MORBIDITY AND MORTALITY IN RECENTLY EXPORTED/IMPORTED BLACK RHINOCEROS

Evan Blumer and David Blyde

TAPE 4B 727

Blumer: To a large degree the empitance for this meeting has its roots in some of the issues that surrounded the morbidity and mortality of the recently imported animals. In a program that was developed between the government of Zimbabwe and the International Rhino Foundation [IRF], which previously was known as the International Black Rhino Foundation, the Foundation developed plans to help Zimbabwe carry out its National Rhino Conservation Strategy, which Mike [Kock] talked about a bit earlier. Among those initiatives in the Zimbabwe strategy that the IRF tried, and is continuing to try to support, was the establishment of some secure... **END TAPE 4B**

BEGIN TAPE 5A

...populations of the 001 subspecies that is found in southern Africa. At that point the predominant population of 002 was in Zimbabwe. The initial plans called for twenty black rhinos to be translocated to the *ex situ* programs, ten to the United States and ten to Australia. I whole heartedly agree with Mike, that after enormous delays which undoubtedly contributed to the eventual problems, ten of those black rhinos were translocated to the United States, and nearly a year later nine individuals were shipped to Australia. Within about six months of each shipment, three animals from each group would die.

To cut to the chase with some of this stuff, I want to just point something out here, so if I get too long winded we can see what I think is maybe one of the most important things. I have gone back and looked over the last 30 black rhinos that have been sent to *ex situ* programs from southern Africa, from Zimbabwe. There were ten animals sent to the US in '89, ten animals to the US in '92 and nine animals to Australia the end of '92, the beginning of '93. If you look at those animals, there have been eight mortalities. Of those eight mortalities, there were a few that really stuck out. One obviously, was this male down here at Western Plains. If you look at his liver, there was no pathology in his liver at all. Also, an animal that previously had been listed as undetermined, or possibly 016 *pneumonia*. I know that Dick [Montali] is not exactly sure how much he agrees with this, but if you look at the two different histopath reports, one from the Texas State Lab and one from the National Veterinary Diagnostic Lab, they list hepatocellular swelling and *bilestasis*, the same syndrome we are seeing everywhere else, as one of the underlying issues. They do not list this as the cause of death. They do not then see the degree of degeneration we are seeing in the other animals. But clearly, this is the most important thing. We are looking at seven out of eight animals. Some of these animals had a whole different array of what they may have been exposed to.

I want to strongly echo what Mike [Kock], and Nancy [Kock] and Dick [Montali] and others are saying here. Certainly there is reason to suspect some common exposures, and creosote has to be high on the list, but it is not the only thing we can look at. There are some things that do not fit with it. At some level I kind of joked, I hoped it was creosote; because if it is creosote we can fix it, we know what it is. If it is not, it is going to be some idiosyncratic reaction to some drug or to some other food toxin or something that just... you know some *mold that grew* and we are going to have one hell of a time trying to track it down. But I think that the conversations that are developing here yesterday, today and tomorrow are going to point us in the right direction. So that is the most important thing that I want to tell you.

Also, if we look at other morbidities that did not result in mortalites, there have been very few. In the 1989 shipment there was one male that knocked off its horn in transit, and that is it. In the 1992 shipment to the US there were no problems in transit, and the only other problems were two young animals at the McAllen Ranch in southern Texas. Shortly after they arrived they were given antihemeltics, and the female passed a five gallon bucket load of worms, is what was described to me. And then proceeded to decide to colic the next day. That passed after about 48 hours and she has done fine since. The male about six months later developed a rather acute and none responsive case of diarrhea. There was a bit of confusion because of some different veterinarians being involved and there not being a regular veterinarian there. But eventually that resolved itself, either because of or in spite of the efforts of the veterinarians involved. But, that is about it. So we are looking at no problems except for this liver situation--we have got to figure it out.

N. Kock: I do not know, maybe I am wrong, but these bomas in Harare that these animals were treated in are not unique in having been treated with creosote. It is an insecticide, it is important to prevent them deteriorating. I would think that any animal kept in Zimbabwe would probably have been held...

Miller: That is one of the questions that we need to know, because in talking with 048 the '89 Bass male we *incurred* a blood with 048 creosote compounds.

049-050? (basically inaudible)

Miller: So we need to track *some of those animals* 050.

Jessup: You are saying that you wonder if there was creosote exposure with those '89 animals?

N. Kock: Yes, I mean I just now remember those bomas, those were treated with creosote.

M. Kock: '89 animals were not exposed to creosote. They were caught in the Zambezi Valley then trucked on flat beds...

N. Kock: The bomas in the Valley, were not those..

M. Kock: No, 053

Jessup: If you consider those all to be possibly a part of the same syndrome, another question we might ask ourselves is, what other types of things are in common with animals that were treated? You are using some of the same immobilization drugs, not that the drugs themselves maybe a problem, but you have got carriers and preservatives in those drugs that ought to be considered. There may be similar

anthelmintics, there may be similar... I am not sure all what you ought to consider, but if you are really considering the same syndrome, perhaps they come from the same place. and maybe creosote is not the common denominator.

Miller: It may not be. I think what Dick [Montali] was saying, was in the US he has only seen this liver syndrome in animals like 060 The Dallas animal lived long-term to have a creosote exposure and then you see them dying of 061 and *bilestasis*.

Jessup: I think another thing you ought to do with some of those animals here is also look at some of their previous treatment records.

Miller: That is right. The one in Dallas is a fairly unremarkable history. It has been treated with bendazoles like other animals in the US. But certainly, it is why Dick [Montali] sent out the questionnaire, to try and pull things out 063 the telephone poll...

Jessup: But, I am not arguing that creosote is not your top differential. But it just seems to me that often times common denominators that are the obvious ones that we pick to look at, may not always be the common denominator at all--if they have a common denominator, which they may not.

Miller: Well, that was the interesting thing about putting *Schmidt* and the Dallas cases together. If you take the common denominator and look at those animals, it is not just something from Zimbabwe, it is not just something from 069 Epidemiologically it makes it much more of an interesting twist.

M. Kock: I think what people must understand is that when we had to move the rhinos into the bomas, we recognized that creosote was a problem, we knew it was. So we took precautions of thatching inside the bomas, as thoroughly as we could. What interests me is this young male that died in 072 The only thing that I can see about that calf is that it *was a cow and calf pair, there where two cow and calf pairs* that stayed 18 months in those bomas. And that is 18 months of exposure to creosote in the bomas. The only difference between those is they were most of the time kept in the largest boma, which is twice the size of the other bomas. Whether that means that there was less exposure. I do not know. But 076

Blumer: It was also at the end of the bomas too, which means there was not a series of walls.

M. Kock: There is no doubt we were concerned about the creosote, we really had no choice but to keep them there. We could have moved them back to the 079 bomas. but politically and logistically... These were creosoted walls, but they had probably three to four centimeters of thatching grass on the inside to protect them. There was definitely a smell of creosote, so there was definitely aerosolization of creosote. Nancy was concerned about that. The ability of the animals to actually get creosote on them was 083 minimal. But we did notice that with some of the animals that there would be a little of creosote on the forehead and stuff like that. So the actual contact exposure was absolutely minimal, but we were not able to prevent it completely.

Toit: You said Mike, alot of those animals 085 had big lesions on the rear. 086 skin heal on the flanks.

M. Kock: Only one animal and that animal is still at 088.

Toit: 088? (inaudible)

N. Kock: There were actually two animals. You were out of town a lot during that time and Keith was keeping an eye on them. But there were two animals with really bad lesions 090.

Blumer: Let us not get too broken down, this is stuff we have to have in our working sessions. I would just like to mention two other brief things about creosote. One is that it is clear that it does not have to be contacted, it does not have to be ingested. There is a lot of information about the volatile aspects of creosote, especially something like Naphthalene if that is an involved component, being involved in other animals where creosote toxicity has been documented. And one more thing, this is more of a question. There has been some question as to whether the crates that the '89 animals came in were made out of older, but creosote treated wood. Do you know anything about that?

Toit: It is just a thing that occurred to me as being a possibility, I am not sure...

Blumer: I do not want to belabor this point anymore than anybody else does.

Jessup: Linda [Munson] have you or Dick [Montali] looked at the tissues? Has a common pathologist looked at the tissues from both the '89 animals and the '91 animals? I mean hepatocellular swelling is a pretty common finding and I guess a lot of times it is written down and you may see it as an incidental finding. I just wonder if those are really similar cases or if they are similar lesions of a much less striking degree.

Blumer: Dick [Montali] thinks that the Bass male is identical. At least the Bentsen female may or may not be, and it is clear it is to a much lesser degree. It is just something that I as a nonpathologist thought was a real interesting commonality, that the one animal that we were not saying was liver disease as the cause of death also had... And it was described similarly by multiple pathologists.

Munson: And the lesions are more strikingly, just some slight hepatocellular swelling. They 109 in all cases

Blumer: I am going to try and focus on the shipments to the US in '92, and the Australia shipments in '92, '93. Dave [Blyde] is going to talk a little bit more about those. Really the problem began prior to shipment as Mike [Kock] and Nancy [Kock] and Raoul [du Toit] have spoken about. And I can not emphasize enough of the bureaucratic delays, so that these animals would be in these bomas in Harare for a long long time. It was well over six months, and they were supposed to be there for two to three weeks. This was after they were in the bomas in the bush for I believe three months. Is that right Mike? They were in 117 bomas for three months, the first group before they even went to...

M. Kock: We caught them in June and 118 December.

Blumer: The first time? Yes, so it was much too long, and if it does turn out to be any kind of toxic 119 that can be related to feeding or holding, or any of those things over there, these delays certainly exacerbated the whole situation; and everybody has to accept that. Two animals died in the bomas, those were well covered by Mike [Kock] and Nancy [Kock], as they said primarily the pathology were these bright green livers, these gross pathology significant intramuscular hemorrhages, and icteric tissues and serum,

hemorrhages also on some of the serosal surfaces. They really did not fit with any of the previous pictures that we had at that point.

In the US the problems began about one month after arrival. An adult female that was thought to be pregnant became very quiet, she stopped eating, but there were really no other signs. She had really increased effort in trying to eat, she seemed painful in trying to masticate. We were only able to get her to eat by hand feeding her moistened bits of alfalfa or lucerne, and a small amount of browse. She really did not take any of the browse that we tried, we tried probably about half a dozen species.

Stover: When did she get sick?

Blumer: She got sick here. And again, for those of you that do not work with rhinos, realize that you can not get a sample anytime you want, especially when they are fresh out of the bush.

Q? 138: Do we know if these guys get hyperbilirubinemia from fasting like horses do? Do we have any idea?

Blumer: I do not think we know.

N. Kock: *We got normal* 140 Mike did some time ago.

Miller: A couple of cases come to mind where they were terminal, where one case was a nasal abscess where they had not been eating 142 no, that is not a scientific study. But cases that are agonal and not eating from other causes we have not seen. You know, bilirubin may double or triple, but not these immense 20 -30 fold increases, no.

Blumer: OK, she appeared painful during mastication, we were worried about oral ulcers. Especially because historically in the old original organization of Fossil Rim, before my time, a couple of black rhino were brought in and died within the first year of horrible oral disease; so that thier gums were sloughing out and their were teeth falling out, and things of this sort. We also saw some pale mucus membranes and icteric sclera. We decided to immobilized the animal for an exam. We found oral lesions that we biopsied. Linda [Munson] came back with a diagnosis of the same syndrome that she will describe to you later. We were not able to get any good urinalysis, she would not let us get anything, so we catheterized her and got a clean sample. She was a plus four on a urine dip stick for bilirubin, but no signs of any hemoglobin. At that point her PCV was 33%, and the total bilirubin in the serum was 11.2 mg/dl and it was kind of mixed between unconjugated and conjugated. And we certainly suspected hemolysis, at least to some degree. Over the next few weeks...

Smith: How have you been handling the samples? Glucose, one of them there is 92, one is 121, one is 180, one is 120. Is that one 261 or 26.1?

Blumer: It should be 261.

Jessup: That is done in house, is that done...

Blumer: It is done on a small glucose can, so I would not rely on those.

Smith: I guess the question is whether the red cells were in contact with the serum for some significant time frame..

Blumer: It is quite likely that they were.

164-167 BREAK IN THE TAPE

Blumer: ...then she began to eat on her own. But at that point, skin lesions really began to develop. They appeared at pressure points and along the margins of the ears.

Jessup: Evan, before you get rid of that, can you just underline the pack cell volumes on that again?

Blumer: The pack cell volumes go 44, 45, 45...

Jessup: She got sick in June?

Blumer: Yes.

Jessup: So between April or in May there...

Blumer: Yes, she [got sick] about right here.

Smith: Based on what Eric [Miller] said, what is this 450,000 retic's. What do you think that is?

Miller: There have been some cases where people *counted*. I do not know if that is the lab or the stuff. In general we sent them off to *Stockton*, we have not had high retic counts. We 177

Blumer: And 177 with a number of different of laboratories here, and I would not put a whole lot of faith in the stuff that my tech did in house.

Paglia: We found about 10 to 15% Heinz bodies in a normal peripheral smear. And I think some people might mistake those for retic's. Because we do not see reticulocytosis.

Blumer: At this point she was pretty naive about doing rhinoceros blood. You can see that the skin lesions were popping up all over the place, at pressure points, the base of the tail, along the ear margins.

Toit: That ear lesion thing was very distinctive on one animal 184 the margins of both ears were 185.

Blumer: Was the skin beginning to slough off at the edges and ooze some serous fluid?

Jessup: What was the story on your animal?

Toit: It was a black rhino that had been held in the same conditions 189 part of the same shipment, but declining conditions to the extent that there was talk 189 take and release it in the wild. In fact we had five rhinos in that category which did die. So in addition to the mortality that occurred in the bomas 191-194

N. Kock: I have got the data on the other two that were sick...

Blumer: This is the urinalysis, a little bit more data here. This is when we catheterized her *you can probably see* a few more things. So the skin lesions began and at that point all four of the animals that came to Fossil Rim were still in the barn after their arrival. This animal was down a lot, so we decided to try and move her out of the concrete floor of the barn to a softer substrate in the paddock and possibly allow her to use a wallow. We moved her outside, her attitude improved, she ate moderately well, she used the wallows. But after several days we developed a pretty severe fly strike and myiasis [started] along the base of her tail. At this point the urine was still remaining positive for bilirubin, but not as elevated as it had been.

We talked to a wide range of people about what, if anything, we could do for treating those skin lesions. We decided to immobilize her to try a few things since she now had a rather moderate hemoglobinuria also, and we wanted to collect some clin-path samples. When we immobilized her at that point she was found to be probably plus four icteric, very pale mucus membranes, PCV was down to 14%. And unfortunately she had a very fowl smelling, bloody purulent vaginal discharge, which is probably what was attracting flies. Obviously, we thought that the fetus was dead, and might be possibly causing her to be toxic or septic; and decided to attempt to pull the fetus, which is one hell of a thing to try and do. We could not get the fetus past the cervix. We tried for a long time, and tried to section the fetus with fetotomes, but still could not get it out. So we decided to give her a rest. At this point we had an animal in standing restraint with a pack cell volume of 14%. We had had her in standing restraint for about two hours. We had to give her a rest. We gave her some drugs to maybe help her along with getting the fetus out and dilate her cervix. We decided to try again in the morning, along with a transfusion.

She did OK recovering from anesthesia, which surprised me. A 14% PCV is not where you want to be using too many drugs on them. She seemed fine most of the night. In the morning she was found agonal, nonresponsive, open mouth breathing. We were successful with the use of emergency drugs, and intranasal oxygen, and getting her up. But this animal was so depressed at this point that I was able to attempt to remove the fetus on her without any drugs. We still could not get the fetus out, and decided to focus on the transfusion.

We immobilized a male and collected almost ten liters of blood from him to do the transfusion. We started the transfusion, running it at the same time with some saline. About one half hour into the process, the animal again went agonal.

Blyde: Where was the catheter Evan?

Blumer: In the ear, because we could not get anywhere else. When she was down, we could not get her up, we could not get to a leg vein. That was our only real choice. So we were going in as slow as we could. That is what we had the saline for, to try and kept things flowing a little bit. So the animal died. It was kind of a weird death. She kind of began to seizure and just stopped at that point, and we ended up with our first dead rhino. Let me just add in one thing here. At this point we had been working with this animal pretty much straight for about 36 hours, and we were not suspecting all of these nasty lesions that we ended up finding. This was the first of the animals that went. So we did something a little novel. It was at the end of the day, so we opened up her belly and packed her with ice. We went to work on her the next morning. From the pathologists we talked to, the tissues actually were in fairly good shape. So if you get in that situation, it is something to consider if you have ice.

In the postmortem exam, the most obvious findings were this bright green liver. One thing we did lose was some of the color over the course of the night. Numerous hemorrhages in the muscle tissue, as people have been telling you, and on the serosal surfaces and also on the mucosal surfaces in some cases. We did not find in either of our animals any mucosal ulcerations. I think you guys did. And obviously a

whole bunch of icterus in the tissues. And obviously a dead fetus that was near full term, probably within days of full-term. It had probably been dead for 48 to 72 hours by the time we had got to it.

Munson: Did Dick [Montali] look at the liver of the fetus?

Blumer: I do not think so. I think it is real interesting to note that in past shipments, and you guys correct me if I am wrong, about 80% of the females have been pregnant, and there has been a fairly high birth rate of animals in captivity that were actually conceived in the field. Of these ten animals that came in, there were six females, and she was the only one that was pregnant. So whether the long-term holding caused some of them to abort or to reabsorb, or what ever, it is hard to say. But they were not in holding so long that they would not have been breed.

N. Kock: 274? (inaudible)

Blumer: OK, aside from the dead fetus, this same basic pattern has presented itself over and over. Other animals had slightly different courses of monitoring and therapy, do to some rather fortuitous findings. Our second animal, the male, was actually the blood donor that we immobilized for the transfusion for this animal. When the transfusion was not successful, and we basically had the bag [of blood] hanging there, Don Paglia flew out overnight to help us with things, he had been so involved. And as we were pondering what we were going to do next, we noticed that the large bag of blood had extremely icteric serum, despite the fact that the PCV was at 45%. Within less than two weeks, that PCV was down to 15% and the animal was dead. Postmortem findings were the same, the same basic course. Basically lethargy, and then they started spilling a little bit of hemoglobin into the urine, they started getting skin lesions, and then they died. And David Blyde may remember that shortly after this his boss John Kelly came to visit. I sent David a video message on the camcorder that said, if these animals are quiet, you can *sign* it. Because that seems to be the hallmark. Every animal that was a mellow animal ended up dying. I do not know what that means.

The third animal was here at White Oak. This also was a bit fortuitous of a finding. It actually ended up having an elevated titer to the *Bretisalava* serovar of leptospirosis. I am sure that Carol [Bolin] will talk about that a little bit later. We got those findings a bit delayed because a lot of the blood went to Don Paglia who had to processed it, and we had to send things on. Actually, we got this finding while there was a rhinoceros husbandry manual meeting going on here, which I did not go to because my second animal was dying. I called Janet [Stover] and she and Eric [Miller] were here and they went and collected the blood sample from their animal and found rather elevated bilirubins, and that began a long course of things here. It turned out that the animal did not have an active leptospirosis infection, but certainly was exposed to a different serovar than we had seen previously.

Another finding that has turned out to be really one of the keys to some of the hemolysis questions, as Don [Paglia] will tell you about tomorrow, is this finding of a sudden severe hypophosphetemia, along with a hypercalcemia. It does seem to be becoming one of the issues that links a few of these things together, and also is one of the issues that is at the heart of the diet study that was initiated with the folks from Purina. Bill Sadler will tell you about that and a number of the other things that came from this. The

bottom line is that it is a really horrible situation to go through this, but one of the things that it has done is that it has provided a tremendous kick in the ass to the people who were previously involved. It has inspired a number of new projects and new research initiatives. It has resulted in both the morbidities and mortalities, and just the *invocations* of these animals has resulted in a huge number of samples. I think Don [Paglia] has probably gotten more done and had more rhino blood in the last year and a half than he has had in the previous years of working on things. So we all have to continue to work together to try and crack these things and take advantage of the opportunities we have, whether they be good or bad. I am going to try and stop here so David [Blyde] can get a few minutes to say some things about the Australian animals.

Morkel: On reflection, with the quantity you have got, what treatment might you be able to give in these cases?

Stover: Nothing I can think of.

Blumer: I can not think of anything. Well, we came across a few things that have now proved quite useful in managing the hemolysis end of things. We started doing phosphorous supplementation and that has now had an evolution, and Don [Paglia] will tell you tomorrow this, we have done some oral phosphorous supplementation. And it has actually, we think, pulled an animal out of a chronic hemolytic situation. I do not want to go too far, but it may in fact be that the ability of phosphorous, we know that hypophophotemia can be a trigger of hemolysis in its own right, and it may be that the ability to remove phosphorous from tissue pools and storage in the body, may be what differentiates in the chronic animals, those that survive and those that do not. So certainly we have learned something about that. The liver, if it turns out to be ultimately liver disease, I do not know if there is anything we can do about them, short of trying to keep them alive long enough to allow them to maybe heal a little bit themselves, and we have not had much success doing that.

Munson: Yes, I do not think any cases have shown any response over time in improvement. They seem to be awful 340. I mean the case here at White Oak, there seems to be no change, so it is not like you are deriving out of it some palliative therapy.

Blumer: And certainly the first animal, we did not know what the hell to do, and the second animal we tried to learn a little bit from the first one. And Janet [Stover] took the next step. We tried to communicate those findings to David [Blyde] and the guys in Australia, and we are trying to make some progress. But it is like having a glass of water in your hand and a burning building in front of you, you just can not do anything.

Q? 346: Did anybody 346

Blumer: For a time.

Q? 347: So it does not sound like the *DIC with some of the hemorrhages*

Blumer: In the end it might be, but certainly not.

Munson: Yes, Larry was just asking about that too. You see microthrombi in the glomeruli of the DIC usually 349. In the case I looked at there was not.

Q? 350:? (inaudible)

Munson: Yes, a diathesis type of thing rather than DIC.

Blyde: We had nine rhino come from Zimbabwe in November of '92, and I guess one of the problems that we had was that we had to quarantine our animals for 60 days on an island out in the middle of the Indian Ocean, which was about 4,000 miles from the nearest mainland and the nearest *lodging*.. So we did not have a lot of things to work with. As soon as we unloaded the animals we noticed that the bull. we only had two bulls in the shipment, the last bull was quiet. And we were worried about his appetite. On day 15 we had to immobilized all these animals to take blood samples. When we did that, we noticed that he was jaundice. We treated him with what we had out there, which was basically antibiotics and vitamins and some anti-362, but he died after 40 days. And this first one was very similar to the ones that Evan [Blumer] spoke about, jaundice, hemorrhages, and the animal *just died, it was fairly* 365.

The second animal that died from the hepatopathy did not get sick until about a month after she arrived in Australia, and that was March of '93. And she left Zimbabwe in November of '92, so it was four months after she had been in those bomas that she got sick. I can basically say the signs, she was just depressed. The guys that were working the rhinos to me said, heh, this animal looks a bit depressed and she is not eating 371 She lasted about 41 days, same as the bull, and we treated her with everything. I talked to Evan [Blumer] about it. I do not believe there is any way you can treat these animals, they are going to die. I think though the best way out is when they start getting real sick, is to euthanize them. Because it is really sad to see those animals. They just push on and push on, they are really tough 377.

Blumer: One thing I would like to echo also, I think Mike [Kock] mentioned this earlier, when we brought the animals into the country, we collected all these blood samples initially for Don's [Paglia] work, and the serum went on to people like Steve *Stockton* who is doing the normal values stuff. These animals by every measure we had, for the most part, were clinically normal when they arrived. Now certainly the liver maybe had to lose a certain amount of function before you see any kind of *deprivation* of the serum chemistry or whatever it is. You have some stuff that epidemiologically would suggest common point exposures, because there are animals that have died here, and died in Australia, and died in the bomas, and died after release from the bomas in Zimbabwe. Yet, the actual scenario is to have a bit of a time lag, and we do not know what is happening in that gap.

Sadler: I will only throw a suggestion out, I am not a toxicologist, but if I remember, creosote is an organic compound that will probably be stored in the fat, and probably accumulate in the fat. And one possibility could be is as these animals left Zimbabwe and they are relatively in good condition, and they basically detoxified the toxin by storing it in the fat, which is not all that metabolically active. Now when they are stressed, and they are brought overseas, and now they are put in poor condition, one of the things they will do is they will shift their enzymatic system to now start using their backpack, because they are maybe not eating as well, or they are stressed. Which will then dump all of these toxins right back into the circulation of the animal. Now, I am not again a toxicologist on this, but that could explain the delay of why

these animals all of a sudden get a burst of these compounds; is that they have been stored and they just not metabolically been able to get rid of them, and out comes the fat, out comes the toxins.

Blumer: Well, we are looking at some fat. We are hoping that we will find something in there, whatever it is, whether it is creosote or any other toxin. Toxin is the issue, not creosote. The unfortunate thing is that if there was a point exposure, or point in common, these animals have died four to six months after that, and we may not find what was there initially.

Toit: I think there is a very important point that comes out in the observation that these animals simply do not respond to therapy. When rhinos are caught in Zimbabwe, say they are caught for shipment, and there is any suggestion that there is a loss of condition... In my experience the animals 411 As soon as an animal shows a little of depression 412 it dies. Unless it can be released. There have been a few cases 414 But I think that somebody should come up with a very strong issue in this workshop, that if animals 415 while pending shipment, and there is the slightest indication, any kind of duress, there is no messing around, no taxing back and forth about what you should do. They should just be moved straight to the wild and released.

Jessup: But you know Rauol, if this is a progressive liver disease... If you release them and they die out there, what is the difference? Have you really gained anything by that?

Blyde: I think once they *yellow* Raoul, they are going to die. It does not matter what you do with them.

Blumer: From this, we have had other jaundice rhinos that have not had this group of changes.

N. Kock: I do not know if this is going to help or not, but if you have a toxin that perhaps does some irreversible damage to the liver, it may take a long time for it to run 428 before it finally gets to the stage where it has to die. Maybe the reason some die this month, and some die six months later, may have to due with exposure. But, never the less, it does irreversible damage.

Blumer: But some, like the animal that bashed its head, have no signs of any...

Blyde: No, certainly, the other ones in Australia are doing fine

N. Kock: 432? (inaudible)

Jessup: The problem of postulating that, is that hepatocytes turn over reasonably quickly.

N. Kock: No they do not.

Bolin: They do not regenerate such that the morphology looks the same, that is why serosis happened and you get all of these nodules. They do not regenerate *in situ* in lovely plates.

Q? 440: But if you are saying that hepatocytes are dying over a period of four, five, to six months...

441? (mixed)

Munson: No, there is a threshold effect with hepatocellular necrosis. You have to have a certain amount of heptocellularcellular necrosis to trigger in that regenerative response. What we are seeing is not that.

Bolin: What Dick [Montali] was showing is that those are sick hepatocytes.

Munson: They are degenerating, they are not necrotic.

Bolin: You do not have fibrosis, large number of cells missing... They are dysfunctional, which is different than dead. I hope we all agree.

N. Kock: They are not 447 cells 447 but they are capable of it.

Munson: There is no histological evidence in the ones I have looked at, maybe Nancy [Kock] can address this too, of any regenerative response either. There was no histological evidence in the ones that I saw of any regenerative response, which again relates to treating them and trying to get them through this. I mean these cases that have been going on for months, there is not the slightest evidence of any regeneration. No fibrosis, no regenerating. Fibrosis is usually a response to necrosis though, and we did not see much necrosis, again it 456.

END

(457-470 inaudible mixed conversation)