence of particular parasites from a variety of hosts, such as malaria in birds from Kern County, California (Herman, et al., 1954). Several reviews have brought the scattered, published data together (Herman, 1944; Halloran, 1955; Herman, 1955).

Epizootics in which large numbers of birds die of a diagnosed cause have been recognized in very few cases. The most dramatic is botulism which causes extensive losses among birds, particularly waterfowl. Development of the disease is related to habitat contamination; a toxin is produced by the grow-ing botulinus bacterium and the birds get sick from consuming this toxin. The bacteria grow best in the absence of oxygen and thus the disease occurs in association with decaying animal or plant matter which produce conditions ideal for such growth. Outbreaks have been reported primarily among water-fowl, shorebirds, pheasants and poultry. Kalmback (1934) also listed several species of icterid birds afflicted with botulism during natural outbreaks.

Bacteria of the Salmonella group are among the chief causes of disease losses among captive birds, such as poultry. These bacteria are pathogens of the intestines and cause disease, often fatal in a wide variety of animal hosts, including man. The poultry industry has expended more effort to control losses from these bacteria than any other disease entity. Infection is usually by mouth in contaminated foods, although some salmonellas are trans-

mitted through the egg. Salmonella infections have been reported from starling, rusty blackbirds and cowbirds in New Jersey (Hudson and Tudor, 1957). Potentially they could cause extensive losses among wild birds but, to date, no severe outbreaks have been noted in North America.

Encephalitis is a virus-caused disease which has had much publicity in recent years. It is fatal to horses and man. Birds are involved in the natural cycle and blackbirds, as well as other species, are known to become infected. However, English sparrows and pheasants are the only species of our wild birds known to have died from encephalitis (Herman, 1962). The virus is usually transmitted by mosquitoes. Although blackbirds frequently have antibodies in their blood sera, there is no evidence that the infection is fatal. In fact, preliminary experiments performed a few years ago in our laboratory on experimentally inoculated cowbirds would suggest that these birds do not ordinarily die from the infection.

Pox, another virus infection frequently recognized in birds, is manifested by the development of small tumors (up to the size of a pea), usually on the beak or feet. Herman, Locke and Clark 1962) reported this infection in cowbirds and grackles. Ordinarily these tumors disappear in time and infected birds recover. However, Beaudette (1953 has shown that under some circumstances birds develop a viremia (virus circulating in the blood) which is rapidly fatal. Tumors do not necessarily develop in such cases. Pox can be transmitted by contact between birds or by mosquitoes. Tumors usually develop at the site of contact or mosquito bite.

The so-called Roux sarcoma virus is another potential source of bird losses. Neither methods of natural transmission nor the extent of occurrence in wild bird populations are known. We are conducting a survey in collaboration with the Department of Pathobiology of the John Hopkins University School of Hygiene and Public Health in an attempt to uncover the prevalence of this virus in the avifauna. The Roux sarcoma virus appears to be connected in some way with the occurrence of leukemia, a disease which takes a large toll in poultry and is known to occur in wild birds as well.

In a survey we have been conducting on blackbirds, we have uncovered at least 65 species of parasites, either by our own examinations or from reports in the literature. All of these parasites must be considered to be potential pathogens, even though we have not yet uncovered evidence of disease that can be attributed to any of them. Each must be studied experimentally to determine mode of infection and the circumstances under which it can be harmful or fatal to the host. The main point I wish to bring out here is that none of these parasites are host-specific to the blackbirds and they can be expected to be found in at least a variety of passeriform birds if not in most species of birds?. Parasites are likely to be more narrowly host specific than bacteria or v ruses, but even with parasites the range of infective hosts can usually be expected to include most of the passeriform species.

Other than the cases cited above, I know of no deaths of blackbirds that have been confirmed as disease-caused. This does not mean blackbirds do not die

of disease. They undoubtedly do, just as do other birds and mammals, including man. It simply means that investigators have been slow to uncover losses or clarify their causes, and this is due, in large part, to a dearth of investigators.

The classic example of a disease agent used to control a wild animal population is provided by the story of myxomatosis and rabbits in Australia. I reviewed the original experiments in an article ten years ago (Herman, 1953). The European rabbit (Oryctolagus cuniculus), common as a wild species in Europe, and ancestor of all breeds of laboratory rabbits, was first introduced into Australia around 1860. The subsequent population explosion and its impact on the sheep raising industry is familiar to all of you.

Prior to release of the myxoma virus (causative agent of myxomatosis) many experiments had been performed to test its control potential. It had been demonstrated that the virus is nearly always lethal to wild and domesticated forms of the European rabbit. Further, it had been demonstrated that all common domesticated animals, as well as representatives of the native fauna (mostly marsupials) and the introduced hare (Lepus europaeus), were refractory.

Early experiments with myxoma virus did not portend the later success. Initial introduction in an arid area in Australia was unsuccessful. Unsuccessful attempts also were made on islands in the North Sea. Early field trials included tests conducted on an island off the South Australian coast and on a dry semi-arid inland area. Mosquitoes were inconspicuous or absent in both areas and there was only very limited spread of the virus. It has been suggested that the launching of a large scale field experiment was delayed because health authorities objected to experimental field studies of the virus except in sparsely populated areas. The importance of mosquitoes as vectors of the disease was not known when these initial field tests were made.

Field trials with myxoma virus were undertaken on a large scale in 1950, particularly in the Murray-Darling drainage in eastern Australia, in a zone of higher rainfall. Since the belief still prevailed that infection would be spread mainly be contact between rabbits, tests were made in autumn, winter and spring. Only a few local outbreaks of infection were observed and at the end of November, 1950, the experiment was considered fruitless and the investigators went home in disappointment. However, in early December (beginning of the Australian summer) rabbits were reported to be dying by hundreds along the Murray River flats; a dramatic epizootic followed. The disease spread rapidly throughout most of the drainage system in areas close to water. It was estimated that the epizootic covered an area that extended 1000 miles from North to South and a slightly greater distance from East to West. High mortalities (90 percent or better) were noted. It was reported that in places previously swarming with rabbits it was possible to drive for a day or more and see only isolated survivors.

Transmission of the virus was reported to be mechanical. Australian investigators havi shown that a number of blood«sucking arthropods are capable of

transmitting the infection but that mosquitoes are the chief vector. The vector has been referred to as a "flying pin" 5 in other words, its mouth parts become directly contaminated with the virus rather than by the virus developing or multiplying within its body. Thus any arthropod which would feed on a lesion on an infected rabbit and then bite a susceptible host would transmit the virus by contamination.

The impact of this disease control experiment on grazing pastures and sheep production in the ensuing years has been a boon to the economy of Australia, This impact, as well as the concurrent scientific advances has been publicized and documented by a number of Australian investigators and in being fully summarized by Dr. Frank Fenner, of the Australian National University, in a book on myxomatosis which is scheduled for publication early in 1965.

An interesting series of events also occurred in Europe, A French doctor, desiring to reduce the native rabbit population on his walled estate in France, released the virus and dramatically reduced his local population. Since this virus can be transmitted by free-flying arthropods, the wall around his estate was no barrier and the disease spread through much of Europe and also to the British Isles. The kill of the native rabbits was as dramatic as it was in Australia. The gains to agricultural interests have been great, but the sportsman lost his most important trophy, and the numerous people who kept a few rabbits in the backyard as a source of food lost this supply of supplementary protein.

For completeness of the myxoma story, i must point out that for several decades outbreaks of myxomatosis have occurred among commercial rabbitries in California. Our native cottontails are presumably the reservoir of infection. The cottontails are susceptible to the virus but develop no characteristic lesions, manifestations of the disease, or fatalities.

Davis and Jensen (1952) reported on experimental, attempts to introduce an epizootic among wild rats living naturally on a farm in Maryland. They inoculated a bacterium, Salmonella enteritidis, into this population which preliminary studies indicated was free of any Salmonella. This Salmonella is considered highly pathogenic to rats. It causes extensive intestinal involvement and is transmitted in contaminated feces from infected animals.

The rats lived in four buildings on a farm and had all the characteristics of a wild population. They were trapped alive in box traps, marked, bled and swabbed, and released at the place of capture. During the 2 years of the study, about 2000 individual rats were caught and there were about 3,000 recaptures. In February 1950, 20 rats in one building were inoculated with the culture of Salmonella. Then in October 1950, another group in the same building was inoculated along with a group in another building. As pointed out by these authors, the determination and comparison of mortality rates in a wild population is complex and difficult. Mortality rate is a rather loose term used to indicate a proportion of deaths. In analysis of their results they substituted "probability of surviving" as their criteria for interpretation. This probability was derived from a statistical analysis of retrapping data; they also

determined agglutinin levels of sera obtained by heart puncture and cultured cloacal swabs and feces. They recognized that their procedures gave only an index of infection, although their data does show evidence of spread of the infection within the population. Greatest spread apparently occurred during May 1951, when there were radical changes in food supply and shelter that apparently caused considerable movement of the rats. There was no more spread during the summer and fall of 1951, and the number of positives gradually declined. The infection was at a low level when the study was terminated in October 1951.

Although Davis and Jensen did not fully explore the population changes, it was certain that the population doubled during the interim of their study, and thus the induced Salmonella could not be considered effective in lowering the population. They point out that their data show clearly that an organism of potential pathogenicity may have no measurable effect on population size, mortality or reproduction. They emphasize that their data indicate the complexity of disease phenomena and warn against hasty conclusions about the role of pathogens in population management.

There undoubtedly have been many unreported attempts to control wildlife populations with disease-causing agents. It is known that as early as the 1880's Pasteur recommended the introduction of a bacterial pathogen to reduce the rabbit population in Australia; in fact he sent one of his assistants to Australia with cultures of the organism. However, cautious government officials vetoed the project.

A number of years ago I was told of a 100 percent successful project to eliminate the wild pig population on a privately owned island off the coast of California by the introduction of hog cholera virus. But to my knowledge this event was never documented. More recently, similar attempts (Schroeder, 1964) to eradicate native swine on another island off the California coast with this virus yielded disappointing results. While it was demonstrated that the disease was well established in a few animals there was little spread and the investigators concluded that the use of live cholera virus in depopulating wild swine is not satisfactory. Because of the repercussions that might occur from potential dangers of introducing a disease into a population, it is only subsequent events, such as resulted from the release of myxoma virus by the French doctor, that bring these attempts to the attention of the public or even the scientific community.

An epidemic (involving man) or epizootic (involving animals) is a complex phenomenon. Its full understanding requires a thorough knowledge of the biology of the causative agent and associated organisms, of the definitive host and vectors; and of the transmitters or intermediate hosts if they are involved. It also involves a knowledge of the inter-relations of various hosts that may become a part of the complex, plus data on ecology, environment, behavior, food supply, immunology, pathology, and more. We cannot, here, go into details of all the possible ramifications of the problem. Instead, I wish to conclude this presentation with a few imperative rules that must be basic to any consideration of introducing a potential disease-causing organism into a wildlife population as a method of controlling that population.

1. The applicant organism must be demonstrated to be highly pathogenic to the prospective subject species. Usually a disease which normally occurs in the subject species is not a potential applicant or it would already be doing an adequate job. Therefore the applicant is more likely to be an organism exotic to the subject species.
2. The potential killing power, residual duration, and ultimate resistance must be anticipated. One should strive for a complete knowledge as possible concerning the long range consequences to the total population and survival of the subject species.
3. The applicant organism must be host specific. We cannot introduce a disease into blackbirds that would be a threat to other birds, livestock, or man.
4. The applicant organism must be available. Not only is it necessary to be able to provide a sufficient supply of infective material for the initial implant, but the natural environment must be favorable for its perpetuation to provide the impact desired. If a vector or intermediate host is essential, it must be present in the environment,
5. If initiated, the control program should be monitored in every detail to insure its progress in the direction anticipated without adverse, detrimental side events not anticipated.

I do not wish to leave you with the impression that control of wildlife populations by implantation of a disease-causing organism is an impossibility. On the contrary, it has much merit if the criteria outlined above can be met. The events that followed application of myxoma virus in rabbits demonstrate this. However, be aware that this is a complex problem. We may have an acceptable applicant organism tomorrow and, again, one may not be discovered during our lifetime,

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I. might add to this that this is necessarily brief. I have not been given the opportunity to give you a full course in immunology, and so forth. They have limited me to 15 or 20 minutes, so some of the points brought out here may sound rather sketcy. 1 would be only too willing to answer any questions to clarify any points or broaden them as much as you will allow me in the way of time.

MR. HUCKENYOS: Could we summarize this by saying that as far *as* bird control, the prospect of achieving control through disease is not too promising?

DR. HERMAN: As of our knowledge at the moment. It is a potential that we shall continue to seek, and sooner or later someone will come up with something that we can experiment on, but it is not available at present.

DELEGATE: Is there very much research work being done with Public Health Service and Fish and Wildlife Service?

DR. HERMAN: Not with this viewpoint. Let us look once again to the history of the myxoma virus. This was first discovered in the eighteen hundreds in a public health laboratory in South America. It was then brought in the laboratory such as the Institute in Paris and the Rockefeller Institute in New York; and a lot of academic studies were conducted with it, because it was an interesting virus with which to work. It was not until the forties that a civilian worker had an outbreak, and they found out they had no screens on the window and mosquitoes had free access. They found that the local cot-ton tail rabbits had the infection and worked out the relationship of the wild rabbits to the domestic ones.

It was not until the middle forties that anyone had the idea that this might be something that we might use as a control tool, and number of workers for the next decade carried on studies of host susceptibility of various animals. It was not until they demonstrated that this was the thing that killed only this particular species of rabbits that anyone dared suggest that this might be the thing to be used in the wild.

There are other ramifications that are sort of clouding the picture, too; in spite of the tremendous impact that this has had on the rabbit population in Australia, it will not remain this way. Immunity builds up. Resistant animals build up. And eventually, the rabbit population of the European rabbit in Australia or even Europe will develop a resistance to this infection, and the impact of Myxoma fifty or a hundred years from now may be nil. How long this immunity takes to build up, we don't know,, It has been a successful project now for fifteen years. They are recognizing immune rabbits, but they are still have effective control.

DR. CRINGAN: Dr. Herman, would it be out of the question to take a disease which does not hit the birds because of the high temperature of birds and begin selecting strains of this organism for high temperature tolerance and try to change the disease rather than look for an absolutely new disease?

DR. HERMAN: This is being done to some extent in labs in various places and the relationship of stress factors to disease is being investigated, and ultimately some of these projects may reach a point where they will revert back to application the problem before us.

There is a tremendous amount of academic work being done. We were discussing, during this coffee break, how limited our knowledge is of the physiology and stress factors of birds contributing to these possibilities, and there has been practically no work done in this. As research in this field develops, and there are several universities trying to develop men on their staff to work in these directions, we will begin to have a better picture as to what we can do with changing the temperature resistance or organ-isms, its relation to habitat, environments, and so forth,. Population densities come into the picture as well. There are very interesting experiments going on, at present. I can cite one that I am familiar with.

We have a leucocytozoan parasite that occurs in waterfowl. It is a malaria type thing. When the bird is first infected, It runs what we call an incubation or pre-patent period that may vary from a few days to a week or more. Then you find the parasites in the blood stream with concurrent fever; and in a matter of days, the parasites disappear or greatly reduce in the blood stream, and the bird, if it recovers, may go on and harbor this for the rest of its life.

The lab work, looking at blood smears at any time of the year, won't find any parasites until about the time of egg-laying in the spring, at which time a few parasites appear in the blood stream, presumably going

back to Darwin's theories, and so forth, occurring so that the vectors can become reinfected to carry on the cycle of the parasite in its existence. In studying these phenomena, one worker found that by changing the light of day and moving it up, he could move up the time at which the relapse occurs as much as a month. Attempts with hormones and endocrine substances did not have any effect on this.

Another worker found — and this has not yet been published — that if he took birds that were naturally infected during the early summer and confined them within groups of fifteen or twenty birds each, at any time from there on, he could take a bird out of one of these groups and put it in Group B or C; and the aggression and fighting that would occur as a result of it being in another group of birds, peck order or what you will, is sufficient that the birds all come down with this relapse at any time that he does this, not just a month ahead of the breeding season or any time. He has done it in January; and he has done it in March, so that here are stress factors, behavior factors that come into the occurrence of disease, and its diagnosis, and undoubtedly its effect on the host. We have very little information on this, but we are delving into it; and as pointed out in the beginning of my paper, which is a thing — well, my wife says I never give a paper on wildlife disease without somewhere in there stating much more work needs to be done. This is probably true of all research, but when we consider the impact of disease in wildlife and compare it with investigators of disease of humans and livestock and how few we have working on wildlife disease, we can see why we are still a long way from having many of the answers we would like to have. It just takes a wide variety of brains, hands, and manpower to get some of these things done.

MODERATOR FAULKNER: Thank you, doctor. That concludes our session

. . . Noon recess . . .