International Meat Crisis

The meat you eat
is contaminated and infected to a
degree never before known in human
history

Harvestime Books



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"Mad cow is the creepiest in a family of disorders that can make *ebola* look like chicken pox."

-Newsweek, March 12, 2001

Richard Lacey, a pioneer mad cow researcher, predicts that, by the year 2015, two hundred thousand Britishers will die each year.

—Richard Rhodes, Deadly Feasts, p. 222

Ground glass was in a shipment of burger shipped to four states. But federal law per- mitted the packinghouse to not tell state in- spectors which stores and fast-food restau- rants the burgers had been shipped to.

-(see p. 93)

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INTRODUCTION

In 1906, Upton Sinclair wrote: "This is no fairy story and no joke. The meat would be shoveled into carts, and the man who did the shoveling would not trouble himself to lift out a rat even when he saw one.

—There were things that went into the sausage in comparison with which a poi- soned rat was a tidbit" (*Upton Sinclair, The Jungle, p. 135*).

Sinclair told the facts about the meat industry. President Theodore Roosevelt read his book, *The Jungle*, and immediately ordered an independent investigation of U.S. slaughterhouses.

But the powerful magnets of the Beef Trust fought back. "Meat and food products, generally speaking, are handled as carefully and circumspectly in large packinghouses as they are in the average home kitchen," wrote J. Ogden Armour in the Sat- urday Evening Post (quoted in Skaggs, Prime Cut, p. 123).

After an angry legislative battle, Congress nar- rowly passed the Meat Inspection Act of 1906, a watered-down version of Roosevelt's proposals that made taxpayers pay for the new regulations.

Nearly a hundred years have passed since then; and conditions in the packinghouses are now, if possible, even worse than before.

But, in addition, a variety of horrible new dis- eases have emerged. For back in Teddy's day, cows

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ate grass out on the range and drank water from the creek. They were not, as they are today, born and raised in ponds of manure,—and fed ungutted dead animals in pelletized form as their only feed.

Times have changed. They are worse now.

HEART DISEASE

Heart disease and cancer are the two big- gest killers in the Western World. We now know a lot more about their causes than earlier.

- A large 1970 study analyzed the relationship between the dietary intake of saturated fat and cho-lesterol and the number one killer, heart disease. The study included 12,000 men in seven countries, including the United States. It found that **the two countries with the highest rate of death from heart disease were the two with the highest consump- tion of saturated fat and cholesterol**: Finland and the United States (A. Keys, ed., "Coronary Heart Dis- ease in Seven Countries," American Heart Association Monograph, No. 29, 1970, p. 211).
- In the mid 1970s a very large study was conducted by Loma Linda University in southern California. Because the eating habits of Seventh-day Adventists are higher than the American norm in whole grains, fruits, and vegetables, and lower in animal products, the diets of 24,000 were studied and compared with the diets of meat eaters (John Robbins, Diet for a New America, p. 215). Seventh- day Adventists who used dairy products (lacto- ovo vegetarians who do not eat meat but use

milk and eggs) ranked one-third as high in heart disease mortality as meat eaters. Adventists who ate no meat or dairy products (vegans) had a rate only one-tenth as high (Peter Cox, The New Why You Don't Need to Eat Meat, 1992, pp. 3-6).

- A 1988 published study of nearly 5,000 Brit- ish vegetarians found the death rate from heart disease of male vegetarians to be 44% of that of the British population. For female vegetarians, it was 41% (Cox, p. 8).
- Here in the United States, lacto-ovo vegetar- ians have cholesterol levels that are 14% lower than meat eaters. Vegans have levels (averaging 128) that are 35% lower (Erik Marcus, Vegan: The New Ethics of Eating, 1997, pp. 10, 14).

CANCER

In addition to cardiovascular disease, the other main killer in the Western world is can-cer.

- A massive population study, known as the China Health Project, concluded that those who eat the smallest amount of animal products have the lowest rates of cancer, heart disease, and several other degenerative diseases (Peter Cox, The New Why You Don't Need to Eat Meat, 1992, pp. 9-10).
- That Chinese research report interested the German Government, so they tracked 1,900 vegetarians for 11 years and found their death rate to be about half that of the rest of the population. The official report concluded that, in order to have a strong nation, everyone should become vegetarians!

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There were less than one-half the expected deaths from cardiovascular disease, in both men and women, and very low rates for cancers of the digestive tract. Most of the vegetarians studied used milk and eggs (*Food Chemical News, September 21, 1992, p. 10*).

Alison Williams was 20 years old and lived in the coastal village of Caernarvon, in north Wales, England. Bright and outgoing, she was a business student who loved to sail and swim in nearby mountain lakes. Athletic, attractive, and in-telligent; she had a happy life before her.

But, when she was 22, her personality changed suddenly. Her father recalls that she lost interest in other people, quit school, and came back to live at home with her parents. She would sit alone for hours, staring out the window.

By 1992, physicians diagnosed her as having a nervous breakdown. By 1995, she had grown para- noid and incontinent. "A month before she died, she went blind and lost the use of her tongue," her fa- ther recalls. "She spent the last five days in a coma." Alison Williams had contracted the human form of mad cow disease.

On July 11, 1997, Lee Harding ordered soft chicken tacos at a restaurant in Pueblo, Colo- rado. It was Friday night, and he and his wife were out to dinner. When the tacos arrived, Harding thought there was something wrong with them. The meat seemed to have gone bad. An hour or so after leaving the restaurant, Harding began to experience severe cramps. It felt like something was eating away

at his stomach. He was fit and healthy, stood six- foot-one, weighed two hundred pounds. But he had never felt pain this intense before. He lay in bed through the night fighting the cramps, tightly curled into a ball. Then came the bloody diarrhea.

At the hospital, he waited three hours before the doctor told him, "It's probably the summer flu." He was sent home with a prescription for an antibiotic. Harding kept sinking, but then it was accidently discovered he had Escherichia coli 0157:H7, a viru-

lent and potentially lethal food-borne pathogen.

Eventually the health authorities discovered the problem was not the tacos, but something in his freezer at home: frozen hamburgers he had bought at the supermarket and partly eaten Friday after- noon. In his freezer a Pueblo health official found a red, white, and blue box, marked "Beef Patties"; it still had a couple in it. Analyzed in the lab, that was where the E. coli came from. The burgers had been produced in Columbus, Nebraska—not in one of the oldest processing plants, but in one of the newest in the nation.

Jim Koepke spent his life as a ranch hand near Fallon, Nevada, tending cattle and sheep. He loved meat and also hunting. As a child he ate elk and deer killed by his father. His widow, Brenda, says that the 6-foot-1 cowboy shrank to less than 120 pounds before he died in 1999 at the age of 39. "I could carry him," she said. He died of mad cow disease.

Francis Will, of Evansville, Indiana, was 68 when he passed away. Local forensic pathologist

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John Heidingsfelder suspects it was mad cow be-cause, in the previous year, he has seen three simi-lar cases, one of which was confirmed by autopsy as mad cow. Yet, at the time Will died, the govern-ment still maintained that there are no human deaths from mad cow in America. Will loved to eat meat sandwiches; in fact he ate them every day. Doctors diagnosed his condition as various things, including anxiety and depression. But a daughter, Kathy Bredahal, a nurse working at a St. Louis hos-pital, suspected it was mad cow disease; for her father's symptoms were similar to those of a man dying of mad cow she had helped care for at her hospital. Eventually Bredahal arranged for her fa- ther to be seen by neurologists at Barnes Diagnos- tic Center. After numerous tests, they decided it must be mad cow.

ABOUT THE NEXT CHAPTER

QUESTION—How many prions can you count in the raw beefburger which forms the back- ground of the front and back covers of this book?

ANSWER—You can't. Only trained scientists in a well-equipped laboratory can tell whether the meat has tuberculosis, trichina, or a couple dozen other diseases. And they do not have time to do that. But not even researchers can know if an animal has mad cow, until it has lived long enough for the prions to fully destroy its brain. Yet prion-infected livestock are often slaughtered before that happens. And when one does die, its brain is not examined. Instead, it is sent to a rendering plant and ground into animal feed.

-BOOK ONE -

MAD COW DISEASE CAN KILL YOU

Public health experts agree that we are on the verge of an epidemic of massive proportions. Some say that it may, within a decade, be-come worse than the AIDS crisis. This is the story of mad cow disease.

BEGINNINGS

It all began in the Fore tribe, living in the jungle near Papua, southern New Guinea. That is where the modern epidemic, known as mad cow disease, got its start.

It was an area unexplored by Westerners until the second half of the twentieth century.

Although they had heard something about it a decade earlier, it was not until the early 1950s that scientists had opportunity to puzzle over a strange brainwasting disease in a tribe in New Guinea. The people there had a tribal ritual dating from the prehistoric past, in which they would eat their relatives, when they died, in order to acquire the mental and physical stamina they had while still alive. Women especially did this in order to increase their fertility. They thought it would help them have more children.

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Scientists found that many of the people in this tribe were dying of a mysterious brain disease which they, the nationals, called "Kuru," because it made its victims act very strange before they died. Kuru was killing up to 80 percent of the women in the tribe. (It was known that the women ate far more dead relatives than did the men, who primarily ate beans and sweet potatoes.)

Once the symptoms showed themselves, it took about 16 months before the victim died. There was a progression of tremors and an unsteady gait, followed by slurred speech, joyless laughter, and finally stupor, and death.

No one knew when the disease first started. Be- cause it occurred within families and mostly among women, researchers initially thought that Kuru was inherited genetically. But it has since been estab- lished that Kuru is infectious and was transmitted by eating the meat of those dead people.

Peoples in the South Pacific, as well as some other backward areas in the world, have had a long history of cannibalism. But the Fore tribe in New Guinea were remarkably consistent in their eating of dead relatives. This practice, continued for centuries, eventually produced a horrible new disease.

Not only horrible, but unique: The disease was not caused by bacteria, viruses, parasites, fungi, or any other regular means of infection.

KILLER PRIONS

In order to understand this, you need to know about prions, BSE, and CJD. First, we will con-

sider prions.

Scientists always used to think that infectious diseases could only be caused by bacteria. But the discovery of prions (pronounced *pree-ahns*) changed all that. It runs contrary to all the experts had been taught in the universities.

Although prions cause diseases, they are not viruses, bacteria, fungi, or parasites. They are simply proteins! Proteins, by themselves, were never thought to be infectious. Organisms are infectious; proteins are not. Or, at least, they never used to be.

But it is prions which cause mad cow disease. As we have learned, when researchers began ex-

amining the cause of a strange disease in the South Pacific, they could not find any pathological cause. By the 1970s, a variant of the disease had en-tered domestic and wild animals in Britain and America; researchers did not recognize the connection of this new disease with the earlier South Pacific disease which attacked humans.

Once again,

they could not find an infective agent.

It was thought that some kind of extremely tiny virus must be the cause—not a bacteria, not a mi- crobe, but a virus, a sub-microscopic speck of life. For decades, scientists had searched for unusual, atypical infectious agents that they suspected caused some puzzling diseases that could not be linked to any of the "regular" infectious or- ganisms. One possibility was that slow viruses— viruses that spent decades wreaking havoc in their hosts—might be the culprits, and these tiny viruses that were leisurely multiplying are hard to isolate. But the truth finally emerged. Here it is:

SixDefinitions 15

Researchers eventually, although reluctantly, ac-cepted the astounding fact that proteins, alone, could be infectious.

These strange proteins, called prions, enter cells and apparently change normal proteins within the cells into prions just like themselves! The normal cell proteins have all the same "parts" as the prions—specifically the same amino-acid building blocks.

There is just one difference: They fold differently. What does that mean?

As soon as a new protein is assembled by other proteins from amino acids within the cell, it folds into a certain pattern. But prions are proteins which fold into a different, incorrect pattern. That little dif- ference renders them deadly.

While other proteins always fold properly, prions are proteins which do not. That little variation makes all the difference—and it results in changes in the brain which produce holes—which look just like the holes in a sponge!

Prions cannot be destroyed by cooking, radiation, or any heat below $800^{\circ}\,\text{F}.$

SIX DEFINITIONS

We have explained what prions are. There are six other special words or phrases which need to be defined:

1 - **Bovine spongiform encephalopathy** (BSE). This is better known as mad cow disease. It is an infectious and incurable disease which slowly at- tacks the brain and nervous system of cattle. Spong- iform encephalopathy is Latin for "sponge brains."

- 2 **Spongiform encephalopathies** is the name given to this type of disease in various animals and in man.
- 3 *Scrapie* is the form of BSE which is found in sheep. The experts are divided on whether it is harm-ful to humans.

But when the dead animals are fed to cattle, BSE is transmitted. The term comes from the fact that infected sheep will scrape their sides and heads on trees, fence posts, or even barbed wire. 4 - *Kuru* was once epidemic in a certain tribe in New Guinea, because people liked to eat other

people. 5 - Drs. Creutzfeldt and Jakob were researchers in Berlin who, in the early 1920s, gave their names to a strange disease which occurred in one in a million patients. But its utter rarity and the total lack of possible causation permitted the condition to be ignored for decades, until Kuru was discovered.

6 - By late 1994, a handful of people in Britain had died from the same disease, which by that time had been named *Creutzfeldt-Jakob disease* (CJD). This is the name for the mad cow disease, when it occurs in people. (It is pronounced "Crewtz-felt Yahkob.") Although the disease existed as a rar-ity prior to the New Guinea research, it did not ex-plode on the world until after Kuru was investigated. 7 - *Alzheimer's disease* is a nonspongiform disease. It figures strongly into the present discus-sion because there is clear evidence that many people, dying in America and elsewhere from Creutz-feldt-Jakob disease, are being misdiagnosed as the victims of Alzheimer's. More on this later.

In summary:

BSE: Bovine spongiform encephalopathy-

This is the animal form of this disease. In cattle, it is called **BSE** or mad cow disease; in sheep, it is called **scrapie**.

CJD: Creutzfeldt-Jakob disease—This is the human form of the same disease. In New Guinea, the nationals called it Kuru; in the Western world, it is called CJD. These words will be repeatedly mentioned. You need to understand that BSE is the animal form of the disease and CJD the human form. In popular literature, they are both called "mad cow disease" or simply "mad cow." In this study, when we speak of BSE, CJD, scrapie, or Kuru, we are talk-ing about mad cow disease. The popular use of "mad cow" or "mad cow disease," as the name for the human form, will also be used. (In the U.S., vCJD, or variant CJD is also used. It is essentially the same.)

WHAT THE SYMPTOMS ARE LIKE

Whether it be Kuru, BSE, or CJD, patients first show symptoms of mental changes, such as problems with coordination, recent memory loss, and slurred speech. Sometimes obvious twitching of muscles can be seen, the facial expression becomes fixed, and the person may stumble and fall over. Over the next few weeks, the person becomes confused and unaware, unable to read or recognize even close relatives. The disease is very similar to Alzheimer's, yet the cause is very different.

Years later, it has been discovered that BSE in cattle, scrapie in sheep, Kuru in New Guinea, and CJD in the Western world all affect the same part of the brain and nervous system! It is the same disease, whether in animals or man.

Whatever variety of spongiform encephalopathy it may be, this is a disease of the brain and always fatal. There is no known remedy for it. Once a per- son contracts it, nothing can be done to remove the prions from his body.

Here is an ominous fact about Kuru: Research- ers discovered that it could take more than 40 years before the person became visibly ill. The disease bores into the brain and nervous system very slowly; but, once established, it rapidly causes dementia and death. No treatment works. Postmor- tems show the brain to be sponge-like and full of holes, hence the name "spongiform." Thus the dis- ease can work quietly, insidiously for years before any symptoms develop. How do we know this? Re- searchers found that Fore tribe women frequently died about 35 years after the last time they ate a dead relative, and some as late as 42 years.

PRIONS ARE DUMPED

You might wonder how a cannibal disease from one little tribe in New Guinea could get to Europe and America. We are not eating people over here! Here is how it happened:

Scientists eventually brought tissue samples home to America and Britain for careful examina- tion. But they found no antibodies and no disease germs of any kind. There was no microscopic lens in the early 1950s which could have identified the source of infection.

It is now believed that BSE went into the food chain, beginning in Britain and America, when those samples were disposed of. They were either flushed into the sewage system, tossed on garbage heaps, or washed down sink drains.

What those researchers did not know was that there was an infective agent present, one which no heat normally used in laboratories, for cleansing purposes, could kill. Prions which cause BSE and CJD are not destroyed by anything less than 800 degrees F. heat! This is far higher than autoclaving. The only way their sinks, for example, could be cleansed—would be to put them in a high-temperature bake oven for an hour!

Those prions from the samples laid on the ground for a period of time until they were eaten by grazing animals in the Western world. Then they passed into the food chain.

Trillions of prions spread on the ground, waited for some low-grazing animal to come munching to- ward them. In England, it was first noticed in sheep; in America, with wildlife and sheep. Previous to this time, CJD had been an extremely rare disease, oc- curring only once in a million cases.

But three facts changed the picture. (1) Tossing out those prions in the early 1950s, which spread an especially virulent form of the disease in the U.S. and Britain and laid the groundwork for what was to come. (2) The feeding of dead, diseased animal carcasses to cows, pigs, and chickens, which we will learn began in the early 1970s. (3) The eating of raw cattle and pig meat, as in the case of those who ate animal glands—a practice which began in the 1960s.

ANIMAL GLANDS IN THE 1960s

The next outbreak of CJD occurred, not in New Guinea, but from eating raw glandular ex- tract. (Warn your friends who purchase "glandulars" in health-food stores.) In the early 1960s, endocri- nologists found that one of the many hormones in the pituitary was the human growth hormone (hGH). They found that it could help dwarfes reach normal heights. Human cadavers were the only known source of hGH, and demand was intense.

So in 1963, the U.S. Government started a Na-tional Pituitary Agency, to harvest and distribute the glands. Over the next two decades, roughly 8,200 children were given the hGH through the agency. Similar programs were started in Europe.

But then, in 1984, four of the recipients, now in their 20s, were showing CJD symptoms. The program was stopped on April 20, 1985, after 27,000 children throughout the world had been fed the raw, dead meat. The number who will die from that program is still unknown. Additional deaths are regularly occurring at the present time. CJD can incubate more than 40 years before killing its vic- tim.

SCRAPIE IN BRITAIN IN THE 1970s

In the 1970s, it first appeared in the sheep herds of *Britain*. British sheepherders called it "scrapie" because the sick sheep had the strange habit of rubbing up against things.

Rams and ewes who had never met a cannibal started exhibiting an odd itch to scrape their heads and hides against fences,—even if the fences were

barbed wire. Frankly, the herders said the sheep acted a little crazy.

There were no antibody markers visible at any time during the incubation period, so veterinarians saw no indication of disease. Sick ewes freely gave their illness to their baby lambs who carried the bug straight to human tables.

To this day, there is still no certainty whether sheep with scrapie can infect humans. But we do know that scrapie sheep can, when eaten by them, infect cows—which, when eaten by people, infect them. The facts are hazy, since human dementia deaths in the 1970s were always ascribed to Alz-heimer's.

So now we have the answer to part of the puzzle. In the South Pacific, the disease was transmitted by cannibalism. People were eating their dead relatives. They contracted Kuru.

Later, a new form of cannibalism would be started in the Western world,—that would spread the "civilized" form of Kuru.

SCRAPIE IN THE U.S. IN THE 1970s

Mad cow disease (BSE) has been killing American sheep since the early 1970s, U.S. cows since the mid-1980s, and humans since at least the late 1980s. The reason it hasn't been made public is that those who had the facts chose to misinter- pret them. There is an extremely important reason for this: It could bankrupt the beef industry.

Something was started in 1970 which, in the 21st century, would eventually destroy the U.S. beef industry and kill millions of people in Europe,

America, and other nations which import beef from them. Here is what happened:

In 1970, the U.S Department of Agriculture and National Institutes of Health (NIH) collected thou-sands of scrapie-infected sheep, examined them, and carefully isolated the diseased animals in pens in upstate New York. Once again, they found no bac-teria or virus responsible for the problem.

But then, according to Howard F. Lyman, of the U.S. Humane Society (an ex-cattle rancher who was well-aware of what happened), the NIH sold the sick animals at low cost to farmers across the U.S.A., who put them into their herds. It was probably done as a way to help pay for the expensive scrapic research which had been completed. But it was the death knell of the meat industry in America.

Eventually as more sheep got scrapie and could no longer stand on their feet, they were then sold to rendering plants which powdered the carcasses and turned them into animal feed.

Upon eating the prion-loaded animal feed, more livestock contracted BSE. They, in turn, were made into more cattle, sheep, pig, and chicken feed. Which sheepherder wants to spend

\$500 for an autopsy on a dead animal, when he can sell it for \$100?

Did you ever hear of "feeder animals"? They are the animals you eat. In the U.S., there is an enor-mous industry based on turning cow corpses into animal feed, to be fed to "feeder cattle." The diseased, dead animals are ground up into pellets and, along with a little soy flour, are fed to livestock,

to fatten them faster. Because of the high-protein content, the pellets do this quite well.

Here is the procedure: Throughout the nation, whenever cattle, sheep, pigs, chickens, and turkeys die of disease, they are sold to rendering plants. Six- year-old dairy cows and two-year-old egg layers (chickens) are prematurely old and worn out, and are also sold. In recent years, dead and diseased dogs and cats are shipped from humane shelters to the same rendering plants.

At the *rendering plant*, the whole animal (in-cluding the intestines and the manure in it) is ground up and turned into "high-protein pellets." These are then sold as "animal feed" to cattle, sheep, pig, chicken, and turkey ranchers

When the creatures reach a certain weight, they are then hauled to the slaughterhouses/processing plants (often houses in the same large facility). The animals are killed on fast assembly lines at the *slaughterhouses* (which, we will learn later in this book, work too quickly to carry out the operation in a clean manner). The *processing plants* cut, wrap, package, and freeze the meat. It is then shipped to grocery stores, fast-food restaurants, and school lunch programs throughout the nation. Meat is also shipped overseas.

Carefully consider the implications of this: **These** "feeder cattle" are cattle raised on meat and soy beans. This turns cattle into cannibals! This practice is so solidly entrenched in America that you can actually trade commodity futures on "feeder animals."

The contents of the feed given to the live-

stock, and the way the animals are slaughtered, are sickening Americans. The powerful livestock industry is doomed to eventually be destroyed by its own practices. This is going to happen, even if they eventually wake up and stop the "feeder ani- mal" business. Among other problems, a major one is that the prions are now in the livestock, and each mother is passing them on to her young at the time of birth.

In summary, this is the formula: If the animal is in bad shape but still can walk, it is sent to the slaughterhouse. If it is too sick to walk or has al- ready died of disease, it is sent to the rendering plant to be turned into pellets to be fed to livestock.

BSE IN BRITAIN IN THE 1970s

Back in the 1970s in Britain, the sheep that had died of scrapie were rendered into powder and put into animal feed. But the Brits were just copying American frugality, as they used their dead sheep to feed the living ones. No one seemed to be con-cerned about the fact that the sheep which had died of a mysterious disease were being fed to healthy sheep which did not deserve to die the same way.

So Brits happily ate their sheep, little realizing they were eating cannibals. The curse that destroyed the people in Papua was passing to them and, through livestock shipments, to the whole world.

As the years passed, British sheepherders continued losing more and more sheep to scrapie. But they kept cutting their losses with cash for corpses sent to the rendering plants.

Trusting British beef farmers bought hi-protein certain-death feed for their cows for the next 18 years. Because the UK had a much higher percent- age of sheep than they had cows, every cow got a daily, heaping serving of kibbled sheep. And poor, trusting Brits ate a lot of the infected sheep too. The British like mutton as well as beef. For the first time outside of New Guinea, humans began contracting prions on an ever-increasing amount in their brains. It should be understood that neither farmers nor butchers fully recognized the problem. At beef slaughter time, the dementia generally had not fully manifested itself. The prions were in the animals, but they had not lived long enough to show the symptoms.

Even if they had, it was not until 1974 that the top UK microbiologist/researcher, Dr. Rich- ard Lacey, and his U.S. counterpart, Dr. Stanley Prusiner, set up their electron microscopes to study prion diseases. Until they did that, research- ers thought prion diseases were merely genetic in nature, just weird malformations which occurred from time to time in nature.

The truth is that the damaged proteins (the prions) were not only injuring the bodies of the sick animals, but were passing into the chromo-somes—and becoming part of the DNA of those animals and their descendants.

It should be kept in mind that part of the problem is that animals are generally sent to the slaughterhouse before the symptoms of mad cow disease show themselves. But the animal can still have mad cow, especially since he was raised

on a ration consisting of dead mad cows and sheep.

BSE IN BRITAIN IN THE 1980s

BSE (the animal form of mad cow disease) has been epidemic in British cattle since the late 1980s. The first confirmed cases were reported in late 1986; but it is believed that the first case may have oc- curred in the county of Hampshire in 1985.

In 1985, British farmers noticed that an ill-ness suspiciously like scrapie turned up in a cow. It was a Holstein dairy cow who started kick- ing, developed an extreme case of the jitters, then fell over dead. Her brain was examined posthu- mously, its Swiss cheese appearance noted, and the disease given the name "bovine spongiform encephalopathy" or BSE. For the first time, the disease in animals had been named.

In a cow, the bug caused more than just an itch to scrape against fences. **BSE was a true "Demen-tia" disease, like Alzheimer's is for humans**, *i.e.* memory loss, motor function changes, loss of large movements like walking ability. Eyesight and the ability to make fine movements with the hands were lost, as well as spacial perceptions needed for park-ing a car, etc. A lot of that is not crucial to a cow, but it was hard for the farmer to milk Bessie when she was splayed on the ground shaking and mooing.

A cow is a lot more valuable than a sheep. So beef farmers demanded answers. At first, no- body connected spastic cows with the scrapie sheep of the 1970s and certainly not with New Guinea can- nibals of the 1950s. But in 1986, a research pro- fessor of microbiology at Leeds University, con-

sultant to the World Health Organization (WHO), Dr. Richard W. Lacey, announced that scrapie, BSE, and CJD were the same thing; and that this beef disease was in the meat supply. In addition, he not only said it could kill humans, but he warned that a wave of deaths would soon hit Britain.

Immediately, the Establishment set to work to destroy Lacey's conclusions, and even his charac- ter. He was said to be a shoddy researcher and op- posed to the best interests of the British people. One publication called him "an airy-fairy, politically sus- pect vegan." Another said he was trying to dismantle the 6-billion-dollar-a-year British beef industry.

The funding for Lacey's research was canceled. But, refusing to give up, he warned that there would eventually be a fatal outbreak that would kill many Britons. In a nation whose economy was heavily keyed to beef production and its overseas export, he said people should stop eating beef and the newspapers should start warning people of the possibility of human infection. Lacey went still further and said that 100,000 people in Britain were already infected.

Something had to be done. Beef eaters were becoming worried and beef farmers were frightened.

Three things were done to solve the problem. First,
Dr. Lacey was fired from his research position at Leeds
University. Second, the government established an Official
Advisory Council. Of course, they left Lacey, the nation's only expert, off the board. Third, the government told the farmers not to worry, that while feeding powdered sheep corpses

to live cows probably wasn't a good idea, Brit farm- ers could do as they wanted. After all, had not the idea been given to them by American ranchers who regularly practiced "the grisly, fleshly humus pile" method for buffing up beef for huge profits.

The year was 1986. Brits happily went back to eating their cannibal-cattle burgers and steaks, and the beef farmers went back to their reward- ing task of supplying them with scrapie-fattened cows to munch on.

Meanwhile, Richard Lacey set to work writing a book on the subject. It was with difficulty that he was able to continue his research; but fortunately, he already had a lot of data in hand. Some friendly researchers also provided secret help. We will dis- cuss the findings of his book in more detail later in this report.

BRITISH BEEF STATISTICS: 1987 - 1994

In late 1987, 700 BSE-infected cows were reported in Britain. By the summer of 1988, the number had climbed to 7,000. Out of one side of their mouth, the experts said they were stumped. Out of the other side, they quietly passed a 1988 law, making the use of sheep and bovine offal ille- gal. ("Offal" is the waste parts of an animal. It in- cludes the intestines, manure residues, and diseased organs.)

But when Europe, Asia, and America heard about this law, they realized the livestock they had been importing from Britain was infected. Immediately they boycotted British mutton and beef, causing millions of pounds sterling profits to vaporize.

Unfortunately, this was a case of too little too late. British livestock were already grazing in every country of the world, and had entered the breeding stock of nearly every nation on the globe. The entire world had been eating imported British beef and lamb chops ever since the disease was sol-idly in place in the 1970s

The world ban on beef and the 1988 law against grinding up sheep did not stop the pro- gression of BSE in England. Cows kept dying. The number of infected dead cows soared from 1988's mere 7,000 to 36,000 in 1992. In eleven years, 160,000 British cows had gone four hoofs to the sky and there still was not an official murmur about human contagion—aside from Crazy Lacey whom no one took seriously.

As already mentioned, the first confirmed cases of the bovine form of the disease (BSE) were reported in late 1986; but it is believed that the first case may have occurred in Hampshire in 1985.

By late 1994, the disease had been identi- fied in nearly 150,000 animals and in just over half of all the cattle herds in Britain. Some sci- entists (including Lacey) have since stated that the only way to tackle the problem would be to destroy all herds which had cattle incubating the disease. The problem is that the ground would continue to have prions in it.

By the 1990s, news of the deaths from the human form of the disease, CJD (Creutzfeldt- Jakob disease), began to enter the public press. More on that later.

THE SOUTHWOOD COMMITTEE

The British Government had been forced into an investigation it did not want! A lot of money could be lost. So it told expert scientists, including its own advisers, to keep quiet lest the hugely profitable meat industry suffer.

In May 1988, the government set up the Southwood Committee, to examine the risks of BSE to both animal and human health. Yet, amaz- ingly, no experts on spongiform encephalopathies were appointed to that committee, and none were consulted! Lacey, of course, was ostracized. Although experts in their own areas, not one of the members of the Southwood Committee had ever done any re- search into spongiform diseases.

In June 1988 after the first meeting, the government, on the advice of the committee, or-dered the compulsory slaughter and destruction of the carcasses of all affected cattle. But it was already too late. Between the date of the first known case of BSE in late 1986 and the middle of 1988, at least 600 obviously diseased cows (plus an un-known number of animals not yet obviously ill) had been slaughtered; and their meat had found its way onto supermarket shelves. Since they received half the normal price in compensation for the car-casses, the hard-pressed farmers were thus en-couraged not to report suspect cattle to the government. The real extent of the problem remained unknown.

The second recommendation of the Southwood Committee was to set up another committee to do more research. But it announced that the problem

was too big for them to handle. Those learned men did not want to be ostracized, as Lacey had.

Elsewhere in the Southwood Report was the admission that spongiform encephalopathies may be a danger to humans and stated: "With the very long incubation period of spongiform encephalopathies in humans, it may be a decade or more before complete reassurance can be given."

The Southwood Committee then stated their theory about the possible ways the disease could be transmitted. Eating the meat was listed as one of the least likely causes. While admitting that all cows had contracted BSE by eating, they were saying that people could not also get the human form of the disease (CJD) by eating. They were suggest- ing one rule for cattle and another for humans.

Two other general conclusions of the South- wood report were these:

- (1) They declared that the risk of *vertical transmission* of BSE (that is, passing the disease from mother to calf) was non-existent. That has since been proven incorrect. Cows can pass spongiform dis- ease to their offspring. It can pass through milk, and through eggs. This is a key point and of the highest significance. **Not only can cattle pass the prions on to their offspring, people may be able to also.**
- (2) Cattle would eventually be shown to be a "dead-end host"; that is, the disease would stop at cows but not infect other species. However, that theory would introduce the revolutionary, new bio- logical concept of a non-infectious infection! Cattle are not dead-end hosts. BSE has been spread from one species to another, and this was known

at the time the Southwood Report was issued.

The report added this ominous statement: "If our assessment of these likelihoods (of possible human infection) are incorrect, the implications would be extremely serious." Their assessments have been shown to be incorrect. And that means we are confronted with a terrible crisis.

THE TYRELL REPORT

A second report—the Tyrell Report—was dated just four months later than the Southwood Report, but was not released to the public until January 9, 1990—7 months after it had been printed. Its conclusions have been largely ignored by the British Government.

For example, this report asked that the brains of cattle, normally sent for slaughter, first be checked to see which ones might have BSE. This would have shown how big the problem really was. Not sur- prisingly, this has never been done, despite nu- merous requests from the UK Parliament. The reason for not doing it was that it would be "too expensive." Too expensive for the people contract- ing the disease or for the meat industry? It was rec- ognized that if consumers ever discovered they were buying infected meat, the meat industry would lose its vast profits.

The Tyrell Report also recommended monitor- ing all UK cases of CJD for 20 years (as a matter of "urgency"), to reassure the public that there was no public health link with BSE. At present, "monitor- ing" only means that a researcher checks death cer- tificates for CJD! **No real investigation was ever**

planned because of what would be revealed.

The Tyrell Report concluded with the comment that additional research was needed; and that current controls, to keep the disease from spreading, were not adequate.

All in all, the report was a fairly good analysis of the situation as it was in 1989. Unfortunately, many of the proposals it made were ignored by the gov-ernment.

Officially, by this time the government was telling beef purchasers everywhere that it was not known whether the disease could pass from cow to calf, whether it was possible for other species to contract BSE, or whether the recent increase in sheep scrapie could be a possible cause for the increase in BSE cases in cattle.

The name of the game was to stall for time; all the while the citizens of the land continued happily chewing their beefburgers and steaks.

Although the official position of the government was that BSE was about to disappear; neverthe-less, in April 1990, it quietly made the Tyrell Committee "permanent." Leaders in the British Government knew they were sitting on top of a time bomb, and they hoped they would all enter upon retirement before it exploded.

THE BAN ON ANIMAL PARTS IN FEED

In order to make the most money, the meat industry throughout the Western world feeds meat to livestock. All leftover bits of animals from slaughterhouses, unsuitable for human consumption, are boiled up to produce fat and protein. The

protein is placed in the animal feed.

Apart from the obvious high risk of different infections being passed on, it seems strange that nobody had actually questioned the biological sense of forcing naturally vegetarian animals to become carnivores, eating the remains of other animals! Both cows and sheep have several stom- achs and long intestines, so they can digest grasses. They should not be given a meat diet!

In June 1988, the British Government im-posed a six-month ban on feeding animal pro-tein to cows and sheep. It was thought this was the most likely way the animals were becoming in-fected. In December, the ban was extended for 12 months; and laws stopped the sale of milk from cattle suspected of having the disease.

But banning infected feed did not stop the rise of BSE. Cases rose from 500 per month in January 1989 to 900 per month in December 1989.

The number of BSE cases per month rose from 800 in January 1990 to 1,500 in December 1990. Yet the Southwood Committee had predicted a maxi- mum of 400 cases per month.

JUMPING THE SPECIES BARRIER

For four years, the British Government reassured the public that BSE could not infect other species. But tests carried out in February 1990 proved the opposite. It was discovered that BSE could be transmitted to mice by feeding them contaminated meat, and it could be passed to other cattle by injection. Cattle were no longer "dead-

end hosts."

The disease had never previously been reported in cats; but, in May of the same year, a domestic cat died from a spongiform encephalopathy. However, in spite of such evidence, the government continued to deny that spongiform encephalopathies could jump species. In fact, that is the very nature of the disease. But by the time 52 other cats had died in July, the government finally admitted they had contracted the disease through eating pet food. As this report is written, over 80 cats in Britain have died of BSE.

The question was no longer "Can BSE affect other species?" but "How many species will it affect?"

THE CRISIS IN BRITAIN DEEPENS

A month earlier, in January 1990, trading stan-dards officers in charge of the cattle yards re- vealed that infected cattle were still being sent to market because farmers were only being given half of the normal price for their cows. In response, a Ministry of Agriculture official denied that BSE was finding its way into our food, but some people were becoming more worried.

In April 1990, Humberside County Council ban- ned the use of British beef in school meals. The number of known cases of BSE had passed the 10,000 mark. In April 1991, the Ministry of Agri- culture predicted that a peak in the number of BSE cases would occur that year and the dis- ease would disappear by 1994.

But, by the end of 1991, 25,025 cases had

been confirmed in Great Britain, providing the first indications that, despite government claims to the contrary, the disease was being passed from cow to calf.

MORE EVIDENCE OF SPECIES JUMPING

In 1992, BSE had been transmitted experimentally to seven out of eight mammal species, including pigs and marmoset monkeys. In four experiments, this was done by eating.

A puma and a cheetah were also reported to have died of the disease. Evidence was mounting of an uncontrollable epidemic, with serious implications for humans.

VERTICAL TRANSMISSION

By 1994, more than 17,000 cases of BSE were confirmed in cattle born after (after) the feed ban, with 500 cases known to have come from mothers which later developed BSE. This meant that BSE was infecting cows by means other than in-fected food. However, the government tried to explain this by blaming farmers, feed compounders, and renderers for breaking the law. They were accused of continuing to put ground-up sheep and cattle into cattle feed.

But that was only an attempt to deny the fact that vertical transfer of BSE was taking place. The mother cows were passing BSE to their calves in the womb. The existence of vertical transfer means that the infectious agent must be in the cow's blood and will therefore be found in virtually all parts of the animal—all beef prod-

ucts.

By 1994 the government had still taken no action to control cattle being moved from BSE infected herds to other herds, nor had they taken any other steps to control the epidemic. The total number of confirmed BSE cases exceeded 137,000 by the end of August 1994. This was more than six times the number predicted by the Southwood Committee in their "worst case scenario."

In April 1994, the government finally admitted that cows did pass BSE on to their calves.

BRITISH PUBLIC LEARNS OF CJD DEATHS

People had been dying from the human form of the disease, CJD (Creutzfeldt-Jakob disease), for years. But it was not until the 1990s that news of it began creeping into the public press. CJD claimed the lives of two dairy farmers who had tended herds with BSE infected cattle. The num-ber of human CJD cases in Great Britain was nearly ten times higher than the annual number recorded

by researchers 25 years earlier and twice as high as the number recorded five years earlier.

Vicky Rimmer, a 15-year-old Welsh girl, devel- oped the symptoms of CJD, despite no family his- tory of the disease or medical mishaps such as faulty blood transfusion. She was also extremely young, considering the very long period it normally takes for symptoms to show. This meant that the disease was most probably contracted from an external source, more than likely food.

A doctor from the CJD surveillance unit was

sent to Vicky's home; and, after examining the girl, he told her mother not to make her daugh- ter's case public. According to the *London Daily Mirror* (January 25, 1994), he told her she should think of the economy and the Common Market.

In 1993, World Health Organization (WHO) figures indicated a total of 250 suspected, and 117 proven, CJD deaths with the average age of the victims being 27 years (descending from the former CJD average of 63 years).

But the bell didn't stop tolling: 56 Brits died of CJD in 1994, followed by 42 cases in 1995.

In the summer of 1995, the Canadian Red Cross had a blood recall, when they discovered two infected Canadians had donated blood. But the press only wanted to talk about a sick bull whose owner refused to destroy him.

In February 1995, Dr. Richard Lacey, the Brit- ish scientist who first predicted this crisis in 1985—and was fired for speaking up—finally pub- lished his bombshell book. More on this later.

After initially castigating Lacey's book, the November 1995 issue of the *British Medical Journal* suggested the possibility that people might get Mad Cow from eating beef. Three million Brits immediately quit eating beef.

On March 20th, 1996, Agriculture Minister Dor- rell announced to the world that British scientists "suspected a link" between BSE and its human equiv- alent, CJD. A link between spongy brains in British cows and the even spongier brains in British politicians was at last officially on the record.

Dorrell's admission caused a furor which put

photos of stumbling, cross-eyed, drooling cows on television screens across the planet and made England's Wimpys and McDonald's burger shops stop serving beef and begin marketing a soy patty (which they did for all of three days until they had some European beef flown in and started resupply- ing the real thing).

Along with that, the news broke on the same day that ten people under the age of 42 had just died from slow, agonizing deaths of CJD. The citizenry awoke to action,—and demanded that some-thing be done.

Not only were these victims far younger than those typically afflicted by CJD (averaging 27 years old), but none had a genetic predisposition to the disease; and autopsies revealed that all had a viru- lent strain of CJD. The government was forced to admit that there was a link between the sick animals that Britons were eating and the sick- ness they were increasingly contracting.

Immediately, 10,000 British schools dropped beef from their menus; Ireland and Europe banned the entrance of British beef; and the World Health Or- ganization (WHO) announced that BSE was indeed linked to CJD.

Autopsies revealed that the brain pathology of those who died of CJD was essentially identical to the brains of the victims of Kuru in New Guinea.

All this furor shot American beef, grain, soy, and especially corn prices sky high in anticipa- tion of a U.S. corner on the feed market.

Staunch and patriotic politicians that they were, **Prime Minister Major and the German and Ital**

ian politicians ate veal chops for lunch in Turin as they haggled over the ban. (They quietly made sure it was Italian, not British, beef.) That recalled the experience of a few months earlier, when a Brit minister force-fed his gagging 4-year-old daughter a burger in front of the press corps.

The Royal Family stodgily continued serving beef at Buckingham Castle, recalling how, during World War II, they patriotically stayed in London dodg- ing bombs alongside commoners.

All this was intended to shore up the British beef industry and keep the people buying its products. And it worked for quite a while. The British people had put up with German V-2 rockets; surely they could live with little things like prions. Besides, those fast-food burgers, doctored up with synthetic (coal-tar) flavors and colors, sure tasted good.

PUBLICATION OF LACEY'S BOOK

Finally, in February 1995, Lacey's book came off the press (although it carried a 1994 copyright). If you want a copy of the book, here is the data: Mad Cow Disease: The History of BSE in Britain, by Richard W. Lacey, Cypsela Publishers, Ltd., Jer-

sey, Channel Islands, 1994.

In his book, Lacey claimed there were already over a hundred dead Britons from mad cow dis- ease. But that implied that something was wrong with the British beef supply. So, immediately, two prestigious medical journals trashed the book in scathing reviews. Not to be undone, the same week a new rock group came on the scene. Calling itself "Mad Cow Disease," it made its Lon-

don debut to rave reviews. Screaming, clapping Brits were thrilled and happily returned to their cannibal-beef dinners. McDonald's was relieved and life returned to near normal.

Year after year, people willingly eat junk, ignoring the fact that their bodies are made up of what they put in it.

Here are some sample quotations from Lacey's book:

- *p. xx*: "The British Government [beyond much reasonable doubt] has at all stages concealed facts and corrupted evidence in mad cow disease."
- p. 58: "After publication of their [Southwood] Re- port, Professor Southwood was promoted to Vice Chancellor of Oxford University, Professor Epstein was knighted and Sir John became Lord Walton."
- p. 17: "Several cases of CJD spread by blood transfusions."
- *p. 86:* "[Scrapie] infectivity was found in the liver, kidney and bones, sometimes at high levels."
- p. 86: "The greatest risk could come from bones because the procedures used to concentrate and purify gelatin could also create a potent source of the BSE prion." [This would include bonemeal in food, i.e. calcium supplements, capsules, and gelatin products.]
- *p. 88:* "With vertical transmission of BSE confirmed in 1993/1994, the infectivity of blood is implicit, at least as far as cattle are concerned." This means that a person can contract mad cow infection from eating any part of an animal. And a mother with the disease can pass it on to her fetus.
 - p. 27: "In England, as many as 30% of BSE in-

fected carcasses [are not incinerated and] end up in landfill sites."

- p.~174: "Over 11,000 BSE cattle have been born after the [contaminated feed] ban."
- *p. 6:* "Pathologists are often unwilling to undertake postmortem examinations of patients considered as having possibly died of CJD."
- p. viii: "The best guess is that 'mad person dis- ease' could emerge an epidemic in Britain" within a very few years.
- *p.* 145: "Virtually all mammals tested were vulnerable, so man is likely to be vulnerable."
- p. 30: "Where a BSE case was confirmed, the entire herd should have been destroyed and incin- erated, with restocking from BSE-free sources on new ground."
- *p. 175:* "There is also the problem of needing to house the new herds on fresh territory to prevent reinfection."

RICHARD RHODES' BOOK

Pulitzer prize winning author, Richard Rhodes, has published a helpful book, *Deadly Feasts*, on the controversy. *Here are some facts you will find in it:*

Nobel Prize winner, Dr. Carleton Gajdusek (one of the foremost researchers of Kuru and other spong- iform diseases), has declared that all the pigs in England are infected with BSE; and that means not only pork, but also pigskin wallets, foot- balls, and catgut surgical sutures. All of these come from pigs (p. 220). Noting that all the chick- ens fed on meat-and-bonemeal are infected he adds

that, in America, beef male cattle are killed at or before age two, before they are likely to show the outward symptoms of the disease.

In America, chicken excreta (manure) is fed to cattle as a good source of nitrogen (*p. 258*). As for the American FDA's ban on feeding meat and animal byproducts to cattle, Rhodes writes "That's a ban with exclusions big enough to drive a cortege of hearses through." Their own BSE advisory com- mittee urged the FDA take stronger measures (*p. 257*).

According to Rhodes' book, bovine spongiform encephalopathy has been detected in America, and not just in cattle. The American form of BSE does not cause the staggers and other behaviors found in British cattle; but instead it results in a more "sedate" collapse of the victim, referred to as "downer cattle." The nature of the brain damage is also distinct, a spongiform with differently shaped and oriented vacancies. Other forms have been trans- mitted via eating wild squirrels and wild bear. Some American zoos have lost animals to BSE.

Dr. John Pattison, Chairman of the British Government's Spongiform Encephalopathy Advisory Committee (SEAC), Dean of the University College of London Medical School, believes 500,000 people may already be incubating CJD in Britain.

Dr. Alsleben has stated that **prions can be found in** white blood cells, contaminated milk, and even in the animal grease used in lipstick.

On page 222 of Rhodes' book, Richard Lacey of the Microbiology Department of Chapel Allerton Hospital, Leeds, points out that "there was no certainty

that the source of infection had been cut off.' 'If it seems that the incubation-period average for CJD in humans begins to be about twenty five years, may- be thirty years,' he told me [Rhodes] grimly, 'then **the peak human epidemic will come around the year 2015**. If the current numbers of variant CJD cases increases by fifty percent per year, as they well might, that would take it to **about two hundred thou- sand [human] cases a year by then**.' " That com- ment is only about mortalities in Britain.

Why is the body filled with the prion infection before CJD symptoms appear? What are the subtle effects long **before the final destruction?** If these prions are indeed the rod-like structures researcher Patricia Merz describes on page 156 of Rhodes' book, then they would tend to impede cellular machinery long before they became long enough to break cell membranes and kill the cells. Thus it is possible that, long before that final break, subtle neurologi- cal effects could become evident. Dr. Merz has defi- nitely located prions in spleen tissue and elsewhere in the animals, long before any outward symptoms were manifest! This is extremely significant. Prions apparently travel freely in the blood of these animals. Therefore all tissue is likely to harbor some prions, not just brain tissue. This means that large amounts of infected cattle have been fed to other cattle which, after becoming in-fected, have been sold to the public. But, since the human form of the disease (CID) is misdiagnosed as Alzheimer's, the medical crisis continues to mount.

COWS AND THE DAIRY INDUSTRY

What about milk cows? BSE has affected all breeds including, significantly, Jersey and Guern- sey cattle on their respective islands. Jersey and Guernsey are the best breeds of milk cows that money can buy. The black and white Friesian Hol- stein (beef) cows are the most commonly affected, simply because there are far more of them in Brit- ain than any other breed. But dairy cows have also been affected. The youngest case so far recorded of a cow showing the symptoms of BSE was 20 months and the oldest 18 years.

The cattle industry in Britain is under constant pressure to produce more milk and dairy products at the lowest possible cost because the public demands it. To provide as much milk as possible, cows are often fed protein-rich concentrated food made from the carcasses of other dead animals that have been sent to stockyards (called knackers yards in Britain) or rendering plants.

Cows only produce milk when they have had a calf. After a nine month pregnancy, the calf is removed within a day or two of birth. A few months later, while still producing milk, the cow is artificially inseminated again. Cows have around three or four pregnancies before their milk yield begins to drop. Each cow is eventually slaughtered at six or seven years old, even though its natural life span would be 20 years or more. Most parts of the cow are used to make burgers, sausages, pies, stocks, and pet food. Until 1989 in Britain, this also included the brain.

More than 90 percent of BSE cases have been

in cows rather than bulls, simply because cows live longer. Beef animals are usually slaughtered around three years old and veal calves at six months. As BSE appears when the animal is around four to five years old, most beef animals are slaughtered be- fore they are old enough to show symptoms, al- though they may have the disease.

WHAT HAPPENS TO THE DISEASED COW?

In cattle, the first signs of the disease oc- curs when the cow is put under any slight pres- sure or stress. Movement to a milking station might induce fear, panic, and stumbling; and the infected animal may stand away from the rest of the herd, holding its head in an awkward posture. Despite a good appetite, the amount of milk she produces may drop and she usually loses a lot of weight.

As the muscles waste away, there may be twitchings, quiverings, and shaking. Strange be-havior can occur, such as grinding teeth; and sometimes the moo is odd.

The cow over-reacts to touch and becomes very jumpy. Eventually, she will shake violently; stagger; and, in the end, be completely unable to stand up. It is the combination of a drop in milk and the fear that the cow will fall and be unable to stand again that makes the farmer call in the vet. If the animal does not recover, it is slaughtered and the head (with its nervous tissue) is removed for examination; it is "officially" believed that this

is the only infected part of the animal.

This is unlikely, as flesh also contains nervous

tissue. It also ignores the possibility of the disease being passed from mother to calf.

The rest of the cow's body should be burned, but as many as 30% of infected carcasses end up in landfill sites—where they could be disturbed by tractors, bulldozers, dogs, or rodents. BSE is an extremely strong disease; it remains infective even after years in the soil. (Recent disclosures indicate that burning bodies could send prions into the air.)

When cattle are killed for food, only the head and some other parts (such as the spinal cord, spleen, and thymus—"specified offal") are re-moved. The rest is sold to the public. The official position of the government is that people will not be at risk when they eat cows. So the flesh (containing infected nervous tissue) is eaten, and the bones are eventually made into gelatin which finds its way into many products.

People can contract CJD from eating the flesh of baby calves. This is another proof of transmis- sion of the disease from the cow to the calf through the blood. Those who regularly eat veal (baby cow meat) are 13 times more likely to develop CJD than those who do not eat calf meat, according to the British Department of Health newsletter (BUAV Newsletter, April 1995).

MEDICAL PERSONNEL FEAR CJD

During the postmortem, extreme care must be taken because the disease is incredibly in- fectious. The pathologist wears a mask, goggles, gloves, boots, and a plastic apron; and any instru-

ments that have been used on patients suffering from CJD have to be thoroughly sterilized. For ex- ample, the silver needles used for the EEG (brain examination) must be treated with high pressure steam for prolonged periods of time or put through six successive heat cycles in a sterilizer. Even then there is no guarantee of destroying the infection. If contaminated instruments are used on another patient (which they will be if the person was not visibly ill with CJD), the disease can be (and indeed has been) transferred.

CJD is so feared by the medical profession that they have refused to perform autopsies on patients suspected of dying from it. Some hospitals have even refused to admit patients suffering with it. They find it far easier to just diagnose the victim as having Alzheimer's, without doing an autopsy.

WHY THE PROBLEM WILL GET WORSE IN AMERICA

There are some reasons why this problem is going to keep getting worse. Here are far more than a dozen of them. Others are explained else- where in this study:

The "mysterious agent" that causes spongi-form encephalopathies is not just found in the brain! It has been found in many of the organs and tissues of animals. For example, cells from the spleen, thymus, and tonsils enter the blood and find their way to many organs, including the liver and bones.

Blood can also contain the disease. Confir-

mation in 1993, that the disease can be passed from the cow to the calf—established that transmission can be by blood.

You can get BSE from any part of an ani-mal. Mammals contract BSE, scrapie, and CJD by eating the flesh of other infected mammals. Blood, corneal transplants, and hormonal injections can also transmit it. This would include pituitary, thy-roid, and insulin injections.

The bones of old cows are one of the major sources of the protein gelatin, used in many foods from peppermints to pork pies. The greatest risk could come from bones because the procedures used to concentrate and purify gelatin could cre- ate a stronger source of BSE.

U.S. and *British* sheep were infected at the same time. Both U.S. and British sheep were infected simultaneously back in the 1950s from research waste discarded by scientists trying to figure out the cause of Kuru.

As early as the 1970s, both the U.S. and British scrapie sheep were being fed to cattle. Scrapie appeared in sheep in both the U.S. and Brit- ain by the 1970s. In both countries, the dead sheep were sent to rendering plants which turned them into protein powder, which was fed to cows. That spread the disease widely.

Cattle are not checked for the disease be- fore they are slaughtered. The USDA in America only studies the brains of 100 cows per every 100,000. That is an extremely small sample.

BSE/CJD cannot be detected during incu-bation.BSE and CJD cause no antibody response.

When infection enters an animal or human, the victim's immune system shows no sign of fighting the infection as it does with bacteria, germs, and viruses. This is because the immune system can nei- ther detect nor fight it. Scientists cannot use the an- tibody-search method to see if someone is sick, as is done with AIDS.

No scientist can tell if a cow or human is in an incubating phase of BSE/CJD. The only exception is brain biopsies, and that is not done until after death occurs. There are no tests, no genetic mark- ers. Prions are not reliably found in urine. Prions can be seen in brain tissue, but you cannot open the skull of a live mammal to scoop them out.

It can take years before the full-blown disease appears. CJD disease takes between 10 and 50 years to eat away the human brain. In cows, death strikes as early as one year after exposure, as late as 8. If a cow whose milk you are drinking has it, her calf, sent to be a veal chop last winter had it when you ate him. An older cow may fall over dead with it, but meanwhile her infected calves have long since been slaughtered and served at dinner tables. The long incubation period means the farmer can- not see that the animal is ill.

BSE can be transmitted to offspring. That fact was established by researchers in Britain. Sheep and cows pass it to offspring. Chickens can put it in their eggs. Could CJD, the human form, also be trans- mitted to your descendants? This is a very serious matter. The FDA has demanded that all donors to the blood supply answer the question, "Has anyone in your family died of Cruetzfeldt-Jakob?" We dare

not wait longer before warning the public that it is no longer safe to eat these foods. They must be told that they must stop eating infected meat.

People have been dying faster from CJD than earlier. It was once thought humans could incu- bate the disease for up to five decades without go- ing into the final dementia stage, but lately British teens have been dying of it.

Farmers make too much profit selling dead cows for animal feed, for them to stop. Farm- ers have to pay \$500 in order to have an autopsy made of a dead cow. But they can sell it to a nearby rendering plant for \$100. Then it is processed into cow food.

The U.S. ban on animals in feed is being ignored. In America, there is now a ban on putting animals that died into feed. But it is well-known that it is being done anyway.

The USDA has not banned blood in animal feed. The U.S. Department of Agriculture banned diseased meat in animal feeds; but, to date, it has not banned putting blood from dead animals into the feedstocks. That link is contaminating the cattle of America today.

It is extremely difficult to kill prions. BSE and CJD prions cannot be killed the way we fought the plague, cholera epidemics, or ebola—which is generally done by burning bodies. BSE/CJD is passed on by means of prions, which are proteins that degrade at 800° F. That is far higher than the temperature which would reduce them to ash.

Infected meat should not be burned. Burn- ing is a bad idea, as prion molecules go up in the

smoke, airborne and fall back on the land. Britain is now considering burning 5 million cows soon, which will loose the prions into the air, to fall back on the land and into lakes and rivers.

Prions infect every part of the body, not just the brain. Although BSE/CJD attacks brains, it is in every part of the victim. Therefore every part of the cow is affected. None of it should be eaten. This contamination cannot be removed by cooking. Do not use blood meal in your garden. A British vegan woman caught CJD simply by dust-

ing her roses with blood meal.

Thousands of cows are mysteriously dying in America. Since 1981, the United States has had thousands of "downer" cows. These are cows which have died mysteriously. Dr. Richard Marsh, a virolo- gist on the Veterinary staff at the University of Wis- consin at Madison, says he has seen 100 cases of BSE in America between 1981 and 1989. If the prions entered U.S. beef 15 years ago and have been multiplying ever since, a million cows could be in- fected. In order not to disturb the public, the fact that so many cows are dying is being kept from them.

More on the "downer cow" syndrome in America. Massachusetts Institute of Technology and the National Institutes of Health first explored a connection between BSE, animal foods and de-mentia as far back as 1981, when American cows began to come down with a mysterious disease known as "Downer Cow Syndrome," suspiciously like BSE. Many of the downers had previously ex-hibited symptoms of the jitters, others just suddenly

dropped dead. Their brains were fed to mink who quickly manifested Mad Mink disease. In any case, downer cow corpses revealed BSE brain pathology; yet not a peep came from these scientists, not a whis- pered word to the farmers to stop rendering sheep into cattle feed, not a warning to the public to stop feeding beef to children. Since then, American farm- ers were allowed to sell sheep corpses for 28 years and cow corpses for 17.

CJD deaths are occurring now in America, but they are being mislabeled as Alzheimer's. CJD mortality figures hide behind the skirts of Alzheimer's. Some U.S. doctors know the truth yet haven't blown any whistles. The U.S. veterans hos- pital in Pittsburgh autopsied 53 sequential Alzheim- er's victims. Sampling #1 showed 5.5% had died of CJD; sampling #2 showed 6.3% had died of CJD. Alzheimer's death tolls are doubling and tripling in America, but this is not characteristic of a genetic disease. The rate of genetic diseases does not continually increase. People dying of CJD are being di- agnosed as having Alzheimer's. That is why, sup- posedly, there are no CJD (mad cow) human deaths in America. A related problem is that labs will not test patients suspected of having CJD.

Private labs are afraid to let CJD tissue in the door to be examined. They would have to burn down the lab in order to be certain they had cleansed it of the prions. Dr. Richard Deandrea, a Los Ange- les physician, who has studied CJD and BSE ex- tensively, tells of his first CJD patient. After her death, which featured symptoms atypical of Alzheimer's (fingers numb, blindness, slurred speech, weak

knees), Deandrea dogged the Center for Disease Control (CDC) in Atlanta for a pathologist who would provide him with an autopsy to see if it might be CJD. CDC evaded his phone calls for three weeks. Finally, a female CDC staffer told him that off the record—she would deny it later—"CJD is an issue no pathologist will deal with, a virtual death sen- tence to a lab. A well-trained pathologist knows the quarantine would never be lifted. You couldn't sterilize the lab to OSHA protocols. It would have to be gutted, incinerated. Forget it. Your patient died of Alzheimer's." So there may be CJD deaths, but there are not likely to be many CJD death certificates.

If you die of CJD, you will officially be listed as an Alzheimer's victim. Because no laboratory in America will knowingly do an autopsy on anyone suspected of having died of CJD. That Pittsburgh hos- pital would not have made those autopsies if the staff had known they were working with CJD! The problem is that the prions cannot be eradicated by normal methods. One researcher said that, while we keep eating our burgers, officially on all our death certificates we are going to die of Alzheimer's, not CJD. That Pittsburgh veterans hospital sampling never hit the major news wires.

Beware! BSE cow parts could be in more products than you thought possible. Gelatin cap- sules, used to enclose vitamins and minerals, come from cows. Break them in your mouth and immedi- ately spit out the gelatin. Glandular supplements from animals come from cattle or pigs. The glue on your envelopes and postage stamps comes from dead cows.

Even more than AIDS, BSE is the most prevalent, virulent disease to hit this planet since the bubonic plague of the Middle Ages. You can only avoid it by refusing to eat anything which contains meat. Forget ebola which kills you so fast you can't move ten feet and give it to anyone else, an epidemic which trained medical personnel can rather quickly eliminate.

Sick scrapie sheep are ground up and fed to dairy cows and beef cattle. That is how the USDA gets around the prohibition on feeding scrapie sheep to the cows you eat! Ted Koppel interviewed Dr. Richard Marsh on Nightline a year ago. It went like this. Koppel: "But we (in the U.S.A.) don't feed sheep brains to cows, do we?" Marsh blinked. "I don't know where your information comes from, but we do." He was instantly cut off by a commercial and did not reappear that night. The truth is, Vi- rologist Marsh, a seasoned veterinarian, knows of what he speaks. He observed this problem in Wis- consin, from 1981 to 1989. Dead sheep were fed to cattle, which, after fattening, was used to feed thou- sands of other cows who have bred thousands of animals.

Pigs and chickens are also fattened on blood and diseased, dead animals which have been ground up into pellets; so ground-up cattle are sold to farmers, to fatten up their livestock.

Prions like it in America. All the same, the beat goes on. On March 20th, 1996, the very day that Minister Dorrell lit the fuse on the Mad Cow bomb in London, a Florida man died of CJD. His wife gave a TV interview describing his shaking

knees, his lack of co-ordination, quick slipping into a vegetative state, followed by death and said that her husband had never traveled abroad in his life. Why would he have to? Prions enjoy the American climate.

How to silence the media. The beef industry frightened the TV networks into a news blackout on the subject. They are now afraid to discuss the subject. The Oprah Winfrey Show interviewed an ex-beef rancher who had seen U.S. sheep and cows dying of BSE. Immediately, beef dropped 150 points on the commodities market; and the beef industry, under the guise of "Texas cattlemen," sued Oprah for daring to openly discuss the subject. Even though she was found not guilty by a Texas jury, no televi- sion network is likely to talk about mad sheep and cows for awhile. But the beef industry actually won. They got their message across to the major networks: Tell too much about mad cow, and we will see you in court.

Small stations are more likely to reveal death toll statistics. In California, KCAL-TV News reported two recent CJD deaths, one in Stockton and the other in San Francisco. Dr. Richard Deandrea knew of a death in Lancaster, California, and another in Minnesota—all in the previous few weeks; yet the NIH claims it knows of only 11 CJD deaths since 1994.

Bypass protein animal feeds are deadly. Scientists who invented the "bypass protein" method of feeding livestock (taking the rendered corpse of a dead animal, grinding it into meat meal, and mixing it in with grains) have turned an attractive planet

into a potential graveyard. Scientists who turned healthy herbivores into cannibals may have shot themselves and humanity in the collective hoof. In order to make a lot more money in the short term, the meat industry will eventually destroy itself.

Feeding diseased animals to grazing stock also produces other diseases in the people who later eat that livestock. Even if there were no prions lurking, when you feed an herbivore protein, its body produces ptomaines, which cause lesions or tubercles in its body. That means tuberculosis. In 1989, the National Association of Federal Veteri- narians decided to create a "test-balloon" state. They allowed California to sell meat infected with tuberculosis, a practice illegal since 1906. TB immedi- ately went up 36% in the sunshine state. We do not need any more test balloons.

Hormones sicken the livestock you eat. Bo- vine immune systems have been destroyed by sev- eral other common practices. One example is the massive daily injections of synthetic growth/lacta- tion hormones which exhaust the cow who is chemi- cally stimulated to give 40% more milk. It costs the dairy farmer \$400 a year for all the drugs and chemicals he has to inject or feed his cows. They are walking chemical plants.

Antibiotics are given to keep the weakened livestock alive. All those medicinal drugs are nec- essary to ward off the multiple infections caused by the other chemicals given to the livestock. One prob- lem leads to another.

And there is more. Consider the painful muti-lation of cows with more than 4 teats. (Many have 8

teats; extras are amputated without anesthetics.) Then there's dehorning, also done without anesthetics. Keep in mind the hormones of grief created in Bessie when her offspring is calf-napped on its sec- ond day of life. This is followed for 305 days while the milk intended for the calf is stolen by the farmer. That ends with a two-day starvation period (no food or water), to dry up her milk and get her ready to "calve" again. A happy cow would live 25 years on a happy farm. A dairy cow is exhausted at 3 to 5 years of age. Her reward: She is slaughtered and herpoor, suffering corpseiseaten by humans as burger. The Prusiner Report. The definitive U.S. re- port on prions was written by a leading prion re- searcher, Dr. Stanley Prusiner. He is a professor of neurology and biochemistry at the University of Cali-

fornia School of Medicine, in San Francisco.

Although his large study goes into some depth on the habits of prions, he never once mentions the danger of eating meat. Unlike Dr. Lacey, Dr. Prusiner remained politically correct.

Hiding behind Alzheimer's. The puzzle pieces have stayed in the box; because, since the 1970s, CJD has been able to hide behind the skirts of Alzheimer's. The Alzheimer's Foundation itself seems to be clueless, saying that if current trends continue, 14 million people will have Alzheimer's by the turn of the century. No mention of CJD from them.

Extrapolating from Pittsburgh figures. It is possible to estimate the number of people who even- tually will contract CJD. If, as the Pittsburgh veter- ans hospital disclosed, 6% of Alzheimer's cases are really CJD, in the next 4 years, 840,000 U.S. hu-

mans could die of CJD. If they were of childbearing age when they caught it, there is the possibility that millions of their offspring could carry it in their genes. Many people could eventually go into spasms, then idiocy and comas, costing their families and the health system \$120,000 per patient. Likewise with all their descendants, forever. Prions are not some-thing to play around with. Yet Western governments have done it for nearly 30 years.

A different estimate, based on the percent- age of Kuru deaths. Among the Fore, the tribe of cannibals who got Kuru in New Guinea, only about one percent of the population seemed affected. This one percent figure suggests a genetic bias, and some genetic biases have been detected. This may serve as a model for predicting human death rates. Evi- dence suggests a one in a million rate of spontane- ous occurrences among susceptible species. Once inserted into a food chain that recycles animal protein, one in a hundred may get it.

In America, that one percent would translate to wellover two and a half million slow, expensive deaths, a far worse epidemic than AIDS!

Genetic diseases do not double and triple their rate. Alzheimer's is a genetic disease, which is apparently doing something today that genetic diseases do not do: It is doubling and tripling its death toll. But it is so handy to blame Alzheimer's. Doing so helps the labs, because they do not want to au-topsy anything savoring of mad cow disease. It helps the meat and fast-food industries; they can keep selling more burgers. It helps the television stations, because they do not want any more lawsuits. It helps

the government, because they want it always said of them that they do everything right.

Silence is golden, even though it can be fatal. Admittedly, if even a whisper of prions in America was voiced, huge losses could result to the \$50 billion-a-year meat industry in America.

In Britain, five days after Dorrell's admission that CJD was caused by BSE and there was the faint possibility of danger in prime ribs, the entire European Union ordered its second ban on British beef exports (the first had expired). A \$6 billion-dol- lar-a-year beef export market collapsed in a single day. Loose lips sink world economies, but silences go before apocalypses.

Getting rid of the whistle-blowers. Dr. Rich- ard Lacey was not the only warning voice. There were others: Haresh Narang, a microbiologist em- ployed by the Public Laboratory Services in New Castle, said CJD in humans came from BSE. Mi- crobiologist Dr. Steven Doeller said scrapie, CJD, and BSE were the same thing. But all the cries of the whistle-blowers were ignored.

Then, in 1995, when Dr. Lacey's book was printed, both the *British Medical Journal* and *New Scientist*, two of the most respected professional journals in England, declared the book unfit for the reading public. His book made the beef industry so nervous that, in December 1995, three more articles were planted in prestigious British journals: *The Economist, Nature*, and *New Scientist*, declaring that there was nothing to worry about; Lacey was dead wrong. Interestingly, all three articles were written by "Anonymous."

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U.S. sheep are still fed to cows. The FDA and public health officials all know that diseased sheep that die are fed to cattle. In the U.S., approxi- mately 200,000 animals are slaughtered daily.

ADDITIONAL U.S. FACTS

Thyroid, insulin, and other medicinal hor-mones. As of late 2000, questions are being raised about medicinal thyroid, insulin, and other hor-monal extracts,—nearly all of which are extracted from pork or beef. Natural thyroid extracts include Armour Thyroid, and synthetics include Cytomel and Synthroid. The natural ones are taken from the thyroid glands of animals, such as pigs.

Rendering only legal in America. In all other countries the "cash for corpses" practice is illegal. In the U.S.A., until 1997, it was entirely voluntary whether a farmer renders corpses; so, because they could not ignore free hundred dollar bills, they regularly sold their dead cattle and sheep to the feed companies. It was not until January 3, 1997, that the practice of rendering bodies and using them for animal feed was finally stopped. On that date, it was announced that offal could no longer be used to feed animals eaten by humans. A stricter ban was laid down soon after. But every part of the ban is known to be ignored by rendering plants. More on this later in this book.

U.S. chickens. In reply, the USDA said that they have never found a chicken sick with BSE. But the reason for that is the fact that U.S. chickens are killed before they are old enough to openly manifest the symptoms. No U.S. fryer lives long enough to

manifest dementia, but it has lived long enough to give the disease to the person who eats it.

U.S. hunters dying of CJD. Between 1998 and the end of 2000, three young hunters in Western U.S. died from CJD. Other deaths are suspected.

Blood donors banned. On January 17, 2001, the FDA ordered a ban on blood donations in the U.S. from anyone who has lived in Britain or Ireland longer than six months, between the years 1980 and December 1996. But it is still legal for renderers to put sick/BSE animals (with the blood in them) into the feed pellets.

IS MAD COW IN AMERICAN CATTLE?

We do know that cattle were imported into the United States for breeding purposes until 1987. We also know that the earliest known cases of BSE in British cattle were confirmed in 1986. (Prior to that time, the disease was in the cattle, but not confirmed by research tests until 1986.) It was not until 1987 that the British Government permitted the news about that finding to be published. Over 400 cases of mad cows were confirmed in Britain that year, but the actual number of cases may have been far higher, as the disease was not yet "notifiable"; that is, there was no legal requirement that veterinar- ians be notified when a cow fell dead with the dis- ease. In order to reduce the number of BSE animals reported, the government said it would pay half the price the farmer could get from the dead cow if, in- stead of reporting it, he sold the BSE-laden animal to the rendering company, to be made into animal feed. Yet, the next year, 1988, over 2,000 confirmed

cases were reported.

So is BSE in American cattle? We know that there is an ever-increasing number of downer cows in the U.S., but the government refuses to conduct autopsies of their brains to see if they have spong- iform. Instead, they are sent to the rendering plant, so the meat animals you eat will fatten up quicker. Is this mass production insanity? The proper name for it is raw greed. America changed when abortion was legalized in early 1973. Human life is no longer valued as it once was.

Although cases of CJD have not substantially increased in America, it should be kept in mind that $\,$

(1) autopsies are rarely performed on people who died of CJD symptoms, and (2) those symptoms are similar to Alzheimer's (a totally different disease), and (3) deaths attributed to Alzheimer's are rapidly increasing.

Here is an indication of what is actually hap-pening:

- Physicians at the Veterans Administration Medical Center in Pittsburgh autopsied 54 patients who died of dementia. It was found that three had actually died of CJD! (*Journal of Neurology*, 1989, 39 (1): pp. 76-79). That is a shockingly high ratio.
- Consider the number of CJD cases that have occurred in the northeastern corner of Texas in two recent years (1996-1998). Eight cases were diag-nosed in this 23-county area with a population of about a million. The victims were between 46 and 65, averaging 57.

Based on the assumption that there is only one CJD victim per million people, there should only

have been one death. But eight cases would make it one in 125,000. That would be equivalent to 1,500 CJD deaths throughout America, per year. Will the ratio get worse with the passing of time? Is this why so many more people are "dying of Alzheimer's"? (Texas Department of Health, August 25, 1997).

It is now known that prion-induced disease, in animals (BSE) and humans (CJD), has for centuries existed as an extremely rare disease in various parts of the world.

WHO says CJD may have spread worldwide. On

ELSEWHERE IN THE WORLD

December 22, 2000, on behalf of the World Health Organization, Dr. Maura Ricketts issued a statement warning that "exposure worldwide" to BSE and CJD may have already occurred. The state- ment went on to say the WHO is going to convene a major meeting of experts and officials from all re- gions to discuss this problem. It will be held in Geneva in late spring 2001. This announcement followed a review of scientific evidence of several ex- perts. "Concerns center on British meat and bone- meal exports in the 10-year period between 1986, when BSE surfaced in Britain, and 1996, when an export ban was imposed on British beef" (Reuters). Over 90 deaths from CJD in Europe. Since October 1996, alone, over 90 people are acknowl- edged to have died of CJD, with more dying each

year than the year before.

Stealing from the zoo. The January 28, 2001, press reports that people are sneaking into the Berlin Zoo, at night, and stealing geese and other ani-

mals and eating them! They are afraid to buy meat at the grocery store. (But, very likely, zoo animals are fed the same rendered rations.)

If the situation wasn't so miserable, it would be funny. Read this:

"Nothing seems sacred any more as Germans, confronted by empty shelves at the supermarkets, go foraging for food. With BSE beef already off the menu, followed by sausages and now pork, filling a German belly is becoming nearly impossible. As hunger grips, no one, not even the dedicated Kreuz- berg zookeepers, will object to a bit of theft" (AP, from Berlin, January 28, 2001).

"Everyone must get used to elk, reindeer, ostrich, crocodile and other exotic meats which have recently turned up at the shops, or go hunting" (*ibid.*).

Two different strains. It appears that there are different strains of BSE in America and Britain. But both are killers. The difference between the symptoms of American downer cows and British mad cows could possibly be explained by different strains of scrapie in the two countries. Cattle injected with U.S. strains of scrapie develop a neurological disorder, but their brains do not show the spongiform pattern which is characteristic of BSE cows in Brit- ain (Reuters, September 23, 1997, citing Dr. Paul Brown of the National Institute of Neurological Disorders and Strokes).

Based on that technicality, the FDA is able to declare that there is no "mad cow disease" in America. But the cows are still dying of a similar brain disease, and those cows are still being fed to other cows which you are eating.

Shipping mad cow everywhere. Here are some facts gleaned from the March 12, 2001, issue of *News-week*:

- British exporters shipped the remains of BSE-infected cattle to 80 nations all over the world as cattle feed. Millions of people in Europe, Russia, and Southeast Asia have eaten cattle raised on it.
- Massive amounts were shipped. For example, between 1980 and 1996, Indonesia imported 600,000 metric tons of cattle feed that was prob- ably infected. Thailand, 185,000; Taiwan, 45,000; Philippines, 20,000.
- Between 1987 and 2000, in addition to other countries, Britain had 180,401 reported BSE live-stock; Ireland, 587; Portugal, 509; Switzerland, 366; and France 241.
- Prion diseases in animals include the follow- ing (dates indicate when first noticed): scrapie in sheep, 1970s; elk, 1980; deer, 1980; minks, 1981; cattle, 1985; cats, 1990; zoo monkeys, 1992.

TOWARD PERSONAL SOLUTIONS

What about cooking the meat or milk? The pasteurizing of milk, at 150 degrees, makes the prion think it's a sunny day. The cooking of meat at 212 degrees makes him think he's in a pleasant sauna. Raising the heat to frying in the 320 range might make him even blink; but you must reduce the prion to total ash at 340 degrees Centigrade (in our American Fahrenheit system that would be 800 degrees), to immobilize him and take away his abil- ity to replicate.

What about the BSE/CJD spore? There is no

solvent known to immobilize the Mad Cow spore. This kind of microbial tenacity is so far-fetched that it frightens the medical community. If you ask a doc- tor to do an autopsy of a patient who died of CJD, he flees, knowing that if he exposes his lab to this disease, the lab will be closed down by government officials. He cannot clean his sink without burning it up! By the way, when asked about this by worried reporters, Paul Brown of the NIH reassured them. He said he could clean prions off his hands with Ivory soap. We welcome him to try it in public.

Any other solution? The medical community has no cure for CJD. It is—very simply—fatal. There is no drug or surgery which can cure it. But Dr. Ri- chard Deandrea says that if you think you've been exposed, enzyme therapy might work, seeing that pro- teins can be dissolved by enzymes which are found in raw foods. But Dr. Prusiner has written that this protein molecule laughs off all the enzymes he tried on it.

More than just meat is infected. If Mad Cow is in meat, it could be in dairy products and eggs. It is in mayonnaise. It's in the gelatin, in candy, or wrapped around a vitamin pill. It's in blood meal fertilizer, urea fertilizer, and the manure clinging to store-bought mushrooms. Animal derivatives are used in vaccines; pharmaceuticals, like Premarin; and in glandular substances used in remedies, such as melatonin. It is in pet food, gloves, film, plastics. British leather was banned by Egypt a week after Minister Dorrell's admission.

The only answer is to go vegetarian. Choose vegetarian proteins like tofu, nuts, or beans. You will

be healthier in every way, as these proteins do not tax the immune system as much as flesh. Immune systems love a whole, live, raw food diet; so eat raw, dark-green salads with nuts, sprouts, and seeds. Cleanse with enemas or colonics. Take periodic raw juice fasts. Besides a vegan diet of vegetables grown on organic soil, take "good fat" supplements like flaxseed. The oils and proteins found in nuts and seeds are good. Be wary of dairy products; they could be infected with prions. Make almond milk, brown rice milk, tofu milk. Take multi-vitamin supplements. Blue green algae, spirulina, chlorella are complete foods with B-12. Animal source of B-12 is danger- ous now. Get rid of other eco-hazards that stress the immune system, such as fluoride toothpaste, per-fume, and solvents like propyl alcohol (used in all soap, detergent, shampoo, and cleaning solutions for all factory food and juice machines). Go 100% natural. Become a vegetarian.

In this study, you have learned a lot of facts. If you want to take the situation seriously, you ought to decide right now to make some changes in your diet!

But you should be warned that some of the media right now has a different message for you: "Everything is peace and safety; there is no dan- ger. Eat as you please; the meat is as disease-free as ever. Government and industry reports confirm that there is no CJD in America, no diseased animals are now being fed to cows, and the U.S. is totally sealed off from the problems in Europe.

American

meat is as disease-free as it has ever been." Decide for yourself whose advice you will follow.

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Do you want to believe the eminent scientists quoted or referred to in this report, such as Drs. Pattison, Lacey, Prusiner, Dorrell, Alsleben, Marsh, Narang, Deandrea, Merz, Doeller, Gajdusek, and Ricketts?

Or do you want to believe the Southwood Committee, the Tyrell Report, the *British Medical Jour- nal*, *The Economist, Nature, New Scientist,* the Brit- ish Department of Agriculture, the USDA, the NIH, the U.S. beef industry, and what you hear on televi- sion?

This is your life. Do what you want with it.

You have just completed Book One. You will find further information by checking the Bibliography at the back of this book. Three excellent sources are: Richard

W. Lacey, Mad Cow Disease: The History of BSE in Britain, Cypsela Publications, 1994; Sheldon Rampton and John Stauber, Mad Cow U.S.A.: Could the Night-mare Happen Here? Common Courage Press, 1997; Ri-chard Rhodes, Deadly Feasts, Simon & Schuster, 1997.

ABOUTTHENEXTCHAPTER

THECURSEOFTHESLAIN—

Thecursehasbeenplacedbythedumbbeastsonthepeo plewillingtofattenthemontheirdiseasedrelatives, splashtheirdeadbodieswithmanure,andthencook and eat the remains!

Never before in human history have so manypeople eaten such an unhealthful diet.

- BOOK TWO -

FROM COW TO DINNER PLATE

First, we will consider what is in the live-stock and then what is in the feed they eat. We will then follow the animals to the slaughter-house and processing plant, and from there to the restaurant.

Book One was written essentially as a conden-sation of data in several books and articles. But, here in Book Two, extensive source references will be provided. Otherwise you would find it hard to believe what you are about to read. Yet it is only through information that we can make intelligent decisions. A more complete Bibliography will be found at the back of the book.

WHAT IS IN THE LIVESTOCK?

In the cattle, hogs, sheep, chickens, and turkeys we find various amounts of diseases, antibiotics, insecticides, and growth hormones. Some creatures have more than others.

1 - MAD COW DISEASE

Cattle were regularly imported into the United States for breeding purposes until 1987. The ear-liest known cases of BSE in British cattle were con-firmed in 1986, but it had to have been in the

British Isles for years before it was confirmed. In 1987, the British Government made public the news about mad cow in cattle. That year alone, over 400 cases of mad cows were confirmed. The actual number of cases may have been far higher; for no requirement was yet in place requiring the re-porting of sick cattle and British farmers could make more selling the dead carcasses to the rendering plants, to be ground up into cattle feed. Yet, the next year, 1988, over 2,000 confirmed cases were re-ported (Richard Rhodes, Deadly Feasts).

Is mad cow disease in American cattle? We know that there is an ever-increasing number of downer cows in the U.S., that downer cows do not show the same brain patterns as British mad cows, and that the U.S. Government refuses to conduct autopsies of cow brains to see if they have spongiform. Instead, they are sent to the rendering plant (Sheldon Rampton and John Stauber, Mad Cow U.S.A.).

2 - OTHER DISEASES

At the back of this book, in the chapter on *Dis-eases of Animals*, you will find abundant evidence that livestock carries a variety of other diseases.

3 - ANTIBIOTICS AND INSECTICIDES

In order to combat mastitis and avoid financial loss, ranchers and feedlot operators inject their cows with massive doses of antibiotics. There are about 80 such antibiotics approved for use in agriculture. Only seven are authorized for use in lactating cows, but the USDA tests for only four of

them (Journal of American Veterinary Association, July 1, 1992).

When milk from grocery store shelves in the northeastern states was analyzed, it was found that 63% had detectable residues of antibiotics (ibid.).

A 1988 Illinois survey found that 58% of the drugs used on dairy farms were not approved for such use (Government Accounting Office Report, Au- gust 1992).

The use of BST (bovine growth hormone) and other drugs weaken the animals, so that more anti- biotics are needed to keep them in production until they are slaughtered. There are extraordinary risks in eating meat and drinking milk, laced with antibi- otics. Among other problems, it can lead to the development of disease-causing organisms which are resistant to antibiotics, and thus are more deadly. Should you be eating drugresistant or- ganisms? (Marc Lappé, When Antibiotics Fail, 1986, p. xii).

Howard Lyman, who used to be a Montana cat-tleman, says cattlemen regularly dump the anti-biotics in all the feed rather than giving it only to sick cattle. By overfeeding the livestock on anti-biotics, the microorganisms become resistant to the drugs. Such bacteria, of course, are extremely dan-gerous (H.F. Lyman, Mad Cowboy, pp. 55-56). When you eat that meat you are getting a lot of drugs with it. If it is undercooked, you are getting drug-resis- tant bacteria in the meal.

Sometimes the drugs used to inoculate cattle were eventually determined by the government

to be dangerous to human health. (Even the government knows that you will consume the drugs in those cows and pigs.) But the USDA always made sure the dangerous drugs were used up by the ranch- ers before the ban went into effect (*ibid.*).

With every cow in a feedlot pen producing 25 pounds of manure each day, the flies get so thick that they actually threaten the cow's ability to breathe. In an effort to do something about the flies around them, the cows kick up so much dust they contract what is called dust pneumonia. To counteract this, the feedlot operator fills up a fly fogger every morning and sprays great quantities of insecticide over the cattle and everything else. The spray, of course, falls into the feed which the cattle eat (Lyman, p. 56).

Certain insects lay eggs on the hide of the cattle, which hatch into grubs which burrow in. The rancher pours other types of insecticide onto the cattle, which soaks in and kills the worm-like larvae. More insecticide inside the livestock, to be served to you later (*Lyman*, *p.* 57).

4 - BOVINE GROWTH HORMONE

Monsanto, one of the largest agriculture/live-stock chemical companies in the world, devel-oped recombinant bovine growth hormone (rBGH). In 1993 it was approved by the FDA in the form of the drug, Posilac. rBGH is a genetically engi-neered copy, although a less than perfect one, of a naturally occurring cow hormone.

Because most people do not want to consume hormones in their meat and milk, **Monsanto calls**

it "recombinant bovine somatotropin" (BST). But "somatotropin" just means "growth hormone." Monsanto has pushed hard to get dairy pro- ducers to buy BST in order to increase milk production in their cows.

When some farmers dis-covered that BST was draining calcium from the cow's bones in order to produce more milk, ultimately sick-ening and killing the cows, Monsanto tried to keep the news from other dairy farmers, without success (Conklin Corporate Newsletter, Vol. 62; Wisconsin Farm-ers Union News Release, October 7, 1995; Fair maga-

zine, May/June 1995).

In order to solve the problem of the cows weak- ened by BST, Monsanto urged that more high-pro- tein pellets, fortified with dead animals, be given to the cows. **BST also increased the percentage of mastitis in the cows. This put more pus into the milk that you drink** (St. Louis Post-Dispatch, Janu- ary 28, 1990; Nature, October 20, 1994).

In order to combat mastitis and avoid financial loss, farmers inject their cows with massive doses of antibiotics. There are about 80 such antibiotics approved for use in agriculture. Only seven are authorized for use in lactating cows, but the USDA tests for only four of them (Journal of American Veterinary Association, July 1, 1992).

Not only does it produce abnormally high milk- fat concentrations, but **BST also increases the amount of Insulin Growth Factor 1** (IGF-1). IGF-1 is a chemical in the body that controls the cellular response to growth hormone, and is the same form in both cattle and humans. An excess in humans can bring about acromegaly, a disease which includes

enlargement of the hands, feet, nose, and chin (*ibid.*). An excess growth harmone in cows, from BST, is known to result in higher levels of IGF-1 in the cows ("Human Food Safety Evaluation," Science 249:875-84, August 24, 1990; Prosser, et al., Journal of Dairy Research, 56:17-26, 1989).

When rats were fed food with IGF-1, it significantly increased their body weight. Those of you who are trying to keep your weight down, keep this in mind (Justevich and Guyer, "BGH: Human Food Safety Evaluation," Science, 249:875-84, August 24, 1990).

Upon learning the facts, the European Union and Canada placed a moratorium on the use of rBGH (bovine growth hormone). But the FDA refuses to also do so. **So it is still in U.S. milk and meat.**

In 1994, Vermont became the first state in the nation to require that labeling for dairy products notify the consumer when they contain bovine growth hormone. The International Dairy Foods Association promptly sued the State of Vermont, be- cause **it did not want the public to know that growth hormones were in the milk.** Its argument in court, in effect, was this: Because there is no dif- ference between milk without the growth hormone, therefore the public should not know when it is in the milk (International Dairy Foods Association News Release, April 25, 1994).

Monsanto lost the suit but won it on appeal. Ver- mont was refused the right to require truthful labeling of dairy products. No state in the na- tion has a standing law mandating labeling of milk products, to let consumers know about milk

additives or whether or not those products are genetically engineered.

Threatening letters were sent by the firm to milk retailers, warning them that they could be violating federal law by advertising that they were selling milk that came from cows free of rBGH. Monsanto, with assets of \$7.7 billion, then sued a small family-owned dairy in Waco, Texas, and some family-owned dair- ies in Iowa that labeled their milk as being free of the artificial hormone ("rBGH and Biotech Foods Fight Continues," Pure Food Campaign Newsletter, Novem- ber 7, 1994).

WHAT IS IN THE FEED?

Next, we consider the rations that are fed to the livestock. One would expect it to contain grasses, alfalfas, grains, and soybeans. But, surprise, the food of the animals which will soon be on your dinner plate is far more exotic.

1 - DISEASED ANIMALS IN THE FEED

- Fact 1: It is at the rendering plants that ani- mals are ground up into "high-protein pellets" for animal feed.
- Fact 2: Only animals which have died—gen- erally sick and diseased animals—are sent to the rendering plants. Healthy animals are never killed and shipped to rendering plants.
- Fact 3: Did you know that a single pellet, containing mad cow prions, could infect an ani- mal? "A feed kernel the size of a peppercorn can transmit BSE to an animal in the feed" (March 12,

2001, issue of Newsweek).

Prior to 1997, dead cattle could be fed to cat-tle but that year the FDA made a new ruling. Here are several facts about it:

First, dead cattle can no longer be officially fed to cattle in the United Sates.

Second, in that ruling, the FDA yielded to demands of the meat industry, that they be allowed to keep feeding cheap dead animals to the animals being fattened to feed American citizens. Therefore the current FDA regulations allow dead pigs and dead horses to be rendered into cattle feed, along with dead poultry.

Third, the regulations not only allow dead poultry to be fed to cattle, they allow dead cattle to be fed to poultry (chickens and turkeys). If you would like to read all the strange things that can be fed to the animals you will eat tonight, we refer you to the following sources: "Substances Pro- hibited from Use in Animal Food or Feed: Animal Pro- teins Prohibited in Ruminant Food; Final Rule," Part II, Federal Register, June 5, 1997; Ellen Ruppel Shell, "Could Mad Cow Diseases Happen Here?" Atlangid Mounthly, September 1998; Rebecca Osvath, "Some Feed and Manufacturing Facilities Not Complying with Rules to prevent BSE, Survey Finds," Food Chemical News, April 3, 2000.

Fourth, although the ban forbids putting of dead cattle (including downer cows which died of a form of mad cow disease) into the cattle feed pellets,—yet it is still being done. **Dead cattle are still being put in the feed given to cattle.** More on this be-low.

Fifth, the FDA has imposed a ban on the use of

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bone, bone marrow, and spinal cord in ground beef, but the FDA continues to permit animal blood and scrapie sheep to be ground into animal feed. Yet, as we earlier noted, research shows that BSE is not just in the brains, but also in the blood and muscles of the animals.

Sixth, In 1989, the American rendering industry announced it would no longer accept sheep heads. This was done to avoid government-imposed restrictions. Sheep heads are obviously a small part of what they grind up. Remember that only at rendering plants are animals ground up into "high-protein pellets" for animal feed (and only sick animals which died are sent to the rendering plants). But an FDA survey, made three years later, found that 15 of 19 plants inspected had not implemented even that ban! No government action was taken against the plants (FDA Survey, "Report of Findings of Directed Inspections of Sheep Rendering Facilities," January 1993).

Seventh, although the FDA bans bone, bone marrow, and spinal cord in ground beef, an Agriculture Department survey in 1997 revealed that those materials were still being included in the ground meat it tested ("USDA Reports Spinal Cord Bits in Ground Beef," Associated Press release, February 21, 1997).

Eighth, the FDA permits blood to be placed in the feed given to all livestock.

To add to the puzzle, Americans who spent more than six months in the United Kingdom during the 1980s are now forbidden to donate blood, in order to prevent the spread of BSE's human variant, Creutz-

feldt-Jakob disease (CJD). That restriction is due to the fact that the USDA is well-aware that mad cow disease can be passed through the blood.

Yet, down to the present time, the rendering plants are still legally permitted to put blood from dead animals in cattle, hog, chicken, etc. feeds! This is incredible, but true.

Someone will reply, "Well, the blood and other animal parts are cooked before being pressed into pellets for feeding to the animals we'll eat."

The fact, known by both the British and U.S. Governments, is that *prions* (the infective agents in mad cow disease) are not destroyed by any heat below 800° F., which is far above that which cooking produces. (Every housewife knows that anything cooked in water never goes above 212° F.) So the cattle blood, which is still put in animal feed in America, can be passing live prions to the hogs, cattle, and chickens you munch on for sup-

per.

Ninth, the FDA ruling does not forbid the inclusion of anything else in animal feed,—so anything else which contains protein is regularly added to the feed pellets.

To add to the profit, the rendering plants sup-plying the large feedlots have worked out a scheme to purchase dead dogs and cats from city animal shelters. Arrangements have also been made with city waste disposal departments, to ship them the road kill which is picked off the streets and roads. A nice arrangement, since all parties (except the eating public) make money on the deal. Millions of dead cats and dogs are fed to the cattle every year

("USDA Reports Spinal Cord Bits in Ground Beef," As-sociated Press release, February 21, 1997).

Tenth, the intestines and their contents are never (never) removed from the dead animals which are fed to the cows, hogs, chickens, and other livestock.

Eleventh, so there are a few safety regulations; but none of them are enforced and most are ignored. Ironically, the U.S. feed ban (which is not obeyed anyway) is less stringent than the one in Europe, which forbids all mammalian meat and bonemeal in any ruminant feed.

Officially, the practice of feeding dead animals to cattle was banned in 1997; but, unofficially, it continues to the present day. FDA reports, released in 2000 and again in early 2001, attested to the fact that the ban has been widely ignored by the feedlots.

The FDA only banned the practice after the situ- ation in Britain had gotten so bad that cattle by the thousands were being slaughtered there, in an ef- fort to stamp out mad cow disease. The evidence was abundant that it had been caused by feeding dead animals to cattle since the 1970s; we have been doing the same thing in America for over 20 years.

But there is more.

2 - ANIMAL WASTE IN THE FEED

Animal manure is collected and put in the animal feed "in order to increase its protein content."

What the cattle are fed also helps spread the dis-

ease. Feedlot owners do not want to feed the animals too much grain, since it is expensive. In 1997, it was reported that 75% of the cattle in the U.S. are routinely fed livestock wastes ("The Next Bad Beef Scandal? Cattle Feed Now Contains Things like Chicken Manure and Dead Cats," U.S. News & World Report, September 1, 1997).

Anything and everything is fed to the animals you will eventually eat. The waste products from poultry plants, including the sawdust and old newspapers used as litter on the floors, to catch the manure, are also being fed to cattle (E.R. Haapapuro, N.D. Barnard, and M. Simon, "Review: Animal Waste Used as Livestock Feed: Dangers to Hu-man Health," Preventive Medicine, September/October 1997).

In Arkansas alone, about 3 million pounds of chicken manure were fed to cattle in 1994 (*ibid.*). Dr. Neal

D. Bernard, head of the Physicians Com- mittee for Responsible Medicine, mentions in the above article that chicken manure may contain dangerous bacteria, such as Salmonella and Campylobacter; parasites, such as tapeworms and Giardia lamblia; antibiotic residues; arsenic;

and heavy metals (ibid.).

An Arkansas farmer was quoted in *U.S. News & World Report* as having recently purchased **745 tons of litter collected from the floors of local chicken-raising operations. After mixing it with small amounts of soybean bran, it is the ration he regularly feeds to his eight hundred head of cattle, making them, in his words, "fat as butter- balls." That farmer is one of many who has found**

manure to be even cheaper food than commercial animal feed, which is a combination of manure and dead animals, plus some soy flour ("The Next Bad Beef Scandal," U.S. News & World Report, September 1, 1997).

A friend from Colorado phoned this week and told of his interview with a former worker at a large hog farm. The worker told him that the pigs were crowded so close together in their growing pens that they could hardly move. Under such conditions, many regularly died. The dead ones, covered with manure, when found were run through a grinder machine and fed to those still living. When the survivors were fat enough, they were shipped off to the slaughterhouse.

3 - INSIDE A RENDERING PLANT

Feed pellets, on which all kinds of livestock are grown and fattened, are produced in ren-dering plants. Here is a view of what happens inside them.

After touring a rendering plant in southern California and doing additional research on the subject, Keith Wood wrote an article on the activities inside a rendering plant. A watered-down version of his report was published in the San Francisco Chronicle and on ABC's 20-20. He later wrote a more complete description which was published in Earth Island Journal. Here is the heart of that report:

"A rendering plant somewhere in southern California—The rendering plant floor is piled high with

what is called 'raw product.' Thousands of dead dogs and cats; heads and hooves from cattle, sheep, pigs and horses; whole skunks; rats and raccoons—all waiting to be processed. In the 90° heat, the piles of dead animals seem to have a life of their own as millions of maggots swarm over the carcasses.

"Two bandana-masked men begin operating Bobcat mini-dozers, loading the 'raw product' into a ten-foot deep stainless steel pit. They are undocu-mented workers from Mexico, doing a dirty job. A giant auger grinder at the bottom of the pit begins to turn.

"Popping bones and squeezing flesh are sounds from a nightmare you will never forget.

"Rendering is the process of cooking raw ani-mal material to remove the moisture and fat. The rendering plant works like a giant kitchen. The job of the man, called 'the cooker,' is to 'blend' the raw product in order to maintain a certain ratio be- tween the carcasses of pets, livestock, poultry waste and supermarket rejects.

"Once the mass is cut into small pieces, it is transported to another auger for fine shredding. It is then cooked at 280° F. for one hour. The continu- ous batch cooking process goes on non-stop, 24 hours a day, seven days a week as meat is melted away from bones in the hot 'soup.' During this cook- ing process, the so-called 'soup' produces a fat of yellow grease or tallow that rises to the top and is skimmed off.

"The cooked meat and bone are sent to a hammer-mill press, which squeezes out the re-

maining moisture and pulverizes the product into a gritty powder. Shaker screens sift out ex- cess hair and large bone chips. Once the batch is finished, all that is left is meat, bonemeal, and yel- low grease.

"As the American Journal of Veterinary Re-search explains, this recycled meat and bone-meal is used as 'a source of protein and other nutrients in the diets of poultry and swine and in pet foods, with lesser amounts used in the feed of cattle and sheep. Animal fat is also used in animal feeds as an energy source.'

"Every day, hundreds of rendering plants across the United States truck millions of tons of this 'food enhancer' to poultry ranches, cattle feedlots, dairy and hog farms, fish feed plants and pet food manufacturers where it is mixed with other ingredients to feed billions of ani- mals; many of which, meat-eating humans will eat.

"Rendering plants press large amounts of the protein powder into pellets which become the staple diet of cattle, hogs, and other livestock. Other by- products are bonemeal, blood meal, fat, tallow, and yellow grease.

"This elaborate system of food production through waste management has evolved into a recycling nightmare. For, you see, **rendering plants are also processing toxic waste**.

"Along with the dead animals (the 'raw prod-uct'), a whole menu of other ingredients are tossed into the 'soup.'

"Pesticides enter the rendering process via poi-

soned livestock, fish oil laced with bootleg DDT and other organophosphates that have accumulated in the bodies of West Coast mackerel and tuna.

"Because animals are frequently shoved into the pit with flea collars still attached, organophosphate-containing insecticides get into the mix as well. The insecticide Dursban arrives in the form of cattle insecticide patches. Pharmaceuticals leak from antibiotics in livestock; and euthanasic drugs, given to pets to put them to sleep, are also in-cluded. Heavy metals accumulate from a variety of sources—pet ID tags, surgical pins, and needles. "Even plastic goes into the pit. Unsold super- market meats, chicken and fish arrive in styrofoam trays and shrink-wrap. No one has time for the te-dious chore of unwrapping thousands of rejected rotten meat packs. More plastic is added to the pits with the arrival of cattle ID tags, plastic insecti-cide patches and the green plastic bags contain-

ing dead pets from veterinarians.

"Skyrocketing labor costs are one of the economic factors forcing the corporate flesh peddlers to cheat. It is far too costly for plant personnel to cut off flea collars or unwrap spoiled T-Bone steaks. Every week millions of packages of plastic-wrapped meat go through the rendering process and become one of the unwanted ingredients in animal feed.

"The following sign was posted outside the main entrance to the rendering plant, as evidence that the animals entering it had been treated in a manner humane societies acceptable to and animalorganizations. But a second sign, indicating that the 'food' that came out of it was also safe for

the poor creatures who would later eat it, was miss-ing:

" 'NOTICE—All Animals Are to Be Destroyed In a Humane Manner and No Processing Is to Begin Until the Animal Has Expired. —The Management'"

CONDITIONS IN THE SLAUGHTERHOUSE AND PROCESSING PLANT

Slaughterhouses and processing plants used to be small operations, scattered around the country. But today, a few gigantic, combinational slaughterhouse/packing plants (actu-ally thirteen just now) are processing and ship-ping out most of the meat in America. The killing and packing of animals is done so fast, that serious contamination frequently results.

1 - THE PROBLEM OF CONTAMINATION

The pathogens from infected cattle are spread in feedlots, at slaughterhouses, and in grocery store hamburger grinders.

The two slaughterhouse tasks most likely to contaminate meat are the removal of an animal's hide and the removal of its digestive system.

The hides are pulled off by machine. If it has not first been adequately cleaned, chunks of dirt and manure may fall from it onto the meat. Stomachs and intestines are still pulled out of cattle by hand. But if the job is not done slowly and care-fully, the contents of the digestive system may spill all over.

The problem is the increased speed of today's production lines. A single worker at a "gut table" may eviscerate 60 cattle an hour. Performing the job properly takes a fair amount of skill. One former "gutter" said it took him six months to learn how to pull out the stomach and tie off the intestines with- out spilling everything onto the meat. At a slaugh- terhouse Shlosser visited in Lexington, Nebraska, the hourly spillage rate at the gut table was as high as 20%. The contents were splattering out of one in every five carcasses.

Then there are the knives. They are sup-posed to be disinfected every five minutes, but this may not be done. Workers know it is important to maintain production quotas, if they want to keep their jobs. If the knives touch manure and are then used to cut into the carcass, bacteria and viruses are transmitted—not to speak of the ever- present *E. coli* bacteria, billions of which are in ev- ery digestive tract.

Slaughterhouse workers are often illiterate and always overworked. They sometimes forget that the meat will be eaten. **Meat is dropped on the dirty floor and then put back on the conveyer belt.** They cook bite-sized pieces of meat in their steriliz- ers (which are supposed to sterilize their knives), which contaminates the sterilizers (*Eric Shlosser, Fast Food Nation, p. 203*).

The most dangerous of all *E. coli* bacteria in the intestines is *E. coli* 0157:H7. Yet a recent USDA study found that, during the winter months, about 1% of the cattle at feedlots carry *E. coli* 0157:H7 in their gut. The proportion rises to as

much as 50% during the summer! Even assuming only a 1% infection rate, that means three or four cattle with that microbe are eviscerated at a large slaughterhouse every hour. (The study was conducted by the USDA's Agricultural Research Service and cited in "Study Urges Pre-Processed Beef Test for

E. coli," Health Letter on the CDC, March 13, 2000.)

Of course, the likelihood that those infected animals will be eaten by many people is greatly increased when the meat is processed into ground beef!

Years ago, burger meat was made in local butcher shops from leftover meat scraps. Cattle were slaugh- tered locally. But today, large slaughterhouses and grinders dominate the nationwide production of ground beef.

A modern processing plant (the new name for "slaughterhouse") can produce 800,000 pounds of ham- or beefburger a day. It is then shipped through- out the nation, which can be a somewhat lengthy process. A single animal infected with *E. coli 0157:H7* can contaminate 32,000 pounds of that ground beef (cited in Armstrong, et al., "Food-borne Pathogens").

Other statistics add to the frightening picture: The animals used to make about one-quarter of the nation's ground beef are worn-out dairy cattle. And those are the animals most likely to be diseased and riddled with antibiotic residues ("Relative Ground Beef Contribution to the United States Beef Supply: Final Report," American Meat In- stitute Foundation, in cooperation with the National Cattlemen's Beef Association, May 1996).

The stresses of industrial milk production make

dairy cows even more unhealthy than cattle in a large feedlot. Some of the fast-food burger restaurants rely heavily on old cows for their burgers, since they cost less, yield lower fat meat, and enable the chain to boast that all its beef is raised in the United States. A single fast-food burger now contains meat from dozens or even hundreds of different cattle (cited in Armstrong, et al., "Food-borne Pathogens").

Upton Sinclair's book (*The Jungle*, mentioned at the beginning of this present book), exposing the meatpacking industry nearly a hundred years ago, led to efforts by the U.S. Government to impose regulations which were bitterly opposed by the meat industry. That warfare has continued down to the present time.

It was during the 1980s, that the new meth- ods of keeping the animals in filthy feedlots and butchering them in gigantic mass-production slaughterhouses were fully developed. As the risks of widespread increased, because of those contamination techniques, the meatpacking industry blocked the use of microbial testing in the federal meat inspection program. A panel appointed by the National Academy of Sciences (NAS) warned, in 1985, that the nation's meat inspection pro- gram was hopelessly outdated; for it still relied on only what the federal inspectors could see and smell, as the carcasses rushed by them. The report stated that dangerous pathogens could not be detected in this manner (NAS, Meat and Poultry Inspection: The Scientific Basis of the Nation's Program, 1985). A sec- ond report, issued three years later by another NAS panel, warned that the public health structure of the United States was not prepared to detect or cope with newly emerging pathogens which might occur (NAS, The Future of Public Health, 1988).

In spite of those reports, the government cut spending for slaughterhouse inspections and for all

U.S. Department of Agriculture (USDA) oversight. Two months after the second NAS panel pre-

sented its report, the USDA launched its Stream-lined Inspection System for Cattle (SIS-C). Under this new arrangement, the meatpacking houses as-signed their own employees to do the inspections at five pilot plants. When USDA inspectors did occa-sionally visit them, company officials were frequently tipped off ahead of time about the time of their ar-rival. Not having to bother with federal inspectors on a daily (or even weekly) basis, packing-house owners speeded up their slaughtering lines at those five plants. This produced more beef at lower costs, but it was filthier than before ("Re-port Calls for Streamlining Federal Meat Inspections," Associated Press, September 17, 1990).

A 1992 USDA study decided that the new SIS-C inspection program was functioning just fine, be-cause the five plants "were no dirtier" than the oth- ers,—so it extended its "streamlined inspection sys- tem" to all the others ("Do Streamlined Beef Inspections Work?" Los Angeles Times, June 18, 1992).

But the federal inspectors knew the truth of what was happening; and interviews of some revealed that, under the new system, the meat was in worse condition than before. At SIS-C slaughterhouses, visibly diseased animals (cattle infected with

measles and tapeworms, covered with abscesses) were being slaughtered. Poorly trained company inspectors were allowing the shipment of beef contaminated with fecal material, hair, insects, metal shavings, urine, and vomit.

On April 30, 1992, the ABC News show, *Prime-Time Live*, broadcast an investigation of the new system for cattle. It had obtained corporate docu-ments showing that some USDA visits were known in advance. Also shown were video shots of meat covered in feces being processed at a processing plant in Greeley, Colorado. (Also see "Unhappy Meals: Colorado Meat Plant Blasted for Disease and Filth," States New Service, June 11, 1992; "USDA is Sued: Where's the Beef Report? Public Interest Group Charges System Lets Dirtier, More Dangerous Meat Reach Con-sumers," Washington Post, July 10, 1990.)

You might find it of interest that, under USDA regulations, whenever a meatpacking company voluntarily decides to pull contaminated meat from the market, it is under no legal obligation to inform the public—or even state officials—that a recall is taking place! In this way, the public does not learn what has happened, but people who are sickened by E. coli 0157:H7 are likely to be misdiagnosed and possibly die as a result. All because state health authorities have not alerted hospitals about the problem.

As an added protection for the meatpackers, the USDA now informs the public about every Class I (non-voluntary) recall that it initiates, but it does not reveal exactly where the contaminated meat is being sold (unless it is being distributed under a brand name at a retail store). This pro-

tects the large fast-food restaurant chains and franchises. But you will never hear their names mentioned in a recall. Both the USDA and the meatpackers argue that details about where a com- pany has distributed its meat must not be revealed in order to protect the firm's "trade secrets." But state health officials have attacked the USDA policy, arguing that it makes outbreaks much more difficult to trace and puts victims of food poisoning at much greater risk. So eat your burgers at your own risk. You will receive no warn- ings ("Stealthy Meat Recalls Leave Consumers in Dark," Denver Post, May 13, 1999; "Recalls Present Tough Decision for Food Companies," Food Chemical News, May 4, 1998; "Backlash: Recalls," Food Processing, August 1, 1999; "Recall of Meat and Poultry Products," FSIS Directive, January 19, 2000).

In February 1999, when one of the packers recalled 10,000 pounds of ground beef laced with small pieces of glass, the company would disclose only that the meat had been shipped to stores in Florida, Indiana, Michigan, and Ohio. Nei- ther the processing plant nor the USDA would pro- vide the names of those stores. "It's very frustrat- ing," an Indiana health official told a reporter, ex- plaining why the beef containing broken glass could not easily be removed from the supermar- ket shelves. "If they don't give [the information] to us, there's not much we can do" (quoted in Allison Young and Jeff Taylor, "Stealthy Meat Recalls Leave Consumers in Dark," Denver Post, May 13, 1999).

To date, Congress has turned down bills (four of them between 1996 and 1999) which would empower the USDA to fine meatpackers when

they sent out bad meat. Yet the government regularly uses fines as a means for regulatory enforcement in the airline, automobile, mining, steel, and toy industries. "We can fine circuses for mistreating elephants," Secretary of Agriculture Dan Glickman complained in 1997, "but we can't fine companies that violate food-safety standards" (quoted in Carol Smith, "[Need for] Overhaul in Meat Inspection No Small Potatoes, Official says," Seattle Post-Intelligencer, January 29, 1998).

RADIATION—In order to solve the problem in another way, the meatpacking industry and the USDA have been advocating the irradiation of meat by radiation from nuclear waste! ("Titan to Put Whammy on Food Bacteria," in San Diego Union- Tribune, May 18, 1999).

Most irradiating facilities have concrete walls that are six feet thick, employing cobalt 60 or ce-sium 137 (a waste product from nuclear weap- ons plants and nuclear power plants), to create highly charged radioactive beams. The Titan Cor- poration is a leader in developing equipment for this purpose ("Beef Industry Recommends Irradiation Rule Includes Ready-to-Eat Meats," Food Labeling News, June 23, 1999).

So far, widespread introduction of the process has been impeded by the fact that most consumers do not want to eat food exposed to radiation. The only way to do justice by the situation is to use labels, warning the consumer that the product is irradiated. Let the people decide what kind of meat they want ("Food Irradiation Acceleration," Packaging Digest, July 1, 1999; "Pasteurized Foods in Your Fu-

ture?" Food Management, October 1999).

An ongoing warfare by the meat and packing industries has been carried on for years against regulation of their activities or public disclosure of the meat crisis. Here is one concluding incident:

A ground beef plant in Dallas, Texas, failed a series of USDA tests for Salmonella in the summer and fall of 1999. As much as 47% of their ground beef contained Salmonella. This was five times higher than what USDA regulations allowed ("Plant that Failed Salmonella Tests Challenges Screening System," Dallas Morning News, December 10, 1999). Every year in the U.S., food tainted with Sal-monella causes about 1.4 million illnesses

500 deaths; and high levels of Salmonella in ground beef indicate high levels of fecal con- tamination. So one would expect this discovery to be regarded as a serious matter (*Meade*, et al., "Food- Related Illness and Death").

Despite the test results, the USDA continued to purchase thousands of tons of meat from that firm for distribution to schools. Indeed, it was one of the nation's largest suppliers to the school meals program, annually providing as much as 45% of all the ground beef eaten in American schools (*Chi-cago Tribune, December 14*, 1999).

On November 30, 1999, the USDA finally sus-pended purchases. The next day, with the full back- ing of the National Meat Association, the firm sued the USDA in federal court, claiming that Salmo-nella was a natural organism, not an adulterant! This had been the first plant shutdown ever at-tempted by the USDA. It lasted one day, for a

federal judge ordered a reversal ("Judge Rebuffs USDA; Agency Tried to Close Dallas Plant," Dallas Morning News, May 26, 2000). Within a short time, the USDA was once again allowing the company to supply ground beef to the nation's schools ("USDA Purchased Meat from Texas Plant after Con-tamination Cited," Atlanta Journal, December 4, 1999).

2 - FOOD-BORNE DISEASES

"Food-borne diseases" is the technical name for diseases people get from eating animals, which are caused (not the diseases the ani- mals themselves have) from bacteria on small bits of contamination (primarily manure) which were on the animal when it left the processing plant on the way to the consumer.

Every year in America, about 200,000 people are sickened by some type of food-borne dis- ease. Of that number, 900 are hospitalized and 14 die (Meade, et al., "Food-Related Illness and Death"). The total number in a year's time in the United States is 76 million illnesses, 325,000 hos- pitalizations, and 5,000 deaths.

The Centers for Disease Control and Prevention (CDC) estimates that more than one-third of all the people living in the United States suffers from at least one food poisoning each year! There is also evidence that the intensity and long-last- ing effects of these attacks has increased in num-ber in the past few years. Most of these cases are never reported to authorities or properly diagnosed. So only a small fraction of the total number that actually occur is reported (ibid.).

This confusion is due to the fact that the acute phase of a food poisoning (which, in minor cases, generally is a few days of diarrhea and gastrointes- tinal upset) is similar to the onset of an infectious disease (James A. Lindsey, Emerging Infectious Dis- eases, October/December 1997).

Recent studies disclose that many food-borne pathogens can precipitate long-term ailments, such as heart disease, inflammatory bowel disease, neurological problems, autoimmune disorders, and kidney damage. (See Tauxe, "Emerg- ing Food-borne Diseases.")

"Newly emerging pathogens" are diseases which formerly were not a problem for humans. We, for-merly, lived carefully and used clean methods to prepare our food. But many of the new outbreaks result from the meat served in fast-food restau- rants or packaged meats in grocery stores.

E. coli 0157:H7 is the most dangerous E. coli bacterium at the present time. E. coli bacte- ria are normally found in the billions in the intes- tines of every mammal, including humans. But when a person swallows live E. coli 0157:H7, it can kill him. E. coli 0157:H7 was first isolated in 1982 and would be no problem to us—if new assembly line methods of raising food animals, slaughtering them, and packaging the contents had not been in- troduced. (See Armstrong, et al., "Emerging Foodborne Pathogens.")

Several *E. coli* types (called *serotypes*) are known. Two of the most dangerous are 0157:H7 and 0104:H21. Both cause severe bloody diar- rhea and, in children, hemolytic uremic syn-

drome (kidney failure and destruction of red blood cells), which can lead to death. The bac- teria can be found in beef, milk (raw and pas- teurized), sausage, apple cider, and venison. Research and news reports focus attention on 0157:H7, so we will also.

Cattle infected with *E. coli 0157:H7* can appear healthy and show few signs of illness. There may have been some infections from it years ago, but the wide dispersion of the disease did not occur until huge feedlots, slaughterhouses, and hamburger grinders took over the meat industry. Making more money is the name of the game, and people who eat meat are in far greater danger than ever before.

American meat production has never before been so automated and centralized. Thirteen large packinghouses now slaughter most of the beef con-sumed in the U.S. ("U.S. Meat Slaughter Consolidat- ing Rapidly," USDA Food Review, May 1, 1997). This meat-packing system arose in order to supply the nation's fast-food chains. But it has resulted in a massive increase in food-borne pathogens in the meat served to you.

In addition to *E. coli 0157:H7* and *0104:H21*, over the past two decades scientists have discov- ered more than a dozen other new food-borne patho- gens, including *Campylobacter jejuni, Crypto- sporidium parvum, Cyclospora cayetanensis, List- eria monocytogenes,* and *Norwalk-like viruses* (cited in Tauxe, "Emerging Foodborne Diseases").

Yet this is only the tip of the iceberg. Incredibly, the CDC estimates that more than three-fourths

of the food-related illnesses and deaths in the U.S. are caused by infectious agents that have not yet been identified! (See "Food-related Illness and Deaths.")

But, be assured, neither the meat industry nor the fastfood industry wants the situation exposed. They would rather that you not know the facts, and keep eating their ham- and beefburgers, fried chicken, and other delicacies.

These pathogens, which have only recently been discovered, tend to be carried by apparently healthy animals. Sometimes the problem lies in the gar-bage (including manure) the animals ate in their "high-protein feed pellets." Sometimes it is the han-dling as waste matter came in contact with the carcasses of those animals during slaughtering or processing.

A nationwide study, published by the USDA in 1996, revealed that 7.5% of the ground beef sam- ples taken at processing plants were contami- nated with Salmonella, 11.7% were contaminated with Listeria monocytogenes, 30% were con- taminated with Staphylococcus Aureus, and 53.3% were contaminated with Clostridium per- fringens

("Nationwide Federal Plant Raw Ground Beef Microbiological Survey, August 1993-March 1994," USDA, Food Safety and Inspection Service, April 1996). All of those pathogens can make people sick.

Food poisoning by *Listeria* generally requires hospitalization and proves fatal in about 20% of the people whom it infects (*Meade*, et al., "Food-re-lated Illness and Death").

How did the pathogens get on that meat you eat?

In the USDA study, **78.6% of the ground beef con-tained microbes that are spread primarily by fecal matter.** In the medical literature, there is con-tinual reference to "coliform levels," "aerobic plate counts," and similar terms. What it all means is that there is manure in the burgers you are eat-ing.

The question is not why they are there. Research- ers know the reason. The question is why do you keep eating the stuff. Every dollar you pay for such inferior food only enriches the industry which is pro- viding you and your loved ones with such indecent food fare.

It is an interesting fact that, back in the first part of the 20th century, ham- and beefburgers were con- sidered unsafe foods. It was well-known back then that the burger-grinding machine in the local meat markets was used to process the most inferior cuts of meat.

Yet today, people think that burgers are the greatest thing in the world. And those burgers changed the food habits of Americans. By the early 1990s, beef production was responsible for almost half of the employment in American agriculture, and the annual revenues generated by beef were higher than those of any other agricultural commodity in the U.S. The average American ate three burgers a week (National Cattlemen's Beef As-sociation Fact Sheet). More than two-thirds were bought at fast-food restaurants (San Diego Union-Tribune, August 27, 1997).

Of that number, **children between the ages of seven** and 13 ate more burgers than anyone else

(a survey by McDonald's, cited in Boas and Chain, Big Mac, p. 218). Yet they are the most susceptible to severe illness and death from E. coli 0157:H7. In January 1993, physicians at a Seattle hospital no-ticed that an unusual number of children were being admitted with bloody diarrhea. Some were suffering from hemolytic uremic syndrome, a pre-viously rare disorder that causes kidney dam-age. Health officials soon traced the outbreak to undercooked burgers at local fast-food restaurants. Before it was over, more than 700 people in at least four states were sickened by those burgers, more than 200 were hospitalized, and four died. Most of the victims were children (CDC report, April 16, 1993). One of the first to become ill was Lauren Rudolph. She ate a hamburger at a San Diego fast-food res-taurant a

week before Christmas. Admitted to the hospital on Christmas Eve, she suffered terrible pain, had three heart attacks, and died in hermother's arms on December 28, 1992. She was only six years old. In the eight years following that outbreak, approximately half a million Americans, the ma-jority of them children, have been made ill by E. coli 0157:H7. Thousands have been hospital- ized and hundreds have died. Yet the government does little to regulate, much less stop, the carnage (based on Meade, et al., "Food-Related Illness and Death").

As mentioned earlier, *E. Coli* is a bacterium which normally is in everyone's intestines. These tiny creatures help us digest food, synthesize B vitamins, and guard against dangerous organisms. *E. coli* 0157:H7 is highly dangerous. It can release a

powerful toxin, called a *verotoxin* or *Shiga toxin*, which attacks the lining of the intestine.

In about 4% of reported cases, the Shiga tox- ins enter the bloodstream, causing hemolytic uremic syndrome (HUS) and the destruction of vital organs. These Shiga toxins can cause sei- zures, neurological damage, and strokes. About 5% of the children which develop HUS are killed by it. Those who survive are often left with per- manent disabilities, such as blindness or brain damage (Meade, et al., "Food-Related Illness and Death").

The elderly, children under five, and people with impaired immune systems are the most likely to experience the worst effects of E. coli 0157:H7. It is now the leading cause of kidney failure among children in the U.S. ("Isolation of E. coli 0157:H7 from Sporadic Cases of Hemorrhagic Colitis: United States," Morbidity and Mortality Weekly Report, CDC, August 1, 1997).

What do physicians use to treat the infection? There is hardly any help that they are able to pro- vide. About all they can do is give fluids, blood trans- fusions, and kidney dialysis. Antibiotics are useless and can make the infection worse by killing the pathogen, thus suddenly releasing all its Shiga tox- ins.

E. coli 0157:H7 is easy to transmit and can live in freshwater or seawater! It can live on dry kitchen countertops for days and in moist envi-ronments for weeks. It is extremely difficult to get it off countertops, sinks, food utensils, and refrig-erators. It is resistant to acid, salt, and chlorine;

and it can withstand freezing as well as tempera-tures up to 160° F. How likely are you to boil the top of your kitchen counters, your sink, your eating uten-sils, or your plates? Put some infected meat on them, and you are in deep trouble.

To be infected by most food-borne pathogens (such as *Salmonella*), you have to consume a fairly large dose: at least a million organisms. But **an in- fection with** *E. coli* 0157:H7 has been shown to be caused by as few as five organisms! One tiny, uncooked particle of ham- or beefburger meat can have enough of the pathogens to kill you (*Arm-strong*, et al., "Food-borne Pathogens").

Small traces of infected raw manure are the cause. People can be infected with this amazing or- ganism by drinking contaminated water, swimming in a contaminated lake or water park, or crawling on a contaminated carpet. Eating undercooked ground beef is a common cause. But you can also get it from contaminated salad greens, raw milk, and unpasteurized apple cider. The pathogen can also be spread by the feces of deer, dogs, horses, and flies (ibid.).

The reason so many cattle and hogs now transmit it is because they are raised in feedlots, where they feed while standing in a slushy pile of manure and urine.

During the Dark Ages, people threw their chamber pots out in the streets and epidemics frequently occurred. Now hogs and cattle live in pools of manure, and you eat them when they are killed. Feedlots are very efficient methods for spreading *E. coli 0157:H7* throughout the population of

our nation. They "recirculate the manure," and that particular bacterium can replicate in cattle troughs and survive in manure (outside the in- testines) for up to 90 days (P. Hammel and H.J. Cordes, Omaha World-Herald, December 15, 1997).

The underlying problem is that the govern-ment should be protecting us from infected meat. It should require much slower operations at process- ing plants. Microbiological analysis should be used. Processing plants should reimburse the government for all inspection and related costs. It should heavily fine firms which ship out infected meat. It should close down those who refuse to clean up their op- erations. It should provide complete disclosure to the public when there is an infectious outbreak. It should require processing plants to provide relevant information.

NIH REPORTS THAT PRIONS CAN MAKE NOR- MAL PROTEINS INTO PRIONS!—"A normal PrPC pro-

tein assumes a new form when it becomes a prion [called a PrPSC]. PrPC spirals out of its tight helical shape into a more open structure. Exactly how [or why] this happens is not clear; but it may involve the coupling of one PrPC with one PrPSC for a molecular 'dance,' in which PrPc follows the lead of PrPSC to fold-unfold the way PrPSC is folded-unfolded.

"Dr. Brian Caughey, a researcher at the National In- stitutes of Health's Rocky Mountain National Laboratory, has been successful in getting PrPC molecules to convert to PrPSC molecules in test tubes. He mixes the two to- gether in the test tube and voila! Many PrPSC molecules result. 'There is conversion of the normal proteins to altered proteins. But are the altered proteins infectious? We don't know that yet,' says Caughey."—nih.gov.

APPENDICES

MAJOR HUMAN DISEASES RESULTING FROM MEATEATING

1 - CARDIOVASCULAR DISEASES

THE CHOLESTEROL FACTOR—If you want to have a good heart, you need to bring your cholesterol level down to 150 or below. Yet the American Medical Association, in spite of the fact that its journal announced in 1961 that "A vegetarian diet can prevent 97% of our coronary occlusions," has, under pressure from the meat industry, only told its doctors to tell their pa- tients to "eat less red meat, watch the intake of fat, and cut down on eggs," in the hope of getting their choles- terol down to the "normal" 200 level. But a 200 level, maintained over a period of time, can killyou.

Over 5,000 residents of Framingham, Massachusetts have been studied since 1948. Researchers have been looking for risk factors of coronary heart disease. In all that time, they have not found a single person to have a heart attack whose blood cholesterol was below 150! Remember that. Above that number, people have them— without any advance warning.

"Eat all the meat you want, but just don't eat much fat," is what you hear every day. But a study (in Italy during 1980) of 127 people put on a "low-fat" (25% of caloric intake as fat) diet containing meat—only lowered their cholesterol by a statistically insignificant 2.8% af- ter four weeks. Then the subjects in the study were placed on soybeans instead of meat for two weeks— and no patient had less than a 10% drop (G.C. Descovich, "Multicenter Study of Soybean Protein Diet

for Outpatient Hypercholesterolemic Patients," Lancet, 2:709, 1980).

HIGH BLOOD PRESSURE—Hypertension is a complicating factor in both cardiovascular and cerebrovascular disease. It is the most common reason for a visit to a doctor in America, and more prescriptions are written for hypertension than for any other disease. Young people happily eat their meat and fat; but, as the years pass, their arteries narrow from cholesterol de- posits. This eventually leads to fatal heart attacks or strokes.

Repeatedly, studies have been shown that **meat eat-ing is a major cause of high blood pressure**. A study published in the *New England Journal of Medicine* com- pared 115 vegetarians with the same number of meat eaters. **The systolic blood pressure** (when the heart contracts) of the vegetarians was 9.3% lower than that of meat eaters. The diastolic (when the heart relaxes) was 18.2% lower (*New England Journal of Medicine*, 292, 1975).

REVERSING CORONARY DISEASE—Dr. Dean

Ornish has a program for reversing the course of coronary disease with a truly low-fat (10% fat) plant-based diet. The only animal products allowed are nonfat milk, nonfat yogurt, and egg whites. After a year on the pro-gram, 82% of his patients demonstrated some measurable average reversal of their coronary artery block- ages. Those were people who knew they were going to die soon if they did not change their ways. Those who went on a vegetarian (vegetables without meat) diet did far better.

BYPASS SURGERY—Another "benefit" of meat eat- ing is heart bypass surgery. **Years on a meat diet clog**

the tiny coronary arteries which nourish the heart muscle. A major study was done on 780 heart patients. Half had a bypass operation and the other half did not. It was found that longevity rates were not improved by surgery! (Julian Whitaker, Reversing Heart Disease, p. 25). It is well-known in the medical field that other or- gans, especially the brain, are damaged during bypass operations. This apparently is due to the fact that, dur-ing the operation, the blood must be pumped through the body through a heart-lung machine. Nearly all pa- tients are damaged to one extent or another; most suffer problems which last for the duration of their lives (Julian Whitaker, Reversing Heart Disease, pp. 36-37). It is far better to live in such a way that you do not need to have bypass surgery!

2 - CANCER

 $\label{eq:mean_mean_mean} \begin{tabular}{ll} MEAT AND CANCER—The evidence is abundant and clear: Meat is a leading cause of cancer. \end{tabular}$

German vegetarians: Over 1,900 vegetarians were studied by the German Cancer Research Center, which found that **they had only 56% of the usual amount of all types of cancer** (R. Frentzel-Beyme, et al., "Mortal- ity among German Vegetarians: First Results after Five Years of Follow-up," Nutrition and Cancer, 11 (2), 1988,

pp. 117-126).

American Adventist men: The study, cited at the beginning of this book, mentions that half of the Sev- enth-day Adventist men were vegetarian; they ate an av- erage of 50% more fiber than the general population and suffered 55% less prostate cancer than other American males.

COLON CANCER—The Association for the Advance-ment of Science found that **people who eat meat are far**

more likely to develop colon cancer. Meat and choles- terol in the colon are the major causes of colon cancer, which is rapidly becoming a leading form of carcinoma (cancer) in the Western world (Science, February, 1974).

The National Cancer Institute investigated deeper into the matter, in order to determine the amount of danger in each type of food. It found that **the incidence of co- lon cancer was over a hundred times worse when any of a variety of dead animals were eaten.** Beef, pork, chicken, shellfish, crabs, river or ocean fish, shrimp; it matters not. They all produced colon cancer (Journal of the National Cancer Institute, December 1973, p. 1771).

BREAST CANCER—Many animal studies have shown that a high-fat diet promotes breast cancer tu-mors. Human breast cancers were implanted in mice, which were then fed various diets. Tumors in mice on a high-fat diet grew and spread much more quickly. The best diet was one high in cruciferous vegetables—broc- coli, cabbage, collards, brussels sprouts (Robert, Kradjian, Save Yourself from Breast Cancer, 1994, pp. 51-52).

Nations like Thailand and El Salvador, which have a comparatively low-fat, plant-based diet, have the lowest breast cancer mortality rates. The highest rates of breast cancer are in the "high-fat countries": America, Neth-erlands, Britain, Denmark, Canada, and New Zealand (Robert, Kradjian, Save Yourself from Breast Cancer, 1994, p. 44).

OTHER CANCERS—Results of a 36-country study were reported in 1991. A strong and direct correlation between consumption of dairy and animal fat and the incidence of prostate cancer, rectal cancer, lung cancer, and breast cancer was clearly shown (Michio Kushi, The Cancer Prevention Diet, 1994, p. 241).

Seventh-day Adventists in the U.S. have markedly lower rates of cervical and ovarian cancer than the rest of the population. A 1989 Harvard study found that **women who ate more dairy products had more ova- rian cancer** (*Kushi*, *p.* 226).

In 1982, the National Academy of Sciences made this statement: "In summary, the incidence of prostate can- cer is correlated with other cancers associated with diet,

e.g. breast cancer. There is good evidence that an increased risk of prostate cancer is associated with certain dietary factors, especially the intake of high fat and high-protein foods, which usually occur together in the diet. There is some evidence that foods rich in Vitamin A

.. and vegetarian diets are associated with a lower risk."— National Academy of Science, 1982 statement, quoted in Kushi, p. 241.

3 - OTHER MAJOR DISEASES

DIABETES—According to the Loma Linda study, Seventh-day Adventists have about half the risk of devel-oping diabetes. A study of diabetics revealed that **those placed on a high-fiber vegetarian diet required 73% less insulin therapy than those on standard diets** (D.A. Snowden and R.L. Phillips, American Journal of Public Health, 1985, p. 75).

Checking into this, it was found that diabetics often need insulin shots—not because their bodies do not pro-duce enough insulin, but because **the insulin produced fails to function properly—because there is too much fat clogging the blood stream.** It was concluded that a low-fat, high-fiber diet can do more to help most diabet-ics than insulin pumps and medication (J.W. Anderson, "Plant Fiber and Blood Pressure," Annals of Internal Medicine, 1983, p. 98).

Throughout the world, "diabetes is rare or nonex- istent among peoples whose diets are primarily grains, vegetables, and fruits. If these same people switch to rich meat-based diets, their incidence of diabetes bal- loons."—*Robbins, Diet for a New America, 1987, p. 277.*

OSTEOPOROSIS—Older people suffer greatly from bone loss. It is caused by a lack of calcium in the diet, so the solution is to drink a lot of calcium-rich milk. Right? Wrong. **Oddly enough, eating meat or drinking milk tends to aggravate the problem** (Cox, The New Why You Don't Eat Meat, 1992, p. 153).

The Chinese eat almost no dairy products and get all their calcium from plant sources, yet they rarely have osteoporosis. Only 6% of their diet is from animal protein (*Robbins*, pp. 193-194).

Eskimos do not drink milk, but do eat a calcium-rich diet of meat and fish;—yet they have a very high rate of osteoporosis (*Robbins*, p. 194).

Bone loss was examined in 1,600 women, half of whom ate meat while the other half were vegetarian. By the time they reached the age of 80, vegetarian women had lost only about half as much bone mineral as meat eaters (*Cox*, *p*. 153).

There is a reason for this: Because the human body cannot store excess protein, it excretes it through the urine, taking calcium with it. Excess proteins load the body with acids which cannot easily be eliminated. In order to chemically neutralize the protein, so it can be excreted, calcium must be taken from the blood and bones and united with the acids. This effort to excrete those acids overloads the kidneys and weakens them.

What should you eat in order to obtain an adequate calcium diet? Eat a variety of calcium-rich vegetarian foods: green, leafy vegetables, such as broccoli and kale, areamong the best. Also good are almonds, sesame seeds,

molasses (blackstrap is the richest), garbanzo beans, and tofu.

GALL BLADDER PROBLEMS—What about the few people who eat meat and fat, and are still able to main- tain lower cholesterol levels? They have a superior abil- ity to excrete it into the liver, but research had disclosed that **the fat builds up there and causes gallbladder and gallstone problems** (90% of gallstones are made of cho-lesterol).

ARTHRITIS AND RHEUMATISM—There are sev-

eral types of strong acids in meat: uric acid, pyruvic acid, etc. These must be eliminated from the body. But, **over a period of time**, **those acids tend to settle in the muscles and joints, where they work great damage**. This can lead to painful and even crippling muscle aches and joint problems in later years. **Gout and other forms of arthritis are especially traceable to meat eating**.

OTHER DISEASES—Eating a low-fat vegetarian diet will reduce the risk of ulcers, intestinal disorders, hypoglycemia, kidney stones, gallstones, asthma, impotence, and even anemia (McDougall, The McDougall Program for a Healthy Heart, 1996, p. 24).

OBESITY—Everyone knows that being overweight eventually leads to one or more of a multitude of physical problems. **The nations whose populations consume a lot of meat are the ones with the most overweight problems.** In America alone, \$40 billion is spent annually on various "diets" to reduce weight (*Richard Klein, New York Times, July 14, 1997*). But in China obesity is extremely rare. Yet it is a remarkable fact that the average Chinese eats 25% *more* calories than Americans do! But they get their calories in greens, rice, and soy- beans (*Cox, p. 9*).

The U.S. now has the highest obesity rate of any industrialized nation in the world. More than half of all American adults and about one-fourth of all American children are now obese or overweight. These proportions have soared during the last few decades, along with the consumption of fast foods ("Land of the Fat," Time International Edition, October 25, 1999). The rate of obesity among American adults is twice as high today as it was in the early 1960s. The rate of obesity among American children is twice as high as it was in the late 1970s (Science, May 29, 1998).

Today, about 44 million American adults are obese. An additional 6 million are "super-obese"; they weigh about a hundred pounds more than they should. No other nation in history has gotten so fat so fast (*Journal of the American Medical Association, October 27, 1999*). In 1991, only four states had obesity rates of 15% or higher; today at least 37 states do. "Rarely do chronic conditions such as obesity spread with the speed and dispersion characteristic of a communicable disease epidemic" (*ibid.*).

To attract more customers, the fast-food meat shops keep offering bigger burgers, fries, and soft drinks. The more that people eat outside the home, the more calo- ries, fat, and protein they absorb—and the less fiber, vitamins, or minerals.

The arrival of McDonald's in 1971 accelerated the shift in Japanese eating habits. During the 1980s, the sale of fast food in Japan more than doubled; the rate of obesity among children soon doubled, too. Today about one-third of all Japanese men in their thirties (the first generation raised on the new fast-food diet) are over- weight. **Heart disease**, **diabetes**, **colon cancer**, **and breast cancer—diseases linked to diets low in fiber and high in animal fats—are becoming endemic** ("De-

cline of the Japanese Diet: MacArthur to McDonald's," East West, October 1990). The same situation is occur-ring in Britain and other overseas nations invaded by our quick-meat restaurants ("7,000 Pounds: Fast Food is Taking Over the World," USA Today magazine, May 1, 1999).

CROHN'S DISEASE—Crohn's disease is often a devastating and incurable intestinal illness. It can cause such symptoms as bloody diarrhea, fever, severe abdominal pain, arthritis, and incapacity, as well as obstruction of the intestines that requires surgery. This disease has bewildered medical scientists for years, as a problem without any known cause.

But recent research has established a link to infection by a germ called mycobacterium paratuberculosis (S.N. Cho, P.J. Brennan, et al., "Mycobacterial etiology of Crohn's disease: serologic study using common my- cobacterial antigens and a species-specific glyColipid antigen from Mycobacterium paratuberculosis," Gut, November 27, 1986. / "Mycobacterium paratuberculosis implicated in Crohn's Disease," Gastroenterology Ob- server, November-December 1995).

This bacterium with the long name is extremely common in animals like sheep and cattle. It causes a chronic intestinal disease called Johne's disease, which affects approximately 25% of U.S. dairy cattle ("Myco-bacterium paratuberculosis implicated in Crohn's Dis-ease," Gastroenterology Observer, November-Decem-ber 1995).

An ominous aspect of this disease is that **healthy-appearing cows can be infected and transmit the germ in their milk.** In one study of an infected Ohio herd, over one in four healthy-appearing cows had the germ in their stools and one in 12 had the germ in their milk (*R.N.*

Streeter, G.E. Hoffsis, et al., "Isolation of Mycobacte- rium paratuberculosis from colostrum and milk of sub- clinically infected cows," American Journal of Veteri- nary Research, October 1995). It is a well-attested fact that **this germ can survive common pasteurization** (I.R. Grant, H.J. Ball, et al., "Inactivation of Mycobacterium paratuberculosis in cows' milk at pasteurization tem- peratures, Applied Environmental Microbiology, Feb- ruary 1996).

DISEASES IN FOOD ANIMALS

It is remarkable how many diseases are in ani-mals. Unfortunately, a number of them can be passed on to humans.

There are six types of animal diseases: bacte- rial diseases, fungus diseases, viral diseases, para- sitic diseases, hereditary diseases, and diseases caused by environmental factors.

Animals suffer from so many diseases. Bacteria causes white diarrhea, which is often fatal to chicks. Distemper and hog cholera result from virus infection. *Coccidiosis*, a protozoan infection, is a destructive dis- ease of poultry; it also attacks cattle, hogs, and cats. As this book is written, Europe, America, and an increas- ing number of other nations are having cattle problems involving mad cow disease, hoof and mouth disease, and *Pfiesteria piscicida* pollution of rivers.

A partial listing of the diseases, a number of which humans can contract from animals (such as **tuberculo-sis** or **brucellosis** from raw milk), are listed below. For further information, check the bibliography at the back of this book.

Question: Can hoof and mouth disease (errone- ously called "foot and mouth disease") cause sickness

in humans? Yes, it can. "Humans have been known to catch hoof and mouth disease from animals. Symptoms in humans are sometimes confused with the flu. For those who are sick or elderly, it could be deadly" (Robert Cohen, Earthlink, March 15, 2001).

BACTERIAL DISEASES—Some bacteria produce powerful poisons or toxins. This would include the **Botu-linum** bacillus, the **Tetanus bacillus**, and the bacillus causing **gas gangreen**.

Other bacteria cause local or general death of body tissues, block the flow of blood, or cause severe irritation. One of the most widespread of these is *Salmonel-losis*, or any disease caused by *Salmonella* bacteria. One of these is *Pullorum* disease, caused by *S. pullorum*, which is a continual threat to the chicken and turkey industry.

Leptospirosis, caused by spiral bacteria of the ge- nus **Leptospira**, kills cattle, dogs, and humans. This bacteria is often in ponds, lakes, and other bodies of water. Rodents may also carry the infection.

Bacteria of the genus *Mycobacterium* can produce **tuberculosis**. Breathing can pass this from man to ani-mals and vice versa. Milk from tubercular cattle can also contain tuberculosis.

Anthrax, caused by *Bacillus anthracis*, affects both humans and domestic animals. Resistant spores are car- ried in the hair, hides of animals, or in floodwaters and can easily be transmitted.

Pasteurellosis, or any infection caused by bacterium of the genus *Pasteurella*, such as **fowl cholera** caused by *P. multocida*, which affects domestic poultry, rab- bits, and other animals.

There are tiny, soft-walled bacteria of the genus *My-coplasma*, which cause a variety of diseases in animals

and humans, including **infectious sinusitis** in turkeys, **pleuropneumonia** in cattle, and **chronic respiratory dis- ease** in chickens.

Parrot fever, formerly thought to be a virus, is now believed to be caused by bacteria of the genus *Chlamy- dia*. Some serious diseases that occur in both humans and animals are in this group.

VIRALDISEASES—There are a broad variety of virus infections. They include equine infectious anemia, Newcastle disease, hog cholera, fowl pox, rabies, ca-nine distemper, encephalitis, along with many others.

Several viral agents cause tumors in poultry, known as leukosis complex.

Influenza viruses cause serious problems in swine, horses, and birds.

Some viruses spread from mother to offspring through the placenta or through the egg. Others are very resistant and can survive in dust. Yet others require in-timate contact to be contagious. Still others are spread by the bite of arthropods (spiders).

As with many other animal diseases, many of their viruses can be passed on to man.

FUNGUS DISEASES—Many serious diseases in ani-mals are caused by fungus diseases, and some can be transmitted to humans. Aspergillus fungi may cause necrosis of the lungs, the nervous system, and other organs. A yeast-like fungus, Candida albicans, may cause death in turkeys and other animals. It is also a problem for humans. Dust-borne fungi, such as Coc-cidioides immitis and Histoplasma capsulatum, pro-duce lung disease or generalized disease in both animals and man.

PARASITIC DISEASES—Parasites attack all ani-

mals, and range in size from tiny **protozoa** to meter-length **kidney worms**. When people eat animals, they can take in some of those worms if the meat is not well-cooked.

Protozoan diseases include the coccidiosis, which affect geese, rabbits, as well as other creatures. The ma-larias include Plasmodium, Leucocytozoon and Hae-moproteus protozoa. An example of flagellate infections includes trichomoniasis (Trichomonas gallinae in birds, T. fetus in cattle) and trypanosomiasis, also known as nagana, surra, and dourine, caused by flagellates related to the agent of African sleeping sickness.

Worms include helminths, and comprise a large group of parasites, including roundworms (nematodes), flukes (trematodes), tapeworms (cestodes), thorny- headed worms (acanthocephalan), and tongue worms (linguatulidae).

The adult **tapeworms** are found in animal intestines; and their larval stages often do great damage in body tissues of secondary hosts (including people). Larval **dog tapeworms** (**echinococcosis**) form large cysts in the liver, lungs, and other organs of humans and animals.

Roundworms, while in the migrating larval stage, cause great damage to lungs and other organs. Capil-laria worms attack the lining of the stomach. Adult heart-worms, Dirofilaria immitis, live in the hearts of dogs and produce microscopic larval stages which swim in the blood. Larvae of Strongylus vulgar causes arterial obstruction, with resultant digestive troubles and even lameness.

We will not take the space in this book to discuss infections which food animals themselves have, other than the **spongiform diseases**. The data could fill a large book. The diseases include **carcinoma** (**cancer**) in cattle, hogs, and chickens; **mastitis** (**udder infection**) in cows,

worms, **trichinosis**, and many other infections. Information on this is available from various sources. Some of these little creatures produce interesting effects. They do not all kill; some just maim for a lifetime. For example, **trichina** are tiny worms which, when infected pork is eaten, travel through your bloodstream and burnow into your muscles. They remain there the rest of your life, causing minor aches and pains.

EMERGING ANIMAL DISEASES

"Emerging diseases of animals" is the technical name for new animal diseases—which apparently never before existed! This is a very serious matter. The animal kingdom is becoming a reser-voir of disease.

"United States Animal Health Association, 1997 Committee Reports, Committee on Foreign Animal Dis- eases.

"Emerging Diseases: An Urgent Issue in Animal Health, Corrie Brown, DVM, Ph.D. Professor and Head, Department of Pathology, College of Veterinary Medi-cine, University of Georgia, Athens, GA.

"In recent years, emerging diseases have moved to center stage in the biomedical community. They have become the focus of numerous scientific reports and the subject of intensive experimental and epidemiological re- search. Most of the attention has been directed to emerging diseases of the human population; in fact, animal diseases are also emerging at an ever-increas- ing rate. The animal health community needs to be aware of the importance of emerging diseases and needs to increase preparedness for dealing with these

newdiseases.

"The factors responsible for emerging diseases in-clude [1] movement to a susceptible population, [2] dis-ruption of the environment, [3] crossing to a new spe- cies, and [4] changes in husbandry. All of these factors are inherent in the global village that exists today. In the following extended quotation, numbers within parenthe- ses refer to cited references listed on p. 122.

[1] "Concern about diseases moving to susceptible human populations captured the attention of the gen- eral public when **ebola** virus was found in Reston, Vir- ginia; and this event was loosely transformed into a best- selling book and movie. Shortly after, in a grisly life-imi- tates-art scenario, the world watched an outbreak of highly virulent **ebola** virus in people in central Africa. Animal diseases, while not stimulating the same level of awareness, have been moving around the world at unprecedented rates.

"In 1993, **foot-and-mouth disease (FMD)** was taken to Italy from Croatia; in 1995, it traveled from Turkey to Greece. Just this year, FMD moved to Taiwan, causing the destruction of 5 million pigs and costing \$5 billion in lost trade opportunities.

"Taiwan had been free of FMD since 1929; incidentally, the last outbreak of FMD in the United States occurred in 1929. Also this year, **hog cholera virus** got into Holland on a manure-contaminated truck from Germany, causing mass animal and economic disruption. With the current trends toward increased movement of animals and people around the world, the term 'ex-otic disease' may become oxymoronic.

[2] "Disruption of the environment has caused new diseases to emerge. All of the **hemorrhagic fevers** of humansfallintothis category. **Lymedisease** emerged as

a human health problem when people began to move closer to the tick vector and vice versa. In the animal health realm, examples of disease outbreaks related to environmental changes include **velogenic viscerotropic Newcastle disease**, **fowl cholera**, and **duck plague**—all occurring with increasing frequency, largely due to con-gregations of waterfowl as wetlands availability decreases

(5). **Phocine distemper** in the North Sea may well be the result of altered migrations due to overfishing (3). The periodic increases in toxic **dinoflagellates**, including **Pfiesteria piscicida**, are related to increased nitrogen content in the water, perhaps due to agricultural waste runoff. Ecotourism, which brings humans and all their microflora into remote areas containing endangered species, is an issue that deserves attention from animal health specialists.

[3] "Crossing to a new species. Bovine spongiform **encephalopathy** crossing the species barrier to humans in the form of variant Creutzfeld-Jakob disease has heightened general awareness about this method of dis-ease transfer (9). This crossing of species boundaries is a well-known phenomenon in numerous diseases of ani- mals. Canine parvovirus is one of the first extensively characterized examples (7). In recent years, canine dis-temper virus has adapted to African lions, causing high rates of mortality in the Serengeti Plain in North Tanza- nia. (1). A new disease of horses, first called equine morbillivirus, has been determined to come from a spe-cies of Australian bat (10). Finch conjunctivitis is a result of Mycoplasma gallisepticum crossing from chickens to house finches (4). A new concern on the ho- rizon is the issue of xenotransplantation. The intimate apposition of pig and human tissue raises possibilities of disease transference in both directions.

[4] "Husbandry changes are becoming increasingly recognized for creating disease emergence. Bovine spongiform encephalopathy may be the most obvious example, as a simple change in rendering procedures is thought to have precipitated this disease (8). Tuberculosis in elk and deer, due to captivity or winter feeding, has emerged as a serious problem (2). The occurrence of E. coli O157:H7, of great concern to the meateating general public, may be related to husbandry practices in the feedlot (6). Antibiotic resistance poses se- rious threats to the control of bacterial diseases: and considerable responsibility rests with animal hus-bandry practices. The recent advent of mammalian clon-ing makes monogenetic animal agriculture a possibility, a husbandry shift that could have devastating disease consequences. As we strive to feed the growing popula- tion of the world with updated and refined technologies, husbandry changes are inevitable.

"Because of all the factors inherent in our highly populated, industrialized, technologically advanced world, it is a certainty that new diseases of animals will continue to arise. With our brisk domestic and international trade, movement of disease agents to susceptible populations will continue to occur as there is ever-increasing traffic of animals and animal products. Environmental disruption, although receiving more preventive attention than in the past, will undoubtedly accelerate, with all the ramifications for new diseases. Growing possibilities of cross-species transfer of agents is inevitable as animals become more crowded and more creative and artificial habitat situations are created. New husbandry changes will continue to be implemented as we strive to devise new ways of growing food for a burgeoning human population.

"The role of animal health specialists in emerging diseases will be to maintain a vigilance with respect to detection of each new entity as it arises and to maintain an awareness to help prevent their occurrence. Another integral role to be played by this community is in the experimental research of both animal and human dis- eases. Since virtually all of the experimental manipula- tions are performed in animals, veterinarians with ad- vanced specialty training in laboratory animal medicine, pathology, and microbiology can provide the greatest ex- pertise on interpretation of changes in animal systems, and can help to make progress in the most informed and efficient way."

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ARE CHICKEN AND FISH SAFE?

Many people think that they can switch to chicken and fish, and avoid the problems produced by beef and pork. But such individuals are still on a meat diet. And there are problems with the meat.

Substituting chicken or fish for red meat will not helpyou avoid any of the health risks associated with

diseases of mammals. It will not save you from heart disease, strokes, diabetes, cancer, high blood pressure, or osteoporosis. The same threats exist as if you eat red meat. This is due to the fact that chicken and fish are high in fat (especially saturated fat), high in choles-terol, too high in protein, high in pesticide residue, and devoid of fiber and complex carbohydrates (McDougall, McDougall Program for a Healthy Heart, 1996, p. 49).

Many people think that chicken and fish are low-cholesterol foods, or at least considerably lower than beef. But a 3.5-oz. serving of beef has 8.5 mg. of cho-lesterol while the same-size serving of chicken (white meat, skinned) also has 8.5 mg. of cholesterol (ibid.). The same-size servings of pork, trout, and turkey— will clog your arteries with 90, 73, and 82 mg. of cho-lesterol, respectively. There are no low-cholesterol flesh foods, and there are no plant foods with any choles-

terol (ibid.).

Here is more about chickens:

More than 90% of the chickens in America are raised on factory farms (Jim Mason, "Fowling the Wa-ters," E: The Environmental Magazine, September/ October 1995). A significant part of their diet is their own fecal matter. So it should not be surprising that a recent Agriculture Department study revealed that more than 99% of broiler carcasses had detectable levels of

E. coli ("Safe Food? Not Yet," New York Times, Janu- ary 30, 1997).

In addition, about 30% of chicken consumed in the U.S. is contaminated with salmonella ("Playing Chicken: The Human Cost of Inadequate Regulation of the Poultry Industry," Center for Science in the Public Interest, March 1996) and 70%-90% with another

deadly pathogen, campylobacter ("Health Concerns Mounting over Bacteria in Chickens," New York Times, October 20, 1997).

Although not well-known, here is what campylobacter brings with it: cramps, abdominal pain, bloody diar-rhea, fever, and 200-800 deaths per year in America alone (*ibid.*). It also induces about 2,000 cases a year of an unusual paralytic disease, Guillain-Barré syndrome, whose victims are usually required to stay for weeks in the intensive-care unit, hooked up to a respirator (*ibid.*). The bacterium has become increasingly resistant to an-tibiotics. The reason is that the same drugs have been used to fight disease in chickens.

According to the Government Accountability Project (an independent organization), up to 25% of the chick-ens on the inspection line are covered with feces, bile, and feed. Chickens are often soaked in chlorine baths to remove slime and odor (Government Accountabil- ity Project, "Fighting Filth on the Kill Floor: A Matter of Life or Death for America's Families," November 9, 1995).

In order to safeguard your health, chicken inspectors examine about 12,000 chickens a day, each for about 2 seconds (Food and Agricultural Issues, Gen-eral Accounting Office, March 16, 1993). As a result, contaminated chickens kill at least 1,000 Americans a year. It is estimated that they sicken as many as 80 million more ("Something Smells Fowl," Time, Octo- ber 17, 1994).

More about fish:

Fish are generally not inspected; it is assumed that they are clean, regardless of the waters they come from. But an in-depth 1992 *Consumer Reports* study, on the safety of the fish Americans eat, found **nearly half**

the tested fish were contaminated by bacteria from human or animal feces. It was in the water the fish were caught in ("Is Our Fish Fit to Eat?" Consumer Re- ports Special Study, February 1992). A lot of people now live along our rivers, by our lakes, and along our coasts.

According to a government report, there are 35,000 cases of food poisoning annually in the U.S. from contaminated seafood (CDC Report, quoted in "What's Wrong with Fish?" Vegetarian Times, August 1995).

Fish begin to spoil when there are 1-10 million colo-nies of bacteria growing, per gram. Sampling fish from markets in the New York, Chicago, and Santa Cruz/San Jose areas, *Consumer Reports* found almost 40% of the fish tested in the beginning to spoil range, and an additional 25% with bacterial counts that "exceeded the upper limits of our test method." That meant they had more than 27 million colonies of bacteria, per gram (*Consumer Reports Special Study, February 1992*).

Fish that reach your dinner table have often been dead for two weeks or more, and the bacteria living on them are not disturbed by refrigeration units. Thawed fish are often labeled "fresh" (ibid.).

Health-minded people eat fish because they are considered cleaner and contain *omega-3* fatty acid. But that valuable nutrient can just as easily be obtained by eating soybeans, pumpkin and flax seeds, dark green vegetables, and wheat germ.

But fish have high cholesterol and a wide assort-ment of mercury; lead; pesticides; and the deadly chemical compound, PCBs—something the above veg- etables lack.

Municipal wastes and agricultural chemicals are continually flushed into local waters, and carried into rivers and to the ocean. They are absorbed in the tis-

sues of fish and shellfish.

The Consumer Reports study found PCBs in 43% of salmon and 25% of swordfish (ibid.). Yet both are often caught far out in the ocean. Catfish had significant levels of DDT, clams had high levels of lead, and 90% of swordfish contained mercury (New England Journal of Medicine, September 12, 1996).

Women who ate fish from Lake Michigan, containing PCBs, gave birth to smaller children with significant developmental problems.

MAD COW DISEASE IN PIGS

It has been increasingly suspected that many Alzheimer's cases are actually CJD (the human form of mad cow disease). In this article, Joel Bleifuss reports that pigs are believed to be a significant cause of mad cow disease. You are going to read about breakthrough research into a serious aspect of the BSE problem in America. A link between BSE and eating clams and oysters is also shown.

This article first appeared in These Times, a Chi-cago-based paper, April 26, 1997.

"Porcine" means relating to pigs, and comes from the Latin: "porcus" for pig. Our English word, "pork," is derived from it.

"TSE" stands for transmissible spongiform encepha-lopathy. This came into usage in the 1990s, and means BSE or CJD which can be passed from one animal/per-son to another. *Here is the article:*

"Some pigs in the United States may be infected with a porcine form of mad cow disease, according to an alarming study by U.S. Department of Agriculture (USDA) scientists that has recently come to light.

"This previously unrecognized form of the disease in swine may be infecting humans, according to epide-miological studies that link pork consumption with mad cow's human equivalent, Creutzfeldt-Jakob disease.

"In late 1978, Dr. Masuo Doi, a veterinarian with the Food Safety and Quality Service, observed signs of a mysterious central nervous system (CNS) disorder in some young hogs that had arrived at the Tobin Packing Plant in Albany, N.Y., from several Midwestern states.

"For the next 15 months. Doi studied 106 of the afflicted pigs. He described their symptoms this way: 'Excitable or nervous temperament to external stimuli such as touch to the skin. Handling and menacing ap-proach to the animals is a common characteristic sign among those affected with the disease.' These symptoms, Doi now notes, are strikingly similar to those of British cattle infected with mad cow disease, which is scientifi-callyknown as bovine spongiform encephalopathy (BSE). "Doi sent the brain material from these pigs to Karl Langheinrich, the head pathologist at the USDA's East- ern Laboratory in Athens, Ga. In a November 1979 report, Langheinrich noted that one pig's brain exhibited what the veterinary reference work, Pathology of Domes-tic Animals, defined as 'the classical hallmarks of viral infection of the central nervous system.' Langheinrich went on to report that the damage in the pig's brain was similar to the damage observed in the brains of sheep afflicted with scrapie and of mink afflicted with transmissible minkencephalopathy, the two other vari- ants of transmissible spongiform encephalopathy (TSE)

known at the time.

"In March of this year, Dr. William Hadlow, a retired veterinary pathologist who is one of the world's leading TSE researchers, examined the microscope slides of pig brain from Doi and Langheinrich's 1979 investigation.

The pig 'could have suffered from a scrapie-like disease,' he reports, but adds that such a conclusion cannot be 'justified by the limited microscopic findings, however suggestive of a TSE they may be.'

"The Government Accountability Project (GAP), a Washington-based organization that supports public-sector whistle-blowers, has been working with Doi to alert the public that a porcine form of mad cow disease may be circulating in the American pig population. In a March 27 letter to Secretary of Agriculture Dan Glickman, GAP points out that if we assume a similar incidence of central nervous system disorders in swine being slaughtered nationwide as that found among swine at the Tobin Packing Plant, 'it is reasonable to question whether, since at least 1979, the USDA has been allowing 99.5 percent of animals with encephalitis, meningitis, and other CNS disorders into the human food supply.'

"And what happens once those thousands of dis-eased pigs are eaten by the American public? Two epide-miological studies found pork to be a dietary risk factor in Creutzfeldt-Jakob disease (CJD). A 1973 study, pub-lished in the American Journal of Epidemiology, dis-covered that 14 of 38 CJD patients (36 percent) ate brains. Further, of those who ate brains, most (10 of the 14) preferred hog brains.

"Another study, published in the *American Journal of Epidemiology*, in 1989, looked at how frequently 26 CJD patients ate 45 separate food items. **Nine of these foods were found to be statistically linked to increased risk of CJD**. Of those nine, six came from **pigs**—roast pork, ham, hot dogs, pork chops, smoked pork and scrapie. (The three that were not pig-derived were roast **lamb, raw oysters/clams and liver**.)"

The authors of the study concluded: "The present study indicated that consumption of pork as well as its

processed products (*e.g.* ham, scrapie) may be considered as risk factors in the development of Creutzfeldt- Jakob disease. While scrapie has not been reported in pigs, a subclinical form of the disease or a pig reservoir for the scrapie might conceivably exist.

"The number of Americans who develop CJD in a given year is in dispute. The Centers for Disease Control (CDC) claims that the human form of mad cow disease occurs at a rate of one in a million. Further, ignoring evidence of a new variant of CJD found in Britain, the CDC maintains that people who eat an infected animal cannot contract the disease. In January, CDC Assistant Director for Public Health Lawrence Schonberger told a Congressional hearing, 'The bottom line from our perspective is that it's a theoretical risk... but it is not as yet a real risk.'

"But does the CDC really know how many Americans contract CJD? Evidence indicates that CJD may often be misdiagnosed, and thus go unreported. A 1989 study at the University of Pittsburgh autopsied the brains of 54 patients who had been diagnosed with Alzheimer's and discovered that three of the patients (5.5 percent of the sample) actually had CJD. A 1989 study at Yale University reported similar findings.

"Postmortem examination of 46 patients who had been diagnosed with Alzheimer's revealed that six (13 percent of the sample) actually had CJD. The New York-based Consumers Union, which publishes *Consumer Reports*, argued in a paper presented to the USDA, 'Since there are over 4 million cases of Alzheimer's disease currently in the United States, if even a small per-centage of them turned out to be CJD, there could be a hidden CJD epidemic.'

"Which brings us to the issue of what the Food and Drug Administration(FDA) is doing to address this food-

borne threat to public health. In the past several months, in response to questions about Doi's 1979 pig research, USDA officials have put out a good deal of misinformation to public-interest groups, the media and even the National Association of Federal Veterinarians. On repeated occasions, officials have said that the slides of the pig brains from the 1979 study were unavailable because they had been sent to scientists in England who were studying mad cow disease. But as it turns out, the USDA never sent any slides to England.

"'Agency officials repeatedly misrepresented sci-entists' investigations and conclusions to consumer groups and government employees and neglected to keep other agencies also working on TSE issues in-formed,' says Felicia Nestor of GAP. 'The USDA had to be pushed to investigate scientific evidence which only they had.'

"The USDA's lackluster response to this public health threat comes as no surprise. For years, the agency has done its best to ignore evidence that a distinct American strain of mad cow disease may already afflict the U.S. cattle population. Veterinary researchers in Mission, Texas in 1979 and Ames, Iowa, in 1992 found that cattle injected with brain matter from scrapie- infected American sheep developed BSE. However the brains of these infected cattle did not exhibit the same spongy holes found in the brains of their BSE-plagued British cousins; yet it is still a spongiform disease. Fur- thermore, cows afflicted with this American strain of scrapie-induced BSE do not go mad; they simply col-lapse and die.

"The distinction is important because the American strain of the disease leads to symptoms that resemble what happens to the 100,000 American cattle that succumb to 'downer cow syndrome' every year.

"Veterinary researchers fear that the widespread practice of feeding downer cows (in the form of rendered protein feed supplements) to other cattle, sheep and hogs could already be fueling a TSE epidemic in the United States like the one that plagued Britain. In fact, in 1979, before BSE was discovered in Britain, Doi pointed out in his study of deranged pigs that many animals have been found to be 'downers' at first observation.

"On January 3 [1997], the FDA finally drafted a rule that would ban the fortifying of animal feeds with 'any Mammalian tissue.' USDA researchers, critical of the government's foot dragging, have been calling for a ban for seven years. But undercutting this important step, the FDA has played a taxonomical shell game and arbi- trarily removed pigs from the class 'mammalia.' [Accord- ing to the U.S. Government, pigs are not mammals!]

"Consequently, if the FDA's proposed rule is adopted,animals being fattened for slaughter will stop eating cow renderings and instead eat only pig re-mains. Since mad cow disease in Britain was spread by feeding mad cows to healthy cows, the FDA's pigs-are-not-mammals proposal gives any porcine form of mad cow disease a point of entry into the human food chain. "On April 28, Consumers Union filed comments with

the FDA on the agency's proposed regulations. The group advocates a complete ban on the use of all mammalian protein in all feed intended for feed animals, as is now the case in England. [That means Britain now has a stricter feed ban than the U.S. does!] 'The draft rule,' says Consumers Union, 'is not adequate to protect public health, because it would continue to leave the door open for a porcine TSE to contaminate pork and other meat.'

"It would be nice if the USDA were as concerned about protecting publichealth as it is about the finan-

cial health of the \$30 billion-a-year pork industry and the \$60 billion-a-year beef industry. Ditto for the *Wall Street Journal*, where editors have put on hold a story by a staff reporter on mad pig disease and the possible link between pork consumption and CJD.

"ABC's World News Tonight has also sat on the information for a couple of weeks. On May 12, the net-work did air a story that examined the fact that CJD was being misdiagnosed as Alzheimer's. But the network failed to note that CJD is the human form of mad cow disease. The network also neglected to mention the pos-sible connection to pork or the fact that the CJD patient featured in the story, Marie Ferris, had been employed at a packing plant where she handled slaughtered pigs."— These Times, a Chicago-based paper, April 26, 1997.

RUINING THE PLANET

The intense grazing and feedlot methods are de-stroying the pasture lands, the streams, the rivers, and the atmosphere. Small farmers are being ruined by what is happening. Here is the story.

Dr. Michael W. Fox summarizes the effects of food animal production in these words: "An estimated 85% of all U.S. agricultural land is used in the production of animal foods, which in turn is linked with deforestation, destruction of wildlife species, extinction of species, loss of soil productivity through mineral depletion and erosion, water pollution and depletion, over-grazing, and desertification" (M.W. Fox, Agricide: The Hidden Crisis that Affects Us All, 1986, pp. 50-51).

Here are several facts to consider:

The modern method of raising food animals harms our air quality in several ways. A third of the annual

increase of carbon dioxide in the atmosphere comes from the burning of the earth's biomass (vegetation). Much of the clearing and burning of forests is done solely to make room for cattle. Tree leaves extract pol- lutants from the air, but they are being destroyed.

It takes roughly 16 pounds of grain to produce one pound of beef. An immense amount of energy is needed to run the tractors, fuel the spray planes, power the combines to harvest them—all for the raising of beef, pork, or chicken. Eighty percent of American grain production is used to feed meat animals. It now takes a gallon of gasoline to produce a pound of grain-fed beef in the U.S. To sustain the yearly beef requirements of an average family of four requires over 260 gallons of fossil fuel. When that fuel is burned, it releases 2.5 tons of additional carbon dioxide into the atmosphere, equivalent to the amount of CO₂ the average car emits in six months of normal operation. All that for one pound of grainfed beef (Jeremy Rifkin, Beyond Beef, pp. 224-225).

Transporting cattle to slaughter and then packaging and freezing the meats are energy-intensive pro- cures. But fruits and vegetables do not need to be frozen and packaged before reaching your table. Energy is also needed for temperature control of the animals in the feedlots, to transport them, to feed them, carry off their wastes, and to manufacture the antibiotics continually pumped into those animals.

According to an Ohio State University study, **even the least efficient plant food is nearly ten times as efficient as the most efficient animal food** ("Energy Costs of Livestock Production," American Society of Agricultural Engineers, June 1975).

Energy consumption always involves unseen pol-lution. It also increases our dependence on foreign

oil and nuclear power plants.

Most of the agrochemical poisons sprayed into the air and falling on the ground are dedicated to the pro-duction of meat. We are poisoning the land in order to raise meat animals.

The amount of waste produced on the feedlots is astounding! None of it is spread over the land, as would occur if the animal were permitted to graze. The average cow produces 25 pounds of waste per day; and 5,000 head of cattle in the feedlot produce enough waste to keep workers busy day and night, at a cost of \$75,000 a year, moving it out onto the land. So, instead, it is just dumped in mountains of waste (or secretly dumped in nearby rivers; more on that later in this chapter). The manure mountain is frequently sprayed to keep down flies (Jim Mason and Peter Singer, Animal Factories, 1990, p. 116).

The fertilizers sprayed on the fields to produce the hay contain only nitrogen, phosphorus, and potassium—and none of the trace minerals, such as zinc or selenium normally present in healthy soil. This rapidly depletes the soil for years to come (*John Robbins, Diet for a New America,* 1987, p. 376).

The 1.3 billion head of cattle in the world emit an estimated 150 trillion quarts of methane gas, which is the second most significant contributor (after carbon dioxide) to the greenhouse effect. Every cow emits up to 400 quarts of methane gas daily. Chopping down trees, to make room for more cows, also contributes to methane production. Felled trees which are not burned are eaten by termites, which produce more methane gas. Scientists estimate that the methane content of the at-mosphere has doubled in the past 200 years (Lynn Jacobs, Waste of the West, 1991, pp. 146, 226).

Then there is the stripping of the land and deser-

tification that result from livestock overgrazing. They especially destroy land which does not receive a lot of rainwater. This results in increased dust in the air. Bared soil is lost to the wind. People are harmed by breathing that dust; and it also traps solar radiation, bringing about climate change. Dust storms have been linked to livestock grazing in Africa, China, Australia, the Middle East, and the western United States (Jacobs, p. 146).

We might also mention the cost of all the ambulances rushing around cities to pick up heart attack and stroke victims.

Cattle hooves widen streams, and cattle manure pollutes it. The widening streams increase in temperature by 5° to 10° F., killing certain fish and multiplying harmful organisms. Algae proliferate, water evaporates more easily, and less dissolved oxygen is available for fish who need it to survive (*Jacobs*, p. 85).

Livestock waste is often dumped into streams as the most efficient way to dispose of it. Feedlot wastes can be several hundred times more concentrated than raw domestic sewage (Robbins, p. 373). Nitrates, am-monia, and bacteria from that waste frequently wind up polluting rivers, streams, and well water. The sheer size of this pollution is astounding! An average feedlot with 10,000 head produces as much as half a million pounds of cow manure every day (Rifkin, Beyond Beef, 1992, p. 221). The largest feedlots, with 100,000 head, have a waste problem equal to the largest cities in America (Robbins, p. 372). Livestock waste exceeds human waste in tonnage nationwide by a factor of one hundred and thirty! ("Animal Waste Pollution in America: An Emerg- ing National Problem," report of the Minority Staff of the U.S. Senate Committee on Agriculture, Nutrition, and Forestry, December 1997).

Big business is taking over U.S. farms. Family farming is almost gone in America. In 1983, there were about 1,250,000 full-time commercial family farms in our nation. Today there are only about 350,000. At this rate, the family farm will be virtually extinct within a few more years. The men with the big money will own it all.

And they are gradually ruining American rivers. Rodney Barker has written a book, *And the Waters Turned to Blood*, in which he tells about the horror that is occurring in an increasing number of U.S. rivers from feedlot runoff.

Dr. JoAnn Burkholder, a University of North Carolina research scientist and professor in aquatic botany became an expert on a previously unknown single-cell organism, named *Pfiesteria piscicida*. **First by the thou- sands and then by the millions, fish were dying in North Carolina waters. People bathing or swimming in those rivers were becoming sick and finding it ex- tremely difficult to recover.** Burkholder proved that the cause was *Pfiesteria*, which emits a deadly toxin.

Many of the fish have open sores. Fishermen and vacationers, when their skin came in contact with river water, developed body sores, acute loss of memory, strange sieges of temper, and nerve seizures.

The cause is primarily waste from hog feedlots. North Carolina ranks second only to Iowa in the number of pig farms. A book could be written about what is happening in North Carolina. Rodney Barker's *And the Waters Turned to Blood* is that book. State officials repeatedly ignored the problem, so as not to injure the tourist trade or the hog farmers association. Burkholder was not vindicated until the number of dead fish and human sicknesses had become very large.

Pfiesteria has since been found in waters from Delaware Bay to the Gulf of Mexico (Barker, p. 322).

In Maryland, the cause was chicken manure from the chicken farms ("Another Waterway is Closed in Mary-land," New York Times, September 15, 1997). It is be-coming dangerous to use animal manure to fertilize your garden, if it can get into your well water (*Pfiesteria* must have nutrient-rich water in order to breed).

Another major problem in America is the grow- ing need for freshwater. Each decade, this problem will get worse.

Livestock production accounts for over half the water consumed in the Northwest. Half of Arizona's water is used for livestock. Stockmen use over half the water in California. Other state statistics could be cited. About 70% of the water used in 11 western states is used to raise animals for food (Jacobs, p. 215).

The water required to produce just ten pounds of steak equals the water consumption of the average household for a year (Lappé, Diet for a Small Planet, Rev. ed., p. 76).

The central states—from Texas to South Dakota—cantap

into the underground Ogallala Aquifer. But nearly half the grainfed cattle in America are raised by farm- ers dependent on the Ogallala to irrigate their crops. Since 1960, about three cubic miles of water has been drained annually from this reserve. Wells are beginning to run dry in parts of Texas, Missouri, Colorado, and Nebraska. Farmsare being deserted and the soil is blow- ing in the wind. Based on the current usage rate, the Ogallala will be nearly empty by 2050 (Rifkin, p. 219). Did you know that U.S. tax dollars pay for more than half the costs of irrigation projects in the U.S.? (Lappé p. 85). It averages \$54 an acre, and the benefits keep going to a few, very large agribusinesses (George Wuerthner, "Public Lands Grazing: The Real Costs," Earth First, August 1, 1989). Yet, as we have

already

discovered, raising animals injures the land, air, and water more than other kind of rural business, other than surface strip mining (*Lappé*, p. 85).

Then there is the problem of ranching on public lands in the West. Public-land ranching results in extraordinary destruction of native vegetation and wildlife; it also causes widespread flooding, soil erosion, and water pollution. It costs the American treasury \$1 billion or more annually, yet produces only 3% of American beef! (*Jacobs, p. 566*). "Ranching has wasted, and is wasting, the western United States more than any other human endeavor" (*Lappé, p. 3*).

The government has admitted that **over 90% of pub-lic lands in the West are in bad condition** (*Arizona Republic, April 1, 1991*). By damaging streams, grass-lands, riparian (river bank) zones, and forests, **livestock winds up devastating plant and animal life in the West**. Pronghorn deer are disappearing, as well as the fish and birds (*Jacobs, p. 117*). A study of an Oregon wildlife refuge found bird counts five to seven times higher in its ungrazed areas, compared to similar areas grazed annu- ally by cattle. **Trout populations are 350% higher in ungrazed portions of Oregon rivers** (*ibid.*).

Not even our National Wildlife Refuges are being protected. Of 109 such refuges in Montana, Wyoming, Colorado, Utah, Kansas, Nebraska, and the Dakotas, 103 are grazed (*ibid.*).

Livestock spread anthrax, brucellosis, encephali-tis, leptospirosis,pneumonia,bluetongue,pinkeye,sca-bies,and rabies to wildlife and sometimes to humans.

We will conclude this chapter with a quick look at the fish in our oceans. **All the world's major fishing grounds have been stressed to their limits.** Hi-tech fish- ing vessels, deploying fishing nets wide enough to haul in a dozen 747 jumbo jets, have depleted our oceans and

pushed many species to the brink of extinction.

Two of the world's most productive fishing areas, Canada's Grand Banks and New England's Georges Bank, are considered commercially extinct. Add to our suicidal overfishing the fact that one-third of the world's catch of fish is turned into fish meal and fed to livestock ("The World's Imperiled Fish," Scientific American, No-vember 1995).

THE PIONEER MAD COW RESEARCHERS

Here is the story of the men and women who unraveled the mystery of prions and mad cow dis-ease. We owe a lot to them. They did most of their work at a time when their efforts were not always appreciated.

Although the earliest researchers could not figure out what caused Kuru, Dr. Carleton Gajdusek, a virologist employed by the National Institutes of Health (NIH), carefully examined the Fore people in the early 1950s. Back in Maryland at the NIH, the similarity between Kuru and CJD, an extremely rare disease, was recognized.

Dr. William Hadlow, a scrapie expert, later found that Kuru brains were like those in scrapie sheep. He wrote a letter to *Lancet*, the British medical journal, suggesting that a researcher check to see if the condition was transmissible. In order to determine that, Gajdusek started injecting chimps and monkeys with the ground brains of Fore tribeswomen. By 1965, they had shown that Kuru was transmissible. After extending the experiments, Gajdusek demonstrated that scrapie, Kuru and CJD could all spread and kill in the same manner. He received a Nobel Prize for his work. But the pathogens

causing the disease were still unknown.

CJD (the human form of mad cow disease) is a hor-rible sickness that usually involves deterioration into blindness, dementia, and loss of nerve function. Both Kuru and CJD were always fatal, and resulted in corpses whose brains were riddled with holes; hence the name, "spongiform" (spongeshaped).

To this day, few scientists want to go near CJD. They prefer not to do research on it, and pathologists avoid undertaking postmortem examinations of patients suspected of death by CJD. The problem is that none of the common solvents and heat sterilization techniques used in bacterial research will decontaminate the dis- ease-causing agentor, for that matter, anything it touches! In addition, the infective agent is extremely infectious.

In those instances in which they do examine remains, researchers wear masks, goggles, gloves, boots, caps, operating gowns, and plastic aprons; all of these can be destroyed afterward!

But none of this was known by scientists in ear-lier decades in the U.S. and Britain when they tried to figure out what Kuru was. When they failed, the test samples were dumped down the sink drain or tossed out. Surely, they thought, there is no infective agent present.

But something was there. And now, in its most virulent form, it was outside the U.S. and British laboratories, infecting passing wildlife.

After losing a patient to CJD, Stanley B. Prusiner, a biochemist at the University of California, worked on the problem and gradually developed a theory that the infective agent in all these spongiform encephalopa- thies was not a virus but a kind of abnormal protein, to which he gave the name, "prion" (Richard Rhodes, Deadly

Feasts, pp. 121-123).

His theory was based on the work of two earlier researchers, Tikvah Alper and Carleton Gajdusek, who speculated that the infectious agent in scrapie might be something which lacked nucleic acid (Richard Rhodes, pp. 160-162). That seemed impossible, yet ex- tracts of scrapie-infected brains retained their ability to transmit the disease—even after being radiated to a de- gree which would destroy nucleic acid (Prusiner, "The Prion Diseases," Scientific American, January 1995,

p. 49). (Nucleic acid is the substance containing DNA, and is present in all viruses.)

Prusiner eventually identified 15 of the amino acids at one end of a prion. Checking deeper, he found that "normal proteins" were in a single gene in both man and all animals that he tested (*ibid.*). The normal ones were no problem. But, due to some strange circumstance, they could become perverted into something terrible— the spongiform diseases.

Eventually, **Prusiner figured out that the wrong type** of protein is able to make contact with normal proteins—and contaminate them! In this way, the disease spreads throughout the brain, as well as into nerve, blood, and bone cells throughout the body.

A normal protein is folded in a certain manner. When the abnormal protein makes contact with it, the normal protein unfolds—and then refolds in the same damaged pattern that abnormal proteins (prions) are in! That is how the prion propagates itself (*Prusiner*, p. 52).

Sounds strange? It surely is. An amazing new plague for our time in history. Some say it will eventu- ally be worse than AIDS; for many people eat meat, and the meat they are eating was fattened on diseased, dead animals—just like those natives in New Guinea who ate

their dead relatives. People should not eat people, and cows should not eat cows. The result, for all concerned, is mad cow disease.

Prusiner had identified an entirely new type of disease agent. But, **next**, **we go to Richard Marsh**, a **re-searcher at the University of Wisconsin**.

The British Government maintained that cattle were a "dead-end host" of spongiform disease. By that, they meant that cows were getting spongiform spontane- ously, and could not transmit it to anyone who ate them. Marsh did not believe that theory.

Marsh suspected, rightly enough, that the British Government was saying this to keep the public from panic—and keep them buying British beef. The theory also made it convenient to keep feeding animals to the British cattle.

To keep the money rolling in, the cycle of ever-increasing doom for an entire nation was permitted to con-tinue.

Are we doing something similar right here in America?

Marsh (who died in 1997) took material out of the brains of infected mink and placed it into the brains of two Holstein steer calves. A year and a half later, the steers suddenly fell over dead. Then Marsh transferred some of their brains back into mink which then died of TME (transmissible mink encephalopathy, the mink form of the disease).

Based on Marsh's research, it was obvious that mad cow disease could, indeed, "jump the species barrier"—and infect human beings!

Further research eventually disclosed that the U.S. form of BSE is called "downer cow syndrome." In- stead of wobbling before dying (as British cows do), U.S.

cows with the disease just fall over dead.

It so happens that slaughterhouses, both in Britain and the U.S., do not accept cattle unless they arrive alive and are able to walk. So cattle which die of BSE—in both the U.S. and Britain—are sold to rendering plants, which cook them at 280° F., and turn them into feed pellets, to be fed to feeder cattle at the feed- lots, which are then shipped to the slaughterhouses, prior to being placed on your table to eat.

There you have the whole procedure.

Add to this the fact that mad cow disease can linger in humans for years before it manifests itself. AIDS kills within eight to 15 years. But **CJD** can go as long as 35 to 42 years, or more, before killing its victim. How do we know this to be true? because some Kuru victims died as much as 42 or more years—after eating a dead relative. (Most lived 10 to 35 years after eating a rela-tive.)

William Gordon, a British researcher, decided to duplicate Marsh's research. He injected scrapie into many a variety of animals (including goats and mice) and caused their death from the same disease (*Rhodes, p. 121*).

But certain significant facts should be noted: Gordon discovered (1) that the amount of infected material, fed or injected in the animal, determined how fast it developed the full-blown disease. (2) Gordon could infect animals, not only with blood or brain, but also with muscletissue.

Do you know what it is that you eat, when you eat meat? You are eating muscle tissue. That is what is sold to you at the meat counter of your corner grocery store or served to you in a wrapper at your nearby fast-food restaurant.

So we have here a mammoth problem: For years, the British and U.S. Governments sided with their meat industries, in declaring that mad cow disease could not "jump the species barrier"; yet Marsh and Gordon had shown that it could. That posed the question as to whether it could also jump the barrier from the animals— and infect our brains.

Another question was whether mad cow disease was in America. The official word has been that it definitely is not.

Yet thousands of "downer cows," with spongiform brains, drop dead in the U.S. every year. (They are immediately ground up and turned into cattle, sheep, hog, and chicken feed.) It is also known that a variety of wild animals in our continent also have a type of the disease. Yet the official statement remains that there is no need to worry; mad cow is only in Europe.

One researcher asked Marsh how the mink used in his experiment contracted mad cow disease. He replied that he could not say for certain, but that those mink had been fed the carcases of downer cows (H.F. Lyman, Mad Cowboy, p. 88).

A major breakthrough occurred in October 1996, when Dr. John Collinge reported in the British jour- nal, Nature, on a new biochemical test for prions. Us- ing it, he found that the biological "fingerprints" of prions in patients were identical to those in laboratory animals with BSE. —Finally, this is definite proof that both humans and animals had mad cow disease! (John Collinge, et al., "Molecular Analysis of Prion Strain Variation and the Etiology [cause] of 'New Variant' CJD," Nature October 24, 1996).

A year later, he reported on **CJD samples taken from** people who had died, which, when placed in testani-

mals, produced BSE! BSE transferred from a mad cow to test animals produced the same BSE. This was the final proof (John Collinge, et al., "Spongiform Encepha-lopathies: A Common Agent for BSE and vCJD," Na-ture, October 2, 1997).

The same week that Collinge's conclusive second *Nature* article was published, Prusiner's research brought him a Nobel Prize in medicine.

It was estimated that, by the fall of 1996, at least 750,000 cattle infected with mad cow disease had entered the human food chain since the start of the epidemic. You may wish to read that sentence again (R.M. Anderson, et al., "Transmission Dynamics and Epide-miology of BSE in British Cattle," Nature, August 29, 1996).

Many people will die because their government refused to tell them it was no longer safe to eat meat. Politicians frequently retire from "public service" with a sizeable amount of money in the bank. There is a rea-son.

The incubation period of the spongiform diseases varies in direct relation to a species' natural life ex- pectancy. Mice will show visible symptoms of the dis- ease within a few months after being infected. It takes cats several years for symptoms to reveal themselves. The incubation period in humans of CJD can vary from 4 to 10 years or more.

Therefore the cases of CJD that have arisen in the first half of the 1990s could well have derived from the eating of infected beef in the early or mid-1980s, before BSE was even diagnosed.

But, obviously, the quicker a person stops eating meat now, the more likely he will avoid contracting the disease. A beef steak is not worth dying for. At the present time, there is no treatment for CJD. If you have it, you will die from it. No drugs, surgery, or natural remedies can remove it from your body. There is no test for CJD to determine if you have it, before symptoms reveal themselves, other than a biopsy of brain material after you are dead.

To date, the U.S. Government refuses to test food animals, to see if they have the American form of BSE. This could easily be done by testing a large sampling of downer cows. They died from what appears to be BSE, but no one will test them. The government and meat industry do not want them tested and researchers are glad to avoid the task; for it would contaminate their laboratories.

Richard Lacey predicts that 200,000 (two hundred thousand!) Britishers will die each year, by the year 2015 (*Rhodes, p. 222*).

DANGER: SURGERY AND MEDICAL EXAMINATIONS

The ramifications of mad cow disease are remarkable. We wish at least some of it was good news. Within a few years (if not now), it may be unsafe to undergo surgery (medical and dental) and even routine endoscopic medical examinations.

The problem is prion-contamination of medical instruments which cannot be sterilized; yet medical experts declare the cost of discarding them after each use in prohibitive.

According to the November 13, 2000, issue of *The Times* of London, England, "Half the surgical instruments used for tonsil operations could be contaminated by variant CJD, according to an expert." The problem is that autoclaving equipment, used in hospi-

tals and dental clinics everywhere to sterilize instruments, does not kill prions—the cause of animal and human variants of mad cow disease. This means that, if a patient that the instruments were used on had CJD, prions can be transferred to subsequent patients.

The estimate for the surgical instruments was based on a conservative estimate of 10,000 people in Britain which are incubating the disease. John Collinge of Im- perial College School of Medicine in London said that the ear, nose, and throat surgeons at St. Mary's Hospi- tal, part of the medical school, "calculated that half the tonsillectomy [instrument] sets in the UK are contami- nated. This is, potentially, a serious problem. It's a major problem to which the Department of Health has given a lot of thought and not much action." The British Depart- ment of Health is consulting with surgeons on whether to spend the extra money to switch to disposable, singleuse instruments—especially in tonsil, appendix, eye, and brain surgeries (Times of London, November 13, 2000).

The following excerpt was written January 14, 1999 by John von Radowitz, medical correspondent. Parallel reports were printed in the British Press Association, in the *Guardian*, for January 15, 1999, and by Steve Connor, Science editor of the *Independent*, January 14, 1999. See the January 1999 issue of the British medical journal, *The Lancet*, for detailed research data. Here are these findings:

"Scientists are to test thousands of people for CJD, the human form of mad cow disease, after discovering evidence in people's tonsils, it emerged to-day. Previously, the disease could only be confirmed after victims had died. The find means it may be possible in the next three years to establish if a CJD time bomb

is ticking within Britain's population. But **it also raises renewed concerns about the risk of infection from surgical equipment in hospitals**, the expert behind the discovery said.

"Scientists plan to screen thousands of people us- ing a new test. A significant positive result would pro- vide early warning of a major epidemic to come and al- low time for action aimed at averting the disaster. Pro- fessor John Collinge, from St. Mary's Hospital, London, said today: 'If we were to screen several thousand ton- sils and found that several were postive that would be a real cause for concern.'

"The concern about infection raised by the new find- ings centers on rogue prion proteins that spread the disease and cannot be cleaned from surgical instru- ments no matter how thoroughly they are sterilized. In the future, therefore, it may be necessary to introduce disposable instruments for certain procedures. A spe- cial committee of experts advising the [British] Govern- ment is already looking at this issue.

"Animal studies have shown that spongiform encephalopathy diseases, which include different forms of CJD, BSE in cattle and the sheep infection scrapie, re- side in the lymph system before attacking the brain. Tonsils are linked to the lymph system. The new re- search by Professor Collinge's team showed this also appeared to be the way new variant CJD behaved in humans. But only the new variant form of CJD, which is effectively mad cow disease transferred to humans through infected beef, was detected in tonsils. The 'classical' form of CJD, which appears for no known rea- son in one person in a million, was not seen in the tis- sue samples.

"Scientists now know they can diagnose new variant CJD by analyzing a surgically removed piece of tonsil

tissue. Until now, scientists have had to wait until the death of a patient to find out definitively if there is CJD infection. [But, in snipping a piece of tonsil to test for the disease, they might give it to the person!]

"No one knows at present what the fallout might be from people eating BSE-infected beef that was routinely used in burgers and other meat products before a [Brit- ish] ban on suspect offals was introduced in 1989. **New variant CJD has a long incubation period which may run into decades**, in which case the trickle of cases seen so far may be just the start of a flood.

"Professor Collinge admitted he was concerned about the possibility of new variant CJD being trans- mitted via infected surgical instruments. This had al- ways been a theoretical risk. But the conclusive evidence of tonsil infection [by prions] meant it was now a real possibility. But throwing away expensive surgical in- struments had to be ruled out because of the cost, said Professor Collinge. He added, 'There is no means of sterilizing surgical instruments adequately for prions."

It should be mentioned here that, in order to cut costs, more and more U.S. hospitals are reusing "disposable" medical catheters and equipment which is infecting people with an increasing number of diseases. This infection is occurring, not only during operations but even routine physical exams (oral, rectal, vaginal, etc.)! For more on this, see such sources as "Risky Recycling," U.S. News & World Report, September 20, 1999. This is a major developing health crisis, brought about by tightening medical costs, and involves both prions and other diseases.

ENDANGERED BLOOD SUPPLY

Douglas P. Starr, in his 1998 book, *Blood: An Epic History of Medicine and Commerce* (Knopf is the publisher), discloses that, first the AIDS crisis and now the prion crisis is destroying the American Red Cross (ARC) blood supplies, both in reputation, number of blood donations, and financial base.

Having failed to live up to its 1988 agreement with the FDA, in 1998, the ARC was required, under court order, to make extremely expensive changes (which increased its costs by \$25-\$35 per unit of blood). The changeover, required by the AIDS crisis, cost the ARC \$287 million.

But then, a new crisis occurred: In 1994, the news broke that a Utah man, Doug McEwen, was slowly dying of CJD—mad cow disease. The worst part was that McEwenhad previously donated over a hundred blood transfusions to the ARC blood supply, part of which had been sent to Canada for distribution. The blood had been intermingled in with many large pools of blood. After months of deliberation, the ARC ordered the destruction of several million dollars worth of blood.

Ultimately, that one case cost the ARC \$130 million.

But who knows how many other AIDS or prion-in-fected people have been donating blood or, in the future, will begin to do so? It is a well-known fact that homo- sexuals make extra money by selling blood, and there is no practical way to test prospective blood donors for prions.

The British medical journal, *Lancet*, for January 9, 1999 reported that, in December 1998, the Canadian Government officially decided that all the blood, con-taminated by McEwen, the Utah man who died of CJD, could be released into the public blood supply! De-

claring that there is "no scientific evidence" that CJD could be transmitted through blood, the Canadian Gov- ernment lifted a 4-year quarantine of blood products made from plasma donated by McEwen "and others with CJD."

In late December, 1998, Health Canada's Bureau of Biologics, under the direction of Keith Bailey, freed Canada's blood agencies to use albumin, immunoglobu-lins, and factor VIII made from plasma from people with CJD, including McEwen. He had donated blood over 100 times, including some after he had begun to show signs of CJD. Canadian blood banks had saved millions of dollars, and hospitals throughout the nation would not experience any blood shortages.

U.S. health authorities only require the withdrawal of plasma from donors with variant CJD. All the rest of the blood they give is used! For more on this, see the *Lancet* article.

CDC ON CJD DEATH RATES

The chart on the next page was prepared by the Centers on Disease Control (CDC), based in Atlanta.

The *bar graph* shows that a majority of people who contract CJD (mad cow disease) incubate the disease for a number of years and do not die until they have passed their late 40s.

The *line graph* indicates death rate per 1 mil- lion people, which peaks at the age of 73.

FOOD SUPPLEMENTS WHICH CONTAIN MEAT

Vitamins, minerals, and herbal supplements are an invaluable aid to health and healing. But you need

to read the label carefully. The following news re-lease, dated September 27, 2000, discusses so-called "herbal" supplements which contain animal prod-ucts.

The following article was released by the Organic Consumers Association, through the Associated Press, February 5, 2001.

"New York: Despite their plant-based image, some herbal supplements contain 'raw animal parts'—including, according to a report, cow brain matter.

"There is no evidence that any herbal product has been contaminated with the agent that causes bovine spongiform encephalopathy (BSE), the mad cow disease that triggers a similar brain-wasting disease in humans who eat tainted beef.

"However, Dr. Scott A. Norton said in an interview, 'I would advise all of my patients not to take supple- ments that contain central nervous system tissue from animals.' [Norton did not realize that CJD can be con-tracted by eating other parts of BSE-infected animals.] The problem is that herbal-supplement labeling is not always clear, Norton writes in a letter in the July 27th issue of the New England Journal of Medicine. Although Norton found one product that listed, as part of its ingredients, 17 cow organs, from lungs to brain mat-ter, other manufacturers are not so forthright. For ex- ample, most consumers would likely not realize that 'hypothalamus' refers to brain tissue, said Norton, a dermatologist and botanist from Chevy Chase. He mentioned one product which contained bull testicles (on the label called "orchis").

" 'The public,' Norton said, 'doesn't fully understand what they're getting into when they buy these products.' The public should at least be aware of what is there,

according to Norton. 'I think a lot of us would feel we've been duped,' he said, 'if we think we're getting a whole- some product and then find out it contains animal parts.' "— Organic Consumers Association, through the Associated Press, February 5, 2001.

Special note: We do well to be very cautious regard- ing the herbal products we use, to make sure no animal products are included in them. Not mentioned in the above article is the danger of taking calcium supplements which may have bonemeal in them.

Here is a second article, with the same date, about Norton's findings. There are those who take food supplements which are not vitamins, miner- als, or herbs, but "glandulars" which consist of vari- ous animal organs.

"Washington (AP): Dr. Scott Norton was browsing through herbal supplements when he spotted **bottles containing not just plants but some unexpected animal parts: brains, testicles, tracheas and glands from cows and other animals.**

"The Maryland physician sounded an alarm: **How can** Americans be sure those supplements, some imported from Europe, are made of tissue free from mad cow disease?

"Norton's complaint has government scientists scrambling to investigate a possible hole in the nation's safety net against mad cow disease and its cousin that destroys human brains . . Just what bulk ingredients containing cow brain or nerve tissue might be slipping from Europe through U.S. ports? . . The FDA inspects less than 1 percent of all imports under its jurisdiction . . FDA officials contend the issue isn't a huge concern. They note the majority of supplements are made from plants,

not animals...

"The [supplement] industry's Council for Responsible Nutrition also calls the worry exaggerated, saying gland-containing supplements account for less than 1 percent of sales. Officials are trying to determine how much is imported and plan to meet soon with FDA."— Organic Consumers Association, Associated Press, Feb-ruary 5, 2001.

OBTAINING ADEQUATE PROTEIN

This chapter deals with the following points: How much protein does a healthy person need? Are there any dangers in eating a high-protein diet? How much protein is assimilated from a meat diet, as opposed to a vegetarian one? Which is the best source of protein?

IMPORTANCE OF PROTEIN—Protein was first identified as a food nutrient in 1838. Protein is needed for muscle function, hormone synthesis, and the production of enzymes. Aside from water, a great proportion of what is in your body is either protein or bone. Growth and repair both require an adequate protein intake.

Protein is built up from amino acids. Of the 20 amino acids in your body, eight are "essential"; that is, they cannot be made within your body. You must get them from the food you eat. The other 12 can be made from those eight.

The issue is not the importance of protein in our diet; all are agreed on that. The question is which is the better protein in our diet: vegetables or meat? *Here are some facts:*

 $\begin{tabular}{ll} \textbf{BETTER ON LOW-PROTEIN DIET} — Earlier in the 20th century, Dr. Russell Chittenden, of Yale University, \\ \end{tabular}$

was a leading athletic trainer. He was one of the first to challenge the nutritional theory, that animal-based foods provided the best strength and energy. Chittenden conducted at least three studies that examined the question of whether meat and high protein were really necessary for optimal performance. One involved a study of well-trained athletes.

At the beginning of the study, all were on a typical meat diet. Chittenden had them switch to a plant- based diet for five months. At the end of the study period when their fitness levels were reanalyzed, the athletes had improved a striking 35%! As Campbell, another researcher, later commented, "only the dietary change could have accounted for these remarkable re-sults."

A recent discussion of Chittenden's research can be found in *T.C. Campbell, Muscling out the meat myth, New Century Nutrition, July 1996.* Dr. Campbell, Pro- fessor of Nutritional Biochemistry at Cornell University, is the director of the massive Cornell-Oxford-China Diet and Health Project.

In their classic study in the 1960s, Mervin Hardinge and his team at Loma Linda University analyzed the complete diets of three groups: meat-eating Americans, lacto- ovo vegetarians (who also eat dairy products and eggs), and vegans. The research team found that the vegans obtained the best mix of essential amino acids, the veg- etarians came in second best, and the meat eaters a poor third (M.G. Hardinge, H. Crooks, F.J. Stare, "Nu- tritional Studies of Vegetarians," Journal of the Ameri- can Dietetic Association, January 1966).

HIGH-PROTEIN DIET ELIMINATES CALCIUM—

An important point concerns the fact that humans do not need a high-protein diet, and it is actually not good for them!

A high-protein diet robs the body of calcium. Healthy young adult men were given carefully controlled diets for nearly four months. During this entire time, they were consuming 1,400 mg. of calcium daily. Their protein in- take was also carefully regulated. Test studies revealed that those who ate 48 g. [grams] of protein per day gained 20 mg. [milligrams] of calcium each day. Those who ate 95 g. lost 30 mg. of calcium. Those who ate 142 g. lost 70 mg. of calcium (H. Linkswiler, M.B. Zemel, et al., "Protein-induced hypercalciuria," Federal Proceedings, July 1981).

The researchers concluded their study with this statement: "The calcium loss of 84 mg. daily, which occurred when the high-protein diet was fed, was substantial and, if continued over a period of time, would result in considerable loss of body calcium." The average American intake of protein is 105 g. per day (USDA, Home Economic Research Report, No. 52, September 1994).

Where did that calcium, lost to the body by eating too much protein, come from? It came from their bone reserves. This is an obvious conclusion, since 99% of the calcium in our bodies is in our bones. The high- protein intake group was losing calcium every day, sim- ply because of their high protein consumption. The ex- cessive protein was leaching calcium from bones, even though they were getting plenty of calcium in their diet (1,400 mg. of calcium daily, when the RDA recom- mended daily allowance is 1000 mg. for women). In con- trast, those on the lower-protein diet helped main- tain thicker and stronger bones.

HIGH-PROTEIN DIET LEADS TO OSTEOPORO-

SIS—Two University of Wisconsin researchers, Richard Mazess and Warren Mather, found that **Eskimos over**

the age of 40 have 10%-15% more bone loss than white Americans in the same age range (R.B. Mazess and W. Mather, "Bone mineral content of North Alaskan Eski- mos," American Journal of Clinical Nutrition, Septem- ber 1974). Checking on their diet, they found the Eski- mos eat more meat than even average Americans. They also found the Eskimos, with their fish, walrus and whale diet, get a very large amount of calcium from that meat (2,500 mg. daily), yet they lose more cal-cium than they take in. "The most obvious factor in the . . higher rate of bone loss in middle-aged Eski- mos would be their meat diet" (ibid.).

These and other studies clearly show that bone loss and osteoporosis is NOT related to a lack of calcium in the diet. The bigger problem is eating too much protein. If you are eating too much of it, you can eat all the high-calcium foods you want (such as vegetables and milk), yet your bone density will continue to decrease to dangerous levels. Western nations, which consume the most meat, have the highest rate of hip fractures (B.J. Abelow, T.R. Holford, K.L. Insogna, "Crosscultural as-sociation between dietary animal protein and hip fracture," Calciferous Tissue, January 1992).

MEAT ACIDS CAUSE THE BONE LOSS—Research

points to the large quantities of acids in meat as the cause of the calcium loss (W.J. Craig, "The Calcium Craze," Nutrition for the Nineties, 1992, pp. 131-146). As the blood becomes more acid, the blood takes cal-cium from the bones to neutralize the acidity. Increased calcium in the urine then provides a telltale sign of this bone loss (R.B. Mazess and W. Mather, "Bone mineral content of North Alaskan Eskimos," American Journal of Clinical Nutrition, September 1974).

As noted earlier, it is the major meat-eating nations

of the Western world which have the most hip fractures (*B.J. Abelow, ibid.*). An extensive study by four major research centers, working together, analyzed the risk of hip fracture in nearly 10,000 white women over 65. They found that a low-calcium intake—even below 400 mg. per day—did not cause hip fractures (*S.R. Cummings*,

M.C. Nevitt, et al., "Risk factors for hip fracture in white women," New England Journal of Medicine, March 1995).

Interestingly enough, higher consumption of veg- etable protein does not appear to be related to os- teoporosis or bone fractures (D. Feskanich, W.C. Willert, et al., "Protein consumption and bone fractures in women," American Journal of Epidemiology, March 1996).

Although calcium intake and hip fractures are unrelated in many studies, some studies have shown that **an increase in calcium intake can prevent osteoporosis, particularly when the calcium consumption is ad-equate and the protein intake is relatively low** before the age of 30 (R.R. Recker, K.M. Davies, et al., "Bone gain in young adult women," Journal of the American Medical Association, November 1992).

For your information, **soy beans and greens are a good natural source of calcium.** Some green leafy veg- etables (such as collards and lambs-quarters) have even more calcium per serving than soybeans. All the calcium a person needs can be obtained from the vegetable king-dom.

In addition, more calcium is assimilated from veg-etables than from dairy products or meat, because veg- etables contain less phosphorus. The excess phospho- rus in a meal tends to lock with calcium, in that meal, and carry it out of the body.

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FOR MUCH MORE INFORMATION

This bibliography will help you locate further information on these subjects. Many additional ar-ticles and reports are cited in this present book.

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