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RESEARCH DISCUSSION

Chairman: John Seubert

J. SEUBERT: The purpose of this session is to informalize a little bit, to give people a chance to ask some of the previous program participants more detailed questions about their presentations, and for members of the panel to ask each other questions.

Up to now it has been obvious to many of you when you hear reports of research and research findings that the main conclusion is, "Well, we have found out this much, but there are still many unanswered questions, we can't see that they can be answered by intuition, and it is going to take basic work in physiology and behavior to answer them." Because of the shortage of manpower and money it is going to take a lot of good judgment to determine which areas of research probably will be the most productive.

There was a question asked Ed Schafer this morning about the Bureau's work with 1339. To elaborate on this a little bit, what we are working on in the Bureau today is a matter of priority that has been set by us in the bureau. These priorities are based on research projects which look the best at the time and on the resources available. Those of you associated with universities, I think, know that the golden days of abundant research funds have disappeared. Well, they have disappeared in many sections of government as well.

Dr. Woulfe and Bob Fringer have reported on Ornitrol and the work with black birds. The question came up as to the mode of action of this material. Maybe it affects the reproductive tract, maybe it depresses appetite, or maybe the bird just isn't getting enough nutrition so that it is in a position to reproduce. Again I think this points to the fact that answers to some of these questions relative to the mode of action need to be worked out in more detail in the laboratory before one conducts field trails. This is tough and expensive research. So compromise must be made between how much we do in the lab before we go in the field, and vice versa.

Well, with that little introduction, I will throw this open to, I hope, a rather informal discussion. I am going to start by asking Bob Schwab what his prognosis would be for the resources that can be brought to bear in California on some of the questions relating to the acetylated form of 1339.

R. SCHWAB: Well, first of all I'm no authority on this form of 1339. I was reporting on evidence that was obtained by Dr. Peoples. Most of my own work was done with the regular hydrochloride form of 1339. During the past two years, I have concentrated mainly on trying to discover what the fate of this compound is in water solution. Now the rationale behind this is that these baits have been, and are being, applied in a variety of forms. You can buy this material

in pellets as well as other prepared baits, and it has also been applied as a spray. Ultimately, because of this, these baits (if they are not picked up) are going to give a net effect similar to what we see with the chlorinated hydrocarbons put into our water systems. There are no two ways about this. Since 1339 is 3-cholro-p-toludine hydrochloride, this implies that it is a chlorinated hydrocarbon. Whether justified or not, this compound bears the stigma of persistence in the environment and concentration in the food chain. I say whether justified or not, because we don't really know about persistence in the environment . Consequently, during the last two years we have been trying to explore just exactly what does happen to 1339 in a water form, and also what effects some physical parameters in the environment have on this compound.

We've tried to explore certain very obvious characteristics such as: cold, heat, light, and how volatile this compound is. To make a long story short, we found that cold does not effect this compound whatsoever. It can be frozen in an aqueous solution again and again: and when it is thawed, there is absolutely no deterioration. Heat on the other hand is a little bit different. There is some deterioration or degradation with heating. If the temperature is warm enough, the compound in aqueous solution will begin to lose about 30% as a precipitate. We haven't checked this precipitate as closely as we should have to find out just how toxic it is. When a solution is put in a soil bath at 100°C, there is some degradation, but what is left is still biologically active. This has to be checked not only by photometric means but also by bioassay, because one or the other sometimes gives misleading results.

The compound oxidizes, and you lose some of it this way. To speed this up, one can put oxygen into a cylinder of 1339; again, an end result is a dark brown precipitate which seems to be toxic. But we don't have as much information on the precipitate as we should.

The compound does evaporate. This is where a lot of it is lost. Now if one goes back to look at some of these polychlorinated biphenols and some of the hypotheses that have been formed regarding the spread of DDT and DDE in the ecosystem, some people postulate that one of the causes of the wide distribution of DDT, is it's slightly volatile. Well, 1339 is also. As I see it right now, this compound vaporizes more or less in a non-toxic form. We have been unable to recondense this compound as a toxic agent. Perhaps, vaporization breaks it some how into a rather innocuous substance.

A number of years ago, Fish and Wildlife Service personnel published a paper about the effects of ultraviolet radiation on 1347, which was the freebase of 1339. In this report they discussed the use of an ultraviolet absorber, which apparently prolonged greatly the biologically active life of this compound under outdoor conditions. In our tests (with 1339) we attempted to degrade the compound with ultraviolet light, and we can indeed do this. We used ultraviolet (25-37 Angstrom units) at an intensity of 30 micro-watts per square centimeter, which is about 400 times the ultraviolet light that one would get under natural circumstances. The amount of degradation is a function of the intensity and the duration of treatment, under a given wavelength. But there is something funny going on in that the compound in an aqueous solution will change color. It becomes dark chocolate brown, but without any precipitate forming. Since the samples were

sealed in quart cylinders, there was little chance for any oxidation to take place. I would think this dark brown color would inhibit the penetration of ultraviolet, so you could only destroy so much of the compound this way. The point is that while one can destroy 1339 with ultraviolet light, such an intensity simply doesn't occur in nature. The natural degradation by ultraviolet is ineffective, I think.

To sum up, there is no effect by cold, there is some effect by heat, some by oxidation, some by volatilization, and some by ultraviolet. We find that just these physical factors of the environment will indeed degrade this compound and degrade it fairly rapidly. Because of this we are quite pleased with the compound, and it does not look like this compound will persist even in a relatively sterile aquatic environment.

We have run some other tests with soil and a certain amount of micro-organisms. I can't say anything about the concentration of this compound in the food chain, but micro-organisms are instrumental in detoxifying 1339 much more rapidly than the physical or nonbiological factors in the environment. You could have a tub of swamp water with some mud in the bottom and dump in 1339; and if there are organisms there, in a matter of days most of the 1339 will disappear. However, there are some problems because the compound tends to adhere to particulate matter, and this slows down its destruction. But it is bio-degradable; there is no question about this. Whether or not it concentrates in the food chain, 1 don't know.

I think the point here is that these results, admittedly incomplete and consequently inconclusive, show strong indications that here is a chlorinated hydrocarbon that is just a dandy. It looks like we would be able to put appreciable amounts of this compound into our ecosystems with good confidence that we have hurt nothing.

To go back to the introduction, it is appalling to me to find out that people are not interested in this compound anymore. If I had to start from scratch, or perhaps 1 should say characterize a compound that I would want to select as the ideal avicide, it would be very close to 1339. It seems incredible to me that the interest in this compound has simply died down. Few people that I know of are really working on this compound anymore, yet it is by far one of the best avicides we have ever owned.

J. SEUBERT: Ed, would you like to comment on some of the adverse effects of 1339 that we have detected, such as the phytotoxicity problem?

E. SCHAFER: I have a comment on some of the things that Dr. Schwab has said. You indicated that 1339 was not decomposed by ultraviolet light, it turned color but no precipitate formed?

R. SCHWAB: No precipitate formed. It was degraded, however.

E. SCHAFER: If you remove oxygen, you will get this precipitate. From what I have been able to find out, the precipitate itself is non-toxic, but it will include some of the 1339 which will give the resin on the bottom some level of toxicity.

R. SCHWAB: How did you determine the toxicity of this residue?

E. SCHAFER: Bio-assay. I have a lot of trouble separating it. I can't identify the components of the precipitate.

R. SCHWAB: Welcome to the club. We are having troubles with this, too.

E. SCHAFER: The vapor, formed from both 1339 and 1347 are colored compounds, we have gotten some to crystallize on the inside of the tube after vaporization.

R. SCHWAB: White crystals?

E. SCHAFER: No, colored.

R. SCHWAB: I get white crystals in my experiments.

E. SCHAFER: We have been hesitant to do a lot with 1339 lately. We feel the use for feed lots or in areas where you can control the bait, for example in grapes and grape vineyards, is very justified. Maybe pigeons in urban areas might be okay. However, we have been extremely concerned about the use of 1339 in agricultural areas where it is applied to cropland. The compound is phyto-toxic. I have a paper which will be published in about a year, I hope, on this.

One of the big problems we found is that 1339 is chronically toxic to birds. Now it is not chronically toxic in the normal sense of the word, because normally we think of a chronic toxicant as something like DDT that is stored in the body and then released under a stressful situation or accumulates to a point where it harms the organism. The damage caused by 1339 doesn't appear to be caused by the chemical being present in the body, but rather by the passage of the chemical through the body. It appears that some function of the kidney or liver is damaged, which with enough of this damage causes the bird to die. We have caused death with starlings down to one part per million fed over a seventy-day period. We have also done this with quail, pheasants, pigeons, blackbirds, and grackles. We haven't gotten down to one part per million with all these species, but on starlings we have.

R. SCHWAB: You mean one part per million day after day after day?

E. SCHAFER: Yes. One part per million takes about seventy days. Two parts per million takes about forty days.

R. SCHWAB: This is strange. A number of years ago in our starling report in California we talked about sublethal doses, and this is very conflicting evidence. We used much higher doses than l ppm.

E. SCHAFER: We have gone up to 50 ppm. You can feed a starling over a period of four days, and he will consume up to two lethal doses per day. If you withdraw treatment, the bird will survive; but if you continue for another day or so, the bird will die. One thing we did try in determining if cumulative effects were involved, was to put the birds on an alternate feeding schedule. They would be fed treated feed one day and untreated feed the next; treated food one day and untreated food

for two days; then treated food and untreated food for three days. No matter what schedule we used, if you fed the birds on alternate days it took twice as long to kill them than it would if they were fed single days. If they were fed every third day, it took three times as long. So the amount of accumulation of 1339 that the birds take in always came out to around fifteen lethal doses. As the feeding levels decreased, the total amount of chemical that was necessary to produce death decreased down to where at the 1 ppm level the bird required only about six lethal doses before he died. I don't know how far this would go down.

We are still working on chronic toxicity with 1339. We are doing some reproductive studies now with Japanese quail, bobwhites, and pheasants. This is the thing that has concerned us about 1339 and its use in agricultural areas.

R. SCHWAB: This is starlings we are talking about now, right?

E. SCHAFER: Starlings, pheasants, Japanese quail, bobwhites, and pigeons.

R. SCHWAB: Well, it certainly is strange that we get such conflicting results in these experiments.

M. DYER: Are there any closely related chemical compounds that you know of without the chlorine? Can these be synthesized? What is the toxic element in 1339?

E. SCHAFER: We tested probably one hundred analogues of 1339. The 3-chloro form, of course, is extremely toxic. The 3-fluoro derivative is even more toxic; 3-bromo is a little less toxic. If you go to the 2 substituted for chlorine, bromine, or fluorine, you get much less activity. It appears that the 3-4 grouping with a halogen and a methyl group is necessary for maximum activity.

M. DYER: So it is a halogen. The reason 1 brought this up is that I recently discussed PCB Compounds with Bob Heath. There appears to be a direct relationship with the amount of chlorine contained in the PCB and its toxicity. Is this the case with 1339? Are we involved with a material that is toxic because of the halogen involved?

E. SCHAFER: The 3-4 di-methyl form is also toxic. M.

DYER: Then it is not associated with the halogens?

E. SCHAFER: Not as much. You may be talking about 100 mg/kg of 3-4 di-methyl when we are talking about 3 or 4 mg/kg of 3-chloro.

M. DYER: Well, the only reason I bring this up is that this goes in with what Bob has said about getting these things into food chains. We must identify what chemical is getting into the food chain and which aspects are being transferred in a food chain.

E. SCHAFER: One thing that seems to be important is that the toxicity of compound 1339 points to interference with some enzyme system. The structure-activity

relationship of the molecule is such that you only get toxicity when the 3,4 positions are occupied. Usually the four position has to have a methyl group on it, while the three position can be, preferably, a halogen, but it can also be some other component, such as nitrogen.

R. SCHWAB: Well, Dr. Peoples believes that ultimately death results not from kidney or liver damage, but that 1339 is essentially a neurotoxin. He has some pretty good evidence for this. The bird dies in uremia but not because of it.

J. BESSER: Are nerve tissues this dissimilar that you get death at 300 mg/kg in sparrows and 3 mg/kg in starlings? Are birds' nerves really this different?

R. SCHWAB: I don't know. You'd think that if there were a genuine neuroeffect that it would affect mammals the same way, but there is no evidence for this. The reason I bring this out is that Dr. Peoples' evidence sets us back a little bit, because there is now some question about the mode of action of the compound. We thought we had it pretty well wrapped up, but apparently we don't.

E. SCHAFER: Well, again with 1339 the mode of action varies in different species. The compound is not toxic to sparrows and finches in the same way that it is to starlings and blackbirds. It causes death to sparrows or finches by central nervous system depression. The effect is such that you can put a sparrow to sleep with 75 mg/kg, and it won't kill him until you get up to about 400 mg/kg. The same thing happens with hawks. The action on mammals is somewhat similar, producing depression right before death.

J. SEUBERT: This confirms, I think, the earlier comments by others and myself that we are right back to questions about pharmacology and toxicology. I believe that you have seen that the Bureau has not lost interest in this compound. It is just that we have had to establish some priorities. One of the other reasons which gave 1339 a lower priority than some other materials near registration was the hazard to non-target forms in certain baiting situations, coupled with questions concerning degradation rates in the soil, and phyto-toxicity. Ed, wasn't persistence in soils of primary concern?

E. SCHAFER: There hasn't been too much done on this. You can grow wheat in a nutrient culture down to 5ppm active compound and you can kill wheat seedlings over a period of about two weeks. If you take the wheat seedling out of this solution, say after ten days and within about two weeks, it will catch up in growth to its sister control plant.

J. SEUBERT: Are there any other questions now about this material?

MR. WOULFE: Yes, how long does it take for the compound to be eliminated from the starling and what is the likelihood of the bird being seized by a predator who would then get a toxic dose? Do you have data to show that this is accumulating in predators?

R. SCHWAB: The life of this compound in an animal is very short. It is a matter of hours and not days. We have fed hawks on starlings that were given lethal doses of the compound and immediately fed to the hawk. We did not find any noticeable, or at least overt, effects when we did this for about four months. That is all the hawk had to eat, and it didn't seem to hurt it any. When the tests were done, we let him go. This was a crude test, but if something was going to happen, you would have thought it would have happened by then.

R. SMITH: I was going to ask what are the uses in agriculture that you anticipate other than what it is registered for?

R. SCHWAB: I anticipate spraying over a roost. R.

SMITH: Is this normally in an agricultural situation?

R. SCHWAB: Well, it depends on your definition of agriculture. I think a feedlot is an agricultural situation.

R. SMITH: What about ground crops?

R. SCHWAB: No, I don't anticipate spraying on crops at all.

R. SMITH: That's the major objection from the Bureau's standpoint at the moment.

J. BESSOR: I believe water has recently been defined as a crop, so if you are going to use this over water where most birds roost, I think you are going to be dealing with a crop.

R. SMITH: The point is the work we have done in the field here has shown this material to be as non-selective as strychnine.

R. SCHWAB: I have worked only with the starling, and I have to go by what's published on other species. And according to Fish and Wildlife Service data it is quite selective.

P. GRANETT: I am interested in the phytotoxicity angle. How was this done? Was it done on bait in the corn or on wheat?

R. SMITH: I think it was three years ago. We applied the material in one strip through a field at three day intervals. I don't know exactly what the concentration was other than that the materials were 2% toxicant in a one to ten ratio of treated to untreated corn. The farmer planted the field to wheat the next fall: the strip where we had treated was "retarded" as one said. The wheat came up, but it didn't grow.

R. SCHWAB: This was the following year?

R. SMITH: This was the same year, fall or winter wheat. I don't know what happened from there on.

E. SCHAFER: We planted wheat seedlings or wheat seeds in individual plastic containers of earth until about one week of age and then transplanted them into nutrient culture. We introduced into the culture amounts of 1339 calculated to yield anywhere from 100 ppm down to one ppm, and grew them for two to three weeks. We noticed phytotoxicity down to five ppm in the nutrient culture. We also did some work in soil flats with soil that had been pretreated with DRC-1339. Depending on the soil type and whether it was sterilized or not, we got phytotoxicity down to 100 ppm, which would indicate that 1339 is probably being bound on soil particles. It is fairly phytotoxic, however. Ten ppm I think works out roughly to about ten pounds per acre if you take six-inch furrow slices.

J. SEUBERT: I would like to move next to the work with chemosterilants, their mode of action, and other physiological stressing agents. Someone remarked that we all seem to have a hang-up on chemosterilants. It must be related to people problems, I guess. I think any agent that will stress an organism to the point of interfering with its reproductive process would be good to use, maybe something like an appetite depressant. Dr. Kare has worked with anorexic materials, and as I recall you can depress feeding in birds.

M. KARE: The thing that is exciting is that anorexic agents are absorbed through the skin of the foot. Food intake will be reduced 50 to 75% for three or four days. The aspect that is appealing about anorexic agents is that they are relatively non-harmful to other species. Since birds have a metabolic rate much higher than man's, they are much more sensitive to reduced food intake than are other species that accidentally get contaminated with the material or if it gets into their feed.

J. BESSER: How about between birds, Morley? I am thinking primarily of ducks which have a very big foot. Are they able to absorb chemicals at a very rapid rate? Would you ever predict specificity between those species?

M. KARE: Well, they wouldn't be superior just on the basis of their feet, but I have to use a parallel situation that doesn't answer your question directly. I recall sometime ago we were working with Patuxent on emetics. The pigeon is the standard animal for judging the efficacy of emetics. We got into this because pigeons were vomiting back some of the chemosterilants. We coated the material with aspirin which doesn't dissolve until it gets well down in the digestive tract. A comparative study of vomiting was undertaken. It included non-vomiting birds, those that feed their young by vomiting, and so forth. There was no rhyme or reason, as to how a species responded to emetics. For example, cowbirds, which normally do not feed their young, are the most sensitive to emitics. Chickens given massive doses gave no response. Pharmacological agents do not act uniformly even on closely related species of birds.

J. BESSER: I know that most of the dermal chemical agents that have caused trouble have been with rails, ducks, and shore birds, animals which are more aquatic.

M. KARE: Incidentally the critical thing is not the chemical itself; it is the carrier with which you disperse the chemical that seems to modify passage. In the case of

ducks you could look around for another carrier that would favor or predispose passage in those species that you are worried about.

J. SEUBERT: Dr. Woulfe, would you care to elaborate on where we go from here?

M. R. WOULFE: Yes, I agree with your comment about stressing birds in any way or form possible. Elder's paper back in 1964 recited a very long list of compounds that he tried out as chemosterilants, from the point of view of inhibition of egg production. He must have looked at two dozen compounds altogether, and indeed he did find a transitory inhibition by stressing. He found some other aberrations in behavior which accomplish much the same things.

I think when we hit on this compound the fascinating thing about it was the duration of action. The long half-life we have found to be unique; some of the analogues of the compound have the same type of action but not for this long duration. I think the point is well taken on disrupting normal behavior in any way that you can.

If I may take off, we should think entirely in different terms for the future in dealing with migratory birds and chemosterilants. Dr. Granett in New Jersey and Dr. Messerschmidt in Maryland have shown that the compound is active in grackles and redwings. But we get into this problem of distribution, and obviously it becomes important for the farmers.

I think we have to think in terms of dermal absorption and possibly inhalation. The idea of inhalation appeals to me to some degree, although dermal absorption is probably the safest route.

As an industry type, I have to think in terms of the total problem. I do not think that we have been given a definition of what the problem is. Is there a problem with redwings or is there only an annoyance with redwings? If you are a corn grower in Northern Ohio, obviously you've got a major problem: but it doesn't affect the farmer in Washington state or Los Angeles county at all. In other words, what is the total problem? What is the market for a compound if it is found? And above all, how do we get it to the target species? I know when I go back someone is going to ask me, "Well, where do we go from here?" Which button on the computer do I push in order to get the type of compound with what structure, active in what areas to do what? Well, I just don't know.

J. SEUBERT: Are there any questions from the floor on this matter of stressing agents and modes of application?

M. DYER: I have a comment about the stressing agents. We cannot always tell cause and effect. I can see how we can have lipid inhibition caused by several different agents that could be interpreted as a chemosterilant. This is a very complex situation in avian biology and physiology. There are no complete description of lipid metabolism that I know of today. Some physiologists are working on this subject, but there is much conflicting evidence.

When we take these compounds to the field, there is no way to assess cause and effect. There is simply no way that we can control the effect after the material has been put to use. It doesn't really make any difference what the action is if we get

a sterilizing effect. But if we have to control these effects and know what is actually being done, then it is too soon to go to a field application other than a small scale test. We must go right back to the analytical laboratory, and we've got to know whether the effect is lipid metabolism inhibition or appetite inhibition or whether it is outright starvation. I don't see any future for these particular compounds in our active work right now, except to discuss and experiment with them. Until we get more information, I don't see that we can use these compounds.

At Sandusky we can give you the population aspects-the population behavior and population dynamics. But if we don't know the mode of action in a test situation, we do not know whether the effects are toxic or chronic for the population.

M. R. WOULFE: But the mode of action-in what, the target species?

M. DYER: In any species. We don't deal entirely with redwinged blackbirds. We deal with all bird species, and we have both behavioral and physiological aspects to worry about. I recognize that when anything is first developed, you have to have some indication that it is going to be useful. That can be done from a simple screening program, and I believe this has already been done. But my main point is, after this fact has been established, then the mode of action must be determined.

M. R. WOULFE: Let me make another suggestion based upon the work in Florida about our knowledge of the physiology and particularly the enzyme system of some of these birds. With red-winged blackbirds and starlings it is rather limited. What would really speed up the whole process would be complete data on the physiology and something of the enzyme systems of these birds. Then let us develop *in vitro* tests so that we can incorporate liver enzyme destruction or whatever into the screening program. This would enhance the possibility of getting an active compound.

M. DYER: This is exactly what is needed, but now we are back to determining the mode of action in the laboratory rather than final effects in the field.

M. R. WOULFE: Well, I think some agency with the competency to develop these individual tests is going to have to make a start.

J. SEUBERT: This is a subject dear to some of our hearts. In the Bureau, we only have one biochemist working on micro-enzyme systems of the red-wing. We all basically agree on what is needed. I think the government is going to have to pick up the ball and work out some of the details on modes of action and efficacy of new products. Are there any further comments from the floor?

E. SCHAFER: Dr. Woulfe, I am interested in your screening test to find these reproductive inhibitors.

M. R. WOULFE: Well, originally it was found that the compound was preventing the onset of puberty and egg-laying in pullets. This was seized upon by Dr. Elder who said, "Well, if it does this to chickens, let's extend it to pigeons and to other things." That is how we fell into it.

E. SCHAFER: Then you haven't done any actual screening for compounds with reproductive effects on birds.

M. R. WOULFE: Not on birds, no.

J. SEUBERT: I'd like to make a comment here. Every so often people in industry stop in to talk to us about the market potential for a bird control agent. The first thing I tell them is that a good repellent that can be sprayed on crops, would result in a multi-million dollar industry. But, of course, there is a great reluctance to invest research dollars to fully explore this potential. It might not even be possible; but I encourage them, since most of them have certain dollars tied to basic screening tests anyway. Why not incorporate a rodent and a wild bird into the tests, which would be cheaper than setting up separate tests for these animals? To give an example of what we are trying to do in relation to the more applied aspects of our research, Mel, why don't you discuss briefly our studies of bioenergetics?

M. DYER: There are many ways to stress an animal, and one would be with an exogenous material which is taken inside the animal. Another is to stress the external portion of the animal, and this has been done as many of you know with wetting agents. In other words, it is possible to destroy the insulatives shell surrounding the bird, and he goes into thermal stress. The theory is simple, but then it becomes very complicated because the bird has some built-in adaptive mechanisms. There are mechanisms that can be employed where he can react to this stress and recover from the shock of the treatment, if it is insufficient. We have recently developed a method to test these things out.

But this development is not simple. First of all you have to establish what the standards are. There are light regimes, day and night rhythms, age and sex differences, and circadian rhythms to worry about. So we have a long way to go to establish what is "normal" in a bird before we can establish what will affect this "normality." There is no doubt that if we get enough water and low enough temperature on the bird that this is a very effective mechanism.

J. SEUBERT: I would like to move now to the area of the sensory mechanisms that Dr. Kare discussed this morning and maybe explore these a little bit further. Why don't you tell them a little about John Roger's work and elaborate on that?

M. KARE: John Roger has developed techniques for monitoring the passage of various materials into the feet of red-winged blackbirds. He uses isotopes, and he studies a variety of different chemicals in a variety of different carriers and measures how rapidly the material moves through the skin of the animal. I imagine he himself will be reporting this before too long. I think this is a useful technique. We have a constant supply of new animals-animals are used only once-and a wide variety of significant compounds can be tested.

J. SEUBERT: I suppose the basis for most of this research is obvious to most of you. In certain parts of the country, roosting blackbirds appear to be good biological targets. In a feed lot situation or in other baiting situations, it is possible to

achieve a high degree of success in baiting discrete flocks of birds. In other situations it is rather difficult. As one of the Bureau's objectives, we are looking for ways to control and/or stress certain roosting populations. To try to bait a large number of birds when they are on the breeding grounds would be an impossible job, because they are spread out all over the country side. On the other hand if one can get something onto the birds by the dermal route in a safe fashion, he feels obligated to explore this means. The problem is that you can't generalize between species and chemicals. You really have to conduct a long drawn-out process of screening.

R. COON: I can visualize, if anorexic materials work well enough to last three or four days and if the carrier is innocuous, spraying this over an upland roost so that it adheres to the trees. The birds come in and land on it; they don't eat for two or three days. If you spray it again, they don't eat for another two or three days. How good are the anorexic materials, and can you stress them with this and then stress them with another material?

M. KARE: We're talking on two levels. One thing: there are going to be a lot of imponderables when you take the chemical agents out into the field. I don't think you have to do it twice, because you would cut down the feeding 50 to 60% for two to three days. They won't survive under the pressures in the wild.

J. SEUBERT: Perhaps we could incorporate two or three different materials and get a combined effect.

DELEGATE: What about turning this around and getting an appetite stimulant that you could spray on that would get the birds to eat while they are in the roost, then you could give them a bait-a poison bait-of some sort. You mentioned earlier that the problem is to get starlings to eat in the roost. Is there some way you could give them an appetite stimulant to get them to eat?

M. DYER: I think the appetite is already there, but these birds are always playing "catch-up". There's only one time of the year when they have excess food reserves; and that is caused by hyperphagia right before migration during the fall when, for some reason, birds increase their intake of food and eat special types of food. Special types of fat are laid down at this particular time. If hyperactivity could be induced at times when food is not available, metabolism would be speeded up and the birds would starve to death.

J. SEUBERT: Maybe you could give them a stimulant like caffeine and increase their metabolic rate by 30 per cent. If they could not compensate for this by feeding, it would accomplish the same thing. You can let your imagination run wild here-what we were trying to do is develop control tools that are safe, humane and practicable as possible.

Let me give you an example, if someone should question you about your humaneness when they would say to you, "Well, how does it affect the birds-does the bird suffer?" We have to come back to the statement, "We really don't know." As far as freezing to death is concerned, we can anthropomorphize a little here and say for a man it's a pretty good way to go; it's like being drunk. Well, this may or may

not carry much weight, but if you have a rabies epidemic in a suburban community and a child had been bitten by a rabid skunk or a fox, I'm sure that you would find that most of the mothers in the community would be very upset. And they would not care how you got rid of those rabid animals.

DELEGATE: What about the time schedule?

J. SEUBERT: Harry Smith is in charge of the Australian Research Group and when the bird strike problem first occurred, people went to Harry and said, "We have a problem with bird strikes at airports." Harry said, "Fine, you furnish us the money, we'll work on the problem, but we won't guarantee anything." So the people said, "What's your time schedule?" This isn't a fair question. We do what we can, but this is on a priority basis. The development time is in relation to resources and sometimes luck.

DELEGATE: The problem here is when information from conferences such as this gets out to the people who are interested in these areas, and they read about all these fantastic new ideas that we're postulating, and they say, "Where are these products? We need them right now."

J. SEUBERT: We live in a free society. We could do this another way and not give out any information about our brain-busting-keep everybody in the dark-but I'm sure that would be more frustrating than the present system.

R. SMITH: I am interested in Dr. Kare's comments about pain in pigeons. Can you give us any indication of the various stages of pain in pest species, particularly pigeons, sparrows, and blackbirds? Can this be evaluated in terms of human pain?

M. KARE: Not very much is known about pain even in humans. Dr. Dukes at Cornell University got involved in slaughtering of cattle--some people were very upset about the fact that they were just cutting the blood vessels. He concluded that this method was no more painful than hitting them over the head.

We are very interested in the sensation of pain because it comes very close to the sensation of taste. Some of the species have their own level of pain. We have no one in our Center working on pain. I am aware of no scientific work on the reception of pain in birds.

R. SMITH: Could you go as far as to say that a bird in convulsions is not in pain? M. KARE: I have no

idea.

R. SMITH: The reason I ask is because these are the questions we are asked and we just have to say, "We do not know." I am just trying to come up with an answer for these questions.

M. DYER: A few years ago, I had some experience when I worked in Canada on the subject of what is discomfort in an animal. The field man can pass the buck by saying that if researchers are going to get anything done, we cannot worry excessively about the situation until we get more information. This kind of logic was received very favorably and we had very few problems. The basic research on this sort of thing is in its infancy.