Adopt a Pathogen
Bovine Spongiform Encephalopathy (BSE)
Creutzfeldt Jakob Disease (CJD)

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Contents

Chapter 1

At a Glance

1.1 Morphology and Size

BSE and human variation call Creutzfeldt-Jakob disease is caused by the presence of abnormal prion PrP^{Sc}. Both the normal and abnormal prion are composed of 253 amino acids the difference between the two arises when observing how the protein is folded. The normal prion has a function folding other proteins, its abnormal counter part does a similar thing but fold other similar proteins into them selves. This creates replication loop similar to a virus.

Note:-

Since prions are simply proteins "sanitizing" prions is extremely difficult as the protein is not "Killed" until it is denatured. This process requires extreme sustained heat.

1.2 Taxonomic Data

Prions are oddly unique as they are a misfolded protein and the only occurrence of a protein replicating like a virus. Currently the only know disease causing prion is PrP^{Sc}. Similar to how viruses evolve into existence prions can enter a disease free population. This likely hood is increases if organisms are frequently consuming themselves.

1.3 Reservoir

The process of infection occurs when a PrP^{Sc} abnormal protein enters an organism that produces PrP^{C} proteins. The abnormal protein proceeds to convert normal proteins into their counterparts. This affects the reservoir as prions can be present in an organism with neurons as PrP^{C} is a required protein with multiple functions within the neuron.

1.4 Mode of Transmission

Prions have been observed transmitting between neuron cells within an organism but an infected individual never becomes infectious. Prions are propagated to other individuals by the ingestion of tissue from an affected organism. In the meat industry is was common for meat scraps to end up as feed for cattle. This create mechanism for the spread of prions that cannot occur naturally.

1.5 Natural History of Disease

The first cases in North America were reported in 1993. This was shortly after the adoption of animal products into animal feed. After this relationship was discovered both the US and Canada passes legislation banning animal tissues entering animal feed. As a result an occasional case or outbreak will occur around once every five years.

1.6 Means of Diagnosis

Currently the only means of diagnosis is by brain biopsy. Often times the diagnosis is suspected by the symptoms not being explained by another more easily diagnose disease. At this point the is not cure, vaccine, or sanitizer to deal with prions.

Chapter 2

History and Burden

2.1 History of Discovery

This first case of BSE was diagnosed in 1986 in the United Kingdoms. This initial outbreak started with 2 infected cows that researchers believe became infected around 1970. As a result of the long onset of symptoms for this disease the presence of prions can go long undetected. Once the protien was identified as the cause the public became concerned with how many cows may have been infected during the time of 1970-1986. Because of the practices in the comercial beef production sector the outbreak had grown quite large.

2.2 History of Burden

In response to the intial outbreak the United Kingdoms implemented strong quarantine and clean up measures. Due to the resilience of the protien prions clean up efforts entailed to eliminating all cows found on farms where cases were reported. After this inital outbreak the United Kingdoms experiences the most human cross overs than any other country and continue to suffer the most burden globally. Since 1996 the UK has reported 178 instances of vCJD the second highest country is France with only 27 cases.

2.3 Current Global Burden Distribution

Currently the majority of the global burden in center in the UK with 5x the cases than any other country. The outbreak has spread globally with a few country reporting cases every 5ish years.

Chapter 3

Countermeasures

3.1 Methods

Currently the majority of prevention efforts go towards surveiling the commercial beef industry. European countries like to randomly test mature cattle to see if in outbreak has occured. Japan has regualtions that tests at least one cow from each farm. Another form of regulation that limits the spread is the special classification of tissues that can contain the prion that prevents is from becoming animal feed in the future.
Inn response to the initial outbreak in the early 2000s places such as the US and Australia implemented ristrictions on Spanish and UK immgrant blood donars. The US lifted these restrictions during the COVID blood shortage.

3.2 Efficacy

Since the prion never causes an infectious period and its spread is solely caused by the commerical cattle feed network industry regulations prove very effective. While the UK deals with the majority of the burden with the most cases globally there only a couple of hundred cases of BSE and a handful of CJD cases. From a modeling perspective the industry regulations destroy the contact network for the prion.

Chapter 4

Modeling

4.1 Modeling Approaches

Since the spread of the prion is a man made side effect models that are between host of a single species population do not yield insightful results. How every as a side effect of the slow developement of the disease a within host model can describe how infectious an individuals tissues may have become.

4.2 Paper Review

The modeling paper I chose to review was "Modeling of bovine spongiform encephalopathy in a two-species feedback loop" by Richard Barnes and Clarence Lehman. This paper's focus was to see the spread of the disease from one feedback loop to another loosely connect population. Some of the core principal were making an age adjusted contact matrix and the effect of lifespan on the progression of the disease.
The paper created a strong foundation in the mathmatical functions used to model the progression of the disease within a host to justify values in the contact matrix. The focus on the within host prion count leads to conclusions about how the disease needs a resivoir with relatively long lifespans.