16CV-03080

Laura was given injections of an illegal drug known to be capable of transmitting an horrific brain disease, and it killed her.

I preface the summary of my lawsuit with background information about the disease, and also to establish a strong economic motivation for governments to hide or minimize public awareness of the risk of CJD.

My pleading alleges facts establishing negligence under the existing common-law doctrine of res ipsa loquitur.

BACKGROUND

Creutzfeldt–Jakob disease (CJD) is one of the most terrible ways to leave this earth. Once contracted, there is nothing anyone can do to mitigate the situation; it is always fatal, 100% of the time.

CJD is a rare neurological disease in the TSE category (Transmissible Spongiform Encephalopathy). An early form of CJD was called Kuru and was found in the Fore tribe on Papua New Guinea in the 1950s. Once the manner of transmission was determined (cannibalism, specifically of brain material) and subsequently eliminated, the cases started to drop off. However, Kuru can lay dormant for up to 40+ years, so at least one final case is expected soon.

In the USA, the official rate of CJD is always quoted as “one in a million.” That said, some agencies report that actual rate may be as high as 1.2+ per million in some years. It is difficult to determine the actual rate of the disease because governments, including ours, collude in hiding the numbers in order to prevent widespread panic. Even more difficult to determine is the rate of CJD prior to the 1990s.

85% of cases are considered to be sporadic Creutzfeldt–Jakob disease (sCJD), which means the cause has not been identified and, in most cases, is never identified. Another 10% to 15% of CJD cases are passed genetically (gCJD). Laura was tested for this during post-mortem autopsy of her brain, and the test for genetically transmitted CJD came up negative. So far, Iatrogenic CJD (iCJD) is responsible for 2% to 5% of CJD cases. Other even rarer forms of CJD include Gerstmann-Straussler-Scheinker Disease (GSS) and Fatal Familial Insomnia (FFI).

Variant Creutzfeldt–Jakob disease (vCJD) AKA “Mad-Cow Disease”

CJD is unofficially considered to be the “mother of Mad Cow Disease,” the official name of which is Variant Creutzfeldt–Jakob disease (vCJD). Variant CJD is acquired by eating meat (usually beef or venison) of an animal with BSE (Bovine Spongiform Encephalopathy). There is no question that the disease has jumped species. There have been at least 177 cases in the UK alone, and at least 52 in other countries.

There have also been 3 cases of vCJD acquired through blood transfusions in the UK. No other forms of CJD can be transmitted by blood, although, just to be on the safe side, anyone who has spent time in the UK during the "Mad Cow" period is not allowed to donate blood in the USA. The official rules are: "One may not donate blood if, from January 1, 1980, through December 31, 1996, you spent (visited or lived) a cumulative time of 3 months or more, in the United Kingdom (UK), or from January 1, 1980, to present, you had a blood transfusion in any country(ies) in the (UK) or France, or you spent (visited or lived) a cumulative time of 5 years or more from January 1, 1980, to present, in any combination of country(ies) in the whole of Europe including Eastern European countries that are now members of the EU."

At the time of this writing, only 4 cases of vCJD have been reported in the USA, and those 4 cases were traced to British beef. Nonetheless, America's beef-testing procedures are seriously deficient when compared to the testing standards in Europe, so in the near future, we may see cases of vCJD originating in America.

CJD and vCJD "Cover-ups"

vCJD has mainly appeared in the UK, resulting in the destruction of the beef industry in that country. Because of the potentially extended latency time, there may be thousands of vCJD cases still to become symptomatic. For 7 or more years, the UK government, intentionally hid the facts about vCJD from the public until 1995-96 in order to reduce the financial devastation they realized would ensue in their lucrative beef industry. I was living in the Netherlands at the time, so I witnessed the banning of British beef on the European continent, and I remember having to wear disposable "booties" whenever walking near sheep farms (vCJD originated in sheep with scrapie whose ground up brains were fed to cows.)

I have read that after Japan had one case of vCJD, their beef industry was wiped out. Subsequently, Japan had a total of 36 cases of vCJD.

Around 1996, Oprah Winfrey stated on her TV show, "Compared to CJD, AIDS is a 'walk in the park!" The next day, USA cattle stocks dropped 10% as well as cattle futures, and continued to drop until the Texas Cattle Ranchers Association filed a many-hundreds of millions of dollars lawsuit against Oprah. Eventually, Oprah won the suit, and the cattle industry recovered.

Discovery of "Prions" as the Transmission Agent for CJD

Also in 1996, Dr. Stanley Prusiner received the Nobel Prize in Medicine for identifying "prions" as the agent that causes CJD. Prions are proteins that are 100 times smaller than other proteins and do not contain any DNA or RNA. These prions sometimes "misfold" for reasons that are currently unknown. When they misfold, they coerce other prions to misfold, and it is this process that eats holes in the brain of the victim, leaving

their remnants as telltale “plaques” that can be identified in a post-mortem brain autopsy, which can also determine the type of CJD, as well as the subclass, and is the only way to conclusively diagnose the disease. Dr, Prusiner’s office did the diagnosis of Laura's CJD a week before she died, and this was confirmed by post-mortem brain autopsy done by the National Prion Disease Pathology Surveillance Center.

In recent years, studies have determined that these same prions play a role in Parkinson’s, early-onset Alzheimers, Lewey Body Alzheimers, Huntingtons Disease, Pick’s Disease, and other rare neurological diseases. It may be for this reason that Creutzfeldt–Jakob disease victims exhibit many or all of the symptoms of those diseases, but not in any fixed order, and therefore CJD is often misdiagnosed as one of the those diseases, or simply as dementia. Those other diseases rarely include a post-mortem brain autopsy, so the extent of this misdiagnosis is unknown. In many states, including North Carolina, CJD is officially a “reportable” disease, but in some other states, one isn’t even required to indicate CJD on the death certificate.

Latency between infection and the appearance of symptoms

CJD is thought to have a long latency before becoming symptomatic, and theoretically that can be 20 months to 40 years, although below I will question the validity of this claim. This is why 85% of cases are considered to be “sporadic Creutzfeldt–Jakob disease” (sCJD), which means the cause has not been identified. The reason this happens is due to the long latency before symptoms commence.

The universal law of cause and effect states that for every **effect** there is a definite **cause**, likewise for every cause there is a definite effect. Once the **cause** of a Sporadic Creutzfeldt–Jakob disease (sCJD) case has been identified, the diagnosis changes to Iatrogenic Creutzfeldt–Jakob disease (iCJD) meaning it was “acquired either through contaminated surgical instruments during brain surgery or corneal transplant, dura matter graft, or through growth hormones or gonadatropins.” This is because the post-mortem brain autopsies are identical for sCJD and iCJD; there is no discernible difference in the amyloid plaques (This is not the case with vCJD or gCJD.)

IATROGENIC CJD (caused by medical examination or treatment)

Surgeons sometimes unknowingly perform brain surgery on a patient whose CJD is still dormant, and then use the same tools on other patients who are thereby infected with the prions that spread the disease. Until very recently, surgeons did not know that CJD prions CANNOT be sterilized off of surgical equipment. This creates a conundrum because dormant CJD is difficult or impossible to detect and surgeons rarely test for it in advance of surgery. If the symptoms haven’t started, then there are no plaques to be seen in the brain during surgery, increasing the difficulty in detecting the possibility of accidental transmission. The Carolinas may have the highest rate of iCJD from surgery, or it may be that the Carolinas have the highest reporting rate of iCJD from surgery. Efforts are being made to minimize panic about this disease.

Japan has an extremely high rate of cases of iCJD contracted from dura-matter grafts (and aneurism clips?), a very common operation in that country, much more common than anywhere else in the world.

iCJD can also be transmitted by drugs containing hormones of a person who has dormant CJD that is pre-symptomatic. Cadaveric glands are another source of hormones extracted from people who have died BEFORE they became symptomatic.This is yet another dilemma.

In the 1970s and 1980s, there were many cases of iCJD due to the use of Human Growth Hormone (HGH) in very young children. North Carolina and Tennessee had a large number of these cases. Children as young as 5 were dying in unspeakable suffering. And for someone who has witnessed an adult die from CJD, the thought of a child dying that way is far beyond the worst nightmare conceivable. Fortunately, most people are unable to imagine such profound agony and terror, having never seen a CJD patient go through it. Seeing someone endure having holes eaten into their brain, with no hope except to die as quickly as possible, transforms one's entire worldview.

In the late 1970s, this high incidence of CJD in children was traced to HGH, their diagnoses were then changed from sCJD to iCJD, and HGH was banned and replaced with recombinant (synthetic) HGH that is presumably safe.

LAURA'S CASE OF CJD

Laura's case was sCJD (type M/M 1), and I believe it will turn out to have been iCJD. I am trying to establish as fact or as a reasonable assumption that Laura acquired CJD through hormone injections.

Laura had degrees in microbiology and epidemiology, and she had heard about Kuru in the late 1970s. Because of this, she had developed a healthy fear of CJD and was extremely cautious about it in her profession. She would not have consented to any treatment that put her at risk for CJD. Moreover, because Laura had decades of experience working as a clinical research monitor, there is no chance she would have allowed herself to be injected with a drug that had been made illegal.

Human Chorionic Gonadotropin (HCG)

Early HCG had been derived from pituitary hormones (p-HCG), and those had been proven to cause CJD, when one donor infected 4 other women in Australia. The FDA had preemptively made pituitary-derived HCG illegal, so there were no reported cases in the USA, thanks to it being banned so early by the FDA. Additionally, cases of HCG infecting people with vCJD occurred in Europe, although this may not be surprising because vCGD is transmitted by blood, beef, and is seen in tonsils and other organs, and seems to have a more varied and greater number of transmission routes.

A study released on March 23, 2011, entitled “Detection of Prion Protein in Urine-Derived Injectable Fertility Products by a Targeted Proteomic Approach” established the fact that infective prions had been detected in urinary-derived Human Chorionic Gonadotropin (HCG, AKA: u-HCG).

Banning of HCG

Although HCG is a fertility drug, many weight reduction clinics claim it will reduce weight —an “off-label” use. That was definitively proven to be 100% a hoax 21 years ago, and since then, the FDA has required warnings, disclaimers, and much more. The clinic injecting Laura did not provide any of the FDA required warnings or disclaimers.

After the 2011 “Detection of Prion Protein in Urine-Derived Injectable Fertility Products” study, mass publicity ensued claiming that CJD could be caught from HCG, and others reported vCJD or “Mad Cow Disease” could be contracted from HCG (not knowing the difference between vCJD and CJD). Further studies in August 2011, confirmed the first study. Then, on December 6, 2011, the FDA banned HCG and ordered many weight-reduction clinics to destroy existing stock, and publicized the banning quite effectively. Just as they had with p-HCG, the FDA pre-emptively banned u-HCG. Unfortunately for my wife, some doctors did not uphold the widely publicized ban.

Weight-reduction doctors in Asheville ignored the FDA’s banning of HCG, and continued to use this illegal drug for an off-label use that had been repeatedly proven to be a hoax. They gave my wife at least five injections 10 months after the drug was made illegal. And 20 months later, her CJD symptoms commenced (Another study shows that when CJD is acquired from fertility Gonadotropin products, it has a latency of 20 months for vCJD and or about 2 years for sCJD.)

CJD caused by u-HCG

Although there have been cases of p-HCG transmitting CJD and u-HCG transmitting vCJD, so far, at the time of this writing, the number of cases of u-HCG resulting in sCJD (meaning the sCJD would be rebranded as iCJD if the cause had been known) has been zero, as far as I can tell, although many cases of CJD are unreported, and, if other women who died of CJD hid their use of u-HCG in the same way my wife did, their cases would never be counted. (Much to my surprise, I discovered the receipts for Laura’s injections of HCG several months before the 2nd anniversary of her death, hence the filing of my lawsuit one day prior to the 2nd anniversary of her death.)

I’ve talked to one of the authors of the “Detection of prions in…[HCG]” and also the CJD researcher who did my wife’s brain autopsy to see if they’d found any cases of CJD from urine-derived fertility products. The response was that although this possibility had been demonstrated in a lab, until there was an actual human case, it was simply theoretical.

Debunking Skeptics

I believe my wife was the first case, or at least the first reported case in the USA.

My conclusion has been met with the claim that millions of women have taken u-HCG with no cases reported. According to my calculations, based upon claims that 300,000 women have been taking u-HCG fertility drugs for 40 years, that means 12 million (or more) women have taken u-HCG injections, so when one believes the rate to be one in a million, it *appears* to not be occurring.

Additionally, some researchers claim that because the division of male vs. female cases has remained constant over the years with women being slightly more at risk then men — I seem to recall it is 51% women and 49% men — that is further proof that u-HCG is not transmitting the disease, because, as women are the only people who take injectable u-HCG, that would have caused a spike in the number of female cases [Of course, they are not counting the many body-building companies touting HCG for men, and if the reader may recall that back over recent years, there have been cases of weight-lifters and body-builders developing strange early dementias (strange as defined by the press) and even killing themselves because of that. Having read their symptoms, I suspect those were cases of CJD caused by u-HCG, but that may be irrelevant here.]

u-HCG requires donor urine from pregnant women (under 40 years of age) who has pre symptomatic CJD

There is one thing many research doctor don't mention, and perhaps they are simply missing the obvious, because they are too close to the focus of their personal research niche. That is the fact that the overwhelming majority of CJD cases become symptomatic after the age of 60, in fact, the mid-60s is the mean age. There are NO pregnant women of that age. In fact there are no pregnant women even in their fifties, and precious few in their forties.

But u-HCG requires urine from a pregnant woman. Moreover, for u-HCG to transmit CJD under non-laboratory conditions, the donor must actually have CJD. The incidence of CJD among women of child-bearing age is very significantly lower than the incidence of women in their 40s, 50s, or 60s who have CJD.

Because the urine-derived HCG must come from the urine of a pregnant women with CJD, she would have to be under the age of 40, and it should be possible to determine the number of cases of pregnant women with CJD or who become symptomatic after donating urine for HCG or who might have had their urine stolen from them for sale to a u-HCG production company. Whatever the figure, it is tempered by the fact that the donor must have been under 40. That is not open to dispute. Women do not get pregnant after the age of 40 in sufficient numbers to effect this issue. Moreover, in order to confirm the transmission, that donor must have subsequently died of CJD after her donation, had her death reported as CJD, and it made it known that she had donated urine for u-HCG; three factors that increase the chance of overlooking the transmittal.

Looking at a scatter chart of CJD age distribution, it seems that the incidence of CJD in women of child-bearing age (younger than 40), may very well be one in 12-million (or many more) which DOES correspond to the number of women who have had u-HCG injections, and if so, Laura could certainly be the very first case. It may be overdue!

Shorter latency period for CJD when transmitted by HCG

The idea of the latency period stretching up to 40 years seems completely incompatible with the concept of "sporadic" CJD, because the very name "sporadic" indicates that the transmission source is unknown and, consequently, the date of transmission is unknown. Therefore, it is impossible to determine the theoretical latency period, and therefore, the latency period is not determinable in most cases of sCJD, and thus, the same goes for iCJD until additional cases prove otherwise. That having been said, concrete data DOES exist for the latency periods of hormonally transmitted iCJD, and this is easily determined for HGH. From what I have read in studies, including those in the Exhibits, for CJD contracted from HCG there is a latency of 20 months or 2 years.

Laura's CJD appeared 20 months after her final HCG injection, and 22 months after her initial HCG injection; consequently, she fits perfectly within the HCG-transmitted CJG latency of 20 months to 2 years.

Considering the above facts, objections to the possibility that Laura could not have been infected due to the "one-in-a-million" incidence rule, or due to the long latency claims (although no-one has objected on the basis of the latency claims) are spurious. They have no basis in reality.

THIS IS WHAT IS KNOWN TO BE TRUE

- Laura was given 5 injections of u-HCG 20 months before she became symptomatic with CJD
- Cases of iCJD contracted from p-HCG are known to have had a latency of about 2 years.
- Cases of vCJD contracted from HCG are reported to have had a latency of 20 months.
- u-HCG has been proven (in the lab) to be able to transmit the prions responsible for CJD
- Subsequent to the initial studies, additional studies verified the findings
- Shortly after this was proven, the FDA made u-HCG illegal
- The drug was made illegal 10 months before Laura was injected.
- Prior to being made illegal, weight-reduction was an off-label use of the drug strongly discouraged by the FDA because it had been determined to be a hoax.
- The FDA had required a warning for about 20 years that such use was completely ineffective.

- Laura was not given that warning, and that warning does not even now appear at the clinic.
- She was not informed of the CJD risk, otherwise she would not have agreed to the treatment.
- For every lab-proven (AKA “theoretical”) danger of any drug, there will be a first human case.
- I believe Laura is that first human case (at least, having been reported in the USA) for u-HCG-transmitted Creutzfeldt–Jakob disease

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SUMMARY: These irresponsible doctors gave my wife injections of an illegal drug, that had been proven to be able to transmit CJD, for an off-label use that had been proven to be a hoax and a notice of that was required by the FDA. The doctors did not inform her of the risks of the drug, or that the drug was illegal. Her CJD symptoms began 20 months after the final injection, and she died two months later, after suffering the most horrible brain disease known to humankind.