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## Interview: Dr. Randall C. Cutlip

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# National lab researcher says: 'Mad Cow' disease test still needs more work

*Dr. Randall C. Cutlip is Research Leader of the Respiratory and Neurologic Disease Research Unit of the National Animal Disease Center, in Ames, Iowa. The center employs about 260 people, and is part of the Agricultural Research Service of the U.S. Department of Agriculture (USDA). The interview was conducted by telephone on April 18 by EIR Agriculture Editor Marcia Merry Baker.*

**EIR:** On April 9, German television publicized work going on at your lab, specifically, that done by Dr. Mary Jo Schmerr, on developing a test that might be used in the near future—perhaps year-end, or even six months from now—for the presence of BSE (bovine spongiform encephalopathy, or “Mad Cow” disease). Such a test could help to restrict the number of cows that would need to be culled, and still protect public and herd health. How far has this test been developed? What is the time frame?

**Cutlip:** We have received lots of questions about that TV coverage, which I haven't seen, but I think that, basically, what was said was correct. But there were a lot of qualifiers with it. The test is still in the development stage. You can't say when it will be complete, and be useful—if ever. That “six months,” I think, was in reference to the expected time of validation of the tests on brain material, where there is a known amount, or a known infectivity, of prion—the causative agent. We have no reason to believe that we will have a test in six months, or a year, or two years. We may never have one.

**EIR:** Is the nature of the work at present to measure the protein substance? [Referred to as “prion,” defined below.]

**Cutlip:** Yes. Dr. Schmerr is working on a competitive inhibition type test, where a peptide that is identical to parts of the prion protein is labeled with something you can identify—in this case, you use fluorescein. And you put antibody with it. The specific antibody will react with both prion and peptide—that's the competitive part of it. And depending on how much of the peptide is tied up, you can tell whether there is any prion there or not. If all the peptide is tied up, then you know that there's no prion there. If just part of it is tied up, then you know the prion is also attracting the

antibody. In other words, the antibody is sticking to the prion as well as to the peptide.

Dr. Schmerr is using the capillary electrophoresis system to identify the labelled peptide. So that's basically what the test is. And it works. She can take a piece of brain from a sheep with scrapie [spongiform encephalopathy—sheep], and identify prion. Whether it will work consistently, and whether it will identify extremely small amounts—it has that potential, certainly—we just don't know yet.

And there are other things we don't know about it. We are talking about a live animal test. We don't know where the prion is located in the live animal, and it has to be some place we can get to.

**EIR:** What have you been looking at so far? Spinal cord?

**Cutlip:** We haven't done anything except on brain. The test that Dr. Schmerr has working, is a test on scrapie, on sheep brains of dead animals. We haven't actually looked at any live animals yet. We've got tissue that has been saved back from certain animals that we will work with first.

**EIR:** There are press reports of a team at the National Institutes of Medicine in Bethesda, Maryland, working on a potential BSE test, which project that technicians could use it to test perhaps 2,000 cows a month. How long has Dr. Schmeer been working on a test, and what's involved?

**Cutlip:** Dr. Schmerr started work on her test several months ago. Almost all of these tests are rather labor-intensive. The test that Dr. Schmerr is working on doesn't take very long, but preparation of the material takes almost 24 hours. However, that doesn't mean it would take 24 hours per test. It's the centrifugation that takes so long, i.e., separating the prion protein from the rest of the material.

**EIR:** What about the pathogen that is called “prion” [pronounced pree-on, for proteinaceous infectious particle]? In the 1960s, there was the first discussion of protein particles with no apparent nucleic acid. Then in the 1970s, came Prof. Stanley Pruisiner, and others, who discussed “prions.” Then, in the 1980s, more became known about so-called “prion diseases.” Could you give some overview about this prion



*This cow was partially paralyzed in giving birth to a calf, but is expected to recover. Because of the current outbreak of bovine spongiform encephalopathy ("Mad Cow" disease), whole herds are being culled, for lack of a test that could determine which animals are actually infected. Such a test is in the early phases of development at the National Animal Disease Center.*

pathogen, and the various patterns of spongiform encephalopathy in different species?

**Cutlip:** The patterns of the lesions vary tremendously, depending on the genotype of the host, as well as the type of the prion protein. Prion protein is produced by the host. It does not replicate the way bacteria, or viruses, do. It is actually a product of the host, that is changed by another prion molecule that has invaded the nervous system. The invading prion is not replicated. The prion of the host animal is changed to look like the part that invades.

**EIR:** This seems to be the context in which questions about "species jump" could be put into perspective. There is a lot of sensationalist speculation in the media.

**Cutlip:** Each species produces, and actually, different animals within a species, can produce, a different type of prion. They have slightly different amino acid sequences. That's based on the genetic type of that individual.

**EIR:** In laymen's terms, could it be said that the reason to worry more about contaminated cattle product potentially infecting humans, than scrapie sheep product infecting humans, is that there might be more compatibility, or similarity on the amino acid level, between cattle and humans, than between sheep and humans? In other words, we have had scrapie for a couple hundred years in different places in the world, and we

haven't been fearfully giving up Easter lamb or mutton stew. But there is worry about BSE "jumping" to humans?

**Cutlip:** Prions of sheep and cattle are both fairly far away from human prion. Sheep and cattle are close. I think cattle is a little bit more *dissimilar* [to humans] than sheep. Cattle prion differs, I think, in about 30 amino acids from human prion. But that doesn't say that there's not a small section of it, a small part of it, that's quite similar.

**EIR:** And that's the interesting part?

**Cutlip:** Well, it could be. Nobody knows for sure. Theoretically, it could be important.

**EIR:** How long has scrapie been around?

**Cutlip:** The first description that appears to be scrapie, was about 250 years ago. In the United States, it's been here only about 50 years. The first diagnosis was in 1947.

It's in most countries. Australia and New Zealand claim to be free of it. It is probably, as far as incidence, as great in the United Kingdom as any place now. Not too long ago there was a paper published indicating that the incidence there could be as much as in the 30% range, or even 40%, of the flocks infected.

In the United States, there are somewhere around 30 to 50 flocks a year, that are officially diagnosed as having the disease.

The United States has had a scrapie control program since 1952. It is now a voluntary program. It is a little unpredictable, what is going to happen now.

**EIR:** I understand that USDA experimentation began around 1979, to see whether the scrapie agent was transmissible to cattle. Was your lab the location of that?

**Cutlip:** That's what we did. We tried to transmit, by injecting scrapie material (brains of sheep), directly into the brains of cattle. We did transmit it that way. The incubation period was 14-18 months; they died up to five months later. And the disease produced was not like BSE; whereas, BSE cattle are hyper-excitable, our cattle were very lethargic. The lesions are different: very little in the way of lesions in ours, whereas in BSE, lesions are very significant. And, the signs were different; and, the distribution of the prion protein in the brain was different: It just wasn't BSE.

**EIR:** Are there other lines of experimentation that should be pointed out? Did you try to transmit by ingestion?

**Cutlip:** Yes. We tried to transmit it by feeding raw scrapie brain, and rendered sheep with scrapie. And so far, in five and a half years, those cattle are still normal.

There was raw brain material one time, one feeding, when they were baby calves. And then we fed rendered material from flocks of sheep with known scrapie. We fed that for about a year.

**EIR:** On the issue of improperly rendered sheep parts, as is reported to have occurred in Britain in the 1980s, when they deregulated the conditions in the feed industry—lower temperature and pressure used in rendering. Do you think that the verdict is in, that that is what caused the BSE to show up in England? Or, do you think that conclusion is unwarranted, though, as a precaution, the looser rendering conditions should still not have been allowed?

**Cutlip:** I think there is a good chance that is how BSE got started. Actually, that practice was started in the United States. Before that, in the batch method, they were using organic solvents to extract fat. And there was concern, or pressure put on the rendering people, to stop that, simply because they were using some carcinogenic agent. It wasn't voluntary.

**EIR:** That sounds like the 1970s greenie movement; was it crazy?

**Cutlip:** Not necessarily. You don't want to be eating a carcinogen either, do you?

**EIR:** No. But I like experts—scientists, not fanatics—to take care of my food and feed supply.

**Cutlip:** Just working with it the way they were doing, was dangerous. I'm not sure of all of the solvents, but I understand

that there was benzene being used sometime—obviously, a carcinogen.

So they quit for that reason, and went to the continuous flow process. The heat isn't quite as high; they can adjust it. But, if you take protein like that, and heat it much above what they were doing, and you destroy the protein, then you reduce the values of feed. So it's a tight range that they're working in.

**EIR:** How is scrapie transmitted in sheep and goats?

**Cutlip:** The route of transmission in sheep isn't known exactly, but it's thought to be primarily around the time of birth. Sheep do eat placenta, as many animals do. Whether that's totally it, or not, nobody really knows.

Again, scrapie can be transmitted by oral route, that's confirmed. Whether the lamb picks it up from inhalation of placental fluids, or just exactly how, that's not really known. But there seems to be something that's associated with the birth-time that's important.

**EIR:** Why did BSE first show up in England? Why didn't it first appear here in the United States?

**Cutlip:** The risk factors are much less here. There are fewer sheep—less scrapie. The ratio of sheep to cattle is much, much less. There are over 100 million head of cattle and 7 or 8 million head of sheep in the United States. Whereas, in the United Kingdom, there are 11 million head of cattle, and something over 40 million head of sheep. The exposure there, was far, far greater than here.

**EIR:** Is the working theory that ingestion of enough contaminated stuff, where the risk factors were present, is sufficient to account for cattle getting the disease? Is the ingestion hypothesis still reasonable, even though the grouping of cattle that you are studying, in the experimental circumstances in which you are feeding the agent, may or may not show the disease?

**Cutlip:** It may be that we don't have the agent. If BSE came from sheep, we may not have the agent in the United States that is present in the United Kingdom. They have far more types of prion there than we do. Researchers have shown that in Great Britain there are at least 20 types. There are different types, depending on differences in amino acids in the prion protein.

Because scrapie occurs primarily in one breed of sheep in the United States, it's been speculated that there are only a few, or one or two types of scrapie prion in the United States. It is primarily in the Suffolk breed in the United States. What we have was imported from England, and it may have come in as a single type of scrapie prion.

**EIR:** Are there any "mad hogs" these days? Or other major food source animals affected? What about the zoo animals?

**Cutlip:** Hogs seem to be quite resistant. There have been

some in England, experimentally infected. Some of them were infected. I am not sure of the number; the study is still ongoing. But I don't think that any of them were affected by feeding material. There are some zoo animals in Great Britain that were affected. These are mainly *Bovidae* class animals. [Bovine, or ruminants, include: oxen, cattle, buffalo, bison, and close relatives.]

**EIR:** Has the U.S. changed the rendering process in recent years, given what happened in England?

**Cutlip:** Not that I'm aware of. They have stopped rendering sheep carcasses.

**EIR:** On the issue of feed: What about the advisability of increasing the protein content of feed by cycling in rendered animal protein product? Was there a big trend to this in the last 15-20 years, and not before that?

**Cutlip:** I think it has increased. It isn't nearly as great in the United States as it is in the U.K., because here we use a lot of proteins from soybeans as supplement, and they have less there. Soybeans cost more. Rendering and recycling by-products is lower cost.

**EIR:** What about the possibility of the 10 cases of non-conventional CJD [Creutzfeldt-Jakob disease, the human form of

spongiform encephalopathy, fatal and rare] now under study in Britain, having occurred because the people ingested something from the BSE-contaminated cattle product?

**Cutlip:** I don't know anything, except what I see in the papers and journals. The link is not absolutely proven, but you can't discount it either. It's a possibility. We won't know that until we have a little more time that passes; a year or so, and we should know.

**EIR:** Obviously, you can't carry out experiments on people by injecting them with the agent. But, what studies are you watching for results that are relevant?

**Cutlip:** Dr. Collinge's work in London. He's worked with mice homozygous for the human prion gene, and this is very interesting: He has a year or two to go on that. It will be interesting to see the results. These are transgenic mice—mice that carry the human gene for prion, and that have the mouse prion gene removed. And they were inoculated with BSE agent.

So far, I think it's been about two years, and none of them have died. If they live, it pretty well means that it is hard to induce BSE in mice with the human prion gene, and therefore it's not likely to occur in people with the same type of prion gene.

**EIR:** And your own tests?

**Cutlip:** The plan is to keep the cattle for 8 years. We've been following them for 5.5 years now, and we will keep them for at least 2.5 to 3 years more.


There are 12 of the ones that were being fed the rendered material. And 8 of the ones that were fed the raw scrapie brain material. The raw material was fed just one time, when they were one to two days old. The others were fed for about a year with the meat and bone meal, and tallow, or fat, that we had.

**EIR:** If there were plenty of money and proper support for public health and animal health, what else do you think we should be doing? What studies? What aggressive approaches against diseases? More compensation for farmers?

**Cutlip:** I think there are other approaches to a method to diagnose it. Particularly, there is a great deal of interest in looking at methods to identify the prion protein in food products, and so forth, and in medicinal products—products from cattle and sheep that are used for other purposes, than used in human foods. A test like that would be quite useful.

We also have some interest in looking at some other agents, particularly in mule deer and elk, that are very similar to scrapie in sheep, and see what that does in cattle. We plan to do that, probably this fall.

**EIR:** What about using irradiation? Is it the case that irradiation won't work for this kind of agent?



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**Cutlip:** That's what the literature says. That experiment was done quite a few years ago, in the 1960s or early 1970s, and there was no effect on it. I don't know of any more experimental work that has been done. Anything that would destroy the BSE agent would pretty well destroy the beef.

**EIR:** As a researcher, are you especially interested in any particular anomalous aspect of the prion diseases? Other animal types? Ranch minks? Is what's coming out so far, falling into line with your own animal prion studies?

**Cutlip:** I think it is. As far as I know, it all fits in; there is just so much that is not known about it. It's hard to make any predictions at all. I feel that it all fits very nicely with the prion theory, as I understand it. What we need to do, is to finally pin down what is going on, to find ways to control it.

It's not really a slow virus; it's not a virus at all, if the prion theory is correct. It's strictly an abnormal host protein that is infectious, in some cases.

**EIR:** There is controversy over what rate of culling is called for. In Ireland, for example, one animal diagnosed sick, meant the whole herd was culled—even presuming the problem was a common feed factor, and not transmission between animals. Whereas, in Britain, that approach was not taken.

**Cutlip:** How many should be culled? If you want my true opinion, I think that the British people who saw this disease early, namely, Dr. Wells, who first diagnosed it, and Dr. Wilesmith, who is the main epidemiologist working on it, and Mike Dawson, who worked on the experimental transmission a great deal—I think they've done an excellent job in understanding, and getting things under control as quickly as they did.

It was diagnosed in 1986, and by 1988, a ban on feeding ruminant by-products back to ruminants was in effect. The whole community got it under control there rather quickly, and, now, the incidence has been dropping dramatically. [The number of BSE cases peaked in December 1992; then declined to less than one-third of the peak rate, and is still declining. Overall, from 1986 to September 1995, an estimated 156,000 head were diagnosed with BSE in Britain, in more than 32,000 herds.—ed.] That's a remarkable control of a disease which we know very little about. I think they've done a good job. I know they are being criticized: I think it's not fair.

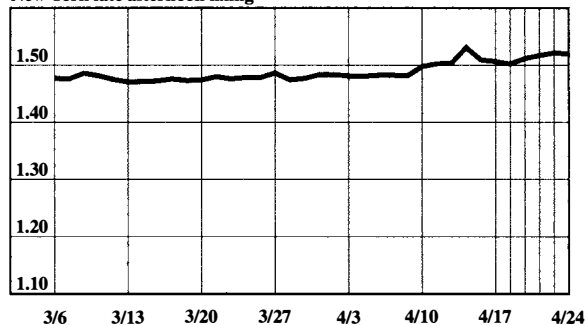
**EIR:** Would you differentiate between some of the epidemiologists, researchers, farmers, and others, as opposed to the ideological people in the Thatcher government from 1979-90?

**Cutlip:** They could have done a lot more. But in the early phases of this disease, they didn't know how devastating this was going to be. They probably looked at it, comparing it to scrapie in sheep, and it doesn't act quite the same way. I think it's been a remarkable control program effort.

## Currency Rates

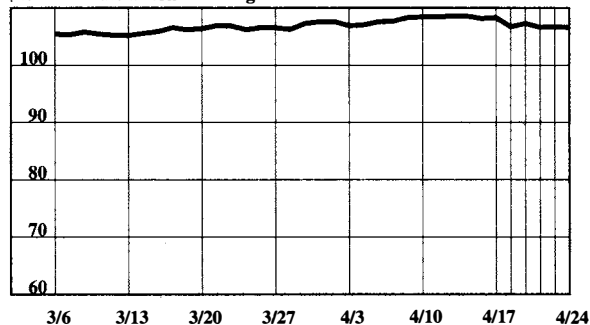
### The dollar in deutschemarks

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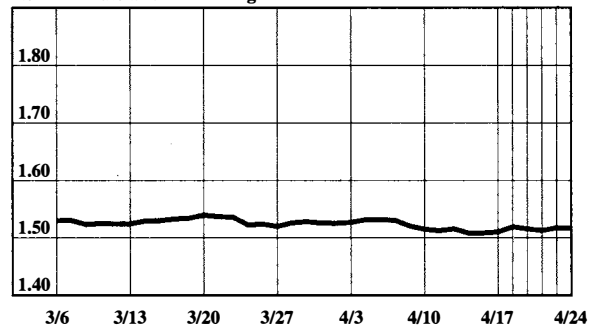
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