The relationship between etiology (persistent diseases) and sources of nutrition (diet and fodder) for animals in Iraq: systemic review

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Abstract:
Definition of fixed, such as those that are not cleared of the virus but is still in certain cells of infected animals infection. It may lead to persistent inflammation and infection stages of both static and product without the injured animal was killed quickly or produce excessive damage to the host cells.
The aim of the study is to find out the relationship between the persistent viral diseases of animals, feed and feed manufacturers
The study type: compile relevant studies concerning systemic review
Mode of action: fear of the occurrence of such diseases in livestock to food relationship to the fact that diseases like so type require years of follow-up, as well as epidemiological spaces to appear in the country producing and exporting livestock for food during the years of the epidemic .thalil food and readjustment.

Recommendations:
1 - There are no ways to detect the process, at the present time.
2 - prion prions in the form of abnormal resistance to most of the thermal and chemical treatments.
3 - The particular food manufacturing processes do not lead to a significant reduction in prion infection through exclusion.
4 - There is no known way of readjustment contaminated foods. To protect food key is to get the beef and meat by-products from animals infected with mad cow disease and protect against contamination of food with high-risk tissues, especially the brain tissue and spinal cord.

Conclusion: Moderate to high quality evidence that BSE transmission to animal (cattle, sheep...) by infected food (feed manufacturers) as well as in human.
Introduction

Persistent infections are characterized as those in which the virus is not cleared but remains in specific cells of infected individuals. Persistent infections may involve stages of both silent and productive infection without rapidly killing or even producing excessive damage of the host cells. There are three types of overlapping persistent virus-host interaction that may be defined as latent, chronic and slow infection (1).

Prions are normal proteins of animal tissues that can misfold and become infectious: they are not cellular organisms or viruses. In their normal noninfectious state, these proteins may be involved in cell-to-cell communication. When these proteins become abnormally shaped i.e., infectious prions, they are thought to come into contact with a normally shaped protein and transform that protein into the abnormally shaped prion. This process causes a geometric increase of abnormally shaped prion proteins until the number of abnormally shaped protein causes overt illness. The "prion theory" of Transmissible Spongiform Encephalopathies (TSEs) is widely accepted (2)(17).

The mechanisms (Pathogenesis ) : persistent infections are maintained involve both modulation of virus and cellular gene expression and modification of the host immune response. Reactivation of a latent infection may be triggered by various stimuli, including changes in cell physiology, superinfection by another virus, and physical stress or trauma. Host immunosuppression is often associated with reactivation of a number of persistent virus infections.

Etiology :transmission of agent causing BSE in cattle by ingestion of contamination diet or folder (3).

The abnormally shaped prions are resistant to most heat and chemical treatments, however certain food manufacturing processes (e.g. gelatin production) do result in significant decrease in prion infectivity through exclusion. There are no known means of reconditioning contaminated foods. The key to food protection is obtaining bovine meat and meat byproducts from animals not infected with BSE and protecting against contamination of food with high risk tissues, especially brain and spinal cord tissue (4).

The virus may induce disease and can be detected in cells of several organs (e.g., kidney, lung, and those of
the digestive or central nervous system. Three kinds of persistent infection can be maintained in cell cultures: chronic focal, chronic diffuse, and latent (5).

Control by interferon and antiviral drugs can reduce the frequency of clinical recurrence and ameliorate clinical symptom, yet the virus continues to remain associated with the host (6).

Prion protein (PrP) molecules are encoded by wild type or mutated cellular genes that are excluded from the particles. The human PrPs gene can be mapped to the short arm of chromosome 20. A long incubation period (often years to decades) with slowly rising and spreading infection precedes the onset of clinical illness and is followed by chronic progressive disease. The host shows no inflammatory response, no humoral or cellular immune response, and no interferon production. Immunosuppression of the host has no effect on pathogenesis or progression of disease. During persistent infections, the viral genome may be either stably integrated into the cellular DNA (7) (18).

Blood meal: Manufactured by passage of a stream of steam through the blood until the temperature reaches 100 m, In order to ensure the sterilization process and then dried by heating with steam and then milled and 80% crude protein and it is used in poultry diets low by 2 3% (8).

Methods:
The data were related to research of local and international scientific journals and scientific books from appearance the disease to the present time collection from 15 years ago. The advantage of the English and Arabic sources, was. Data and analysis through programs and methods of modern research has put this to encourage systematic and scalable methods to repeat in the narrative synthesis and enhance the transparency of reporting and assessment of the robustness of the results. Provide guidance and tools different methods for the auditors. We used specific criteria to give each country a degree of authenticity. It was a discussion and review of scientific research and numerous statistical methods. Data were studying after the removal of a group of studies is believed to be possible sources of bias. We studied the relationship causal food consumed and how affected the results by assessing the effect of removing the causative.

Results:
Systemic review in this study depending of collection results had retrospective studies among 30 years until 2015.
**Figure (1):** In this map free Iraq and neighboring countries show of BSE infection by WHO 2015 and the reason is now using agricultural residues such as wheat and barley and to sow clover and alfalfa or because of the lack of study on the virus.

**Figure (3):** Show from 1980-2010 that infected animals entering food supply and related with incidence of disease, all study and reported the sought outcome describes the follow up outcome for each study. During ten years (1985-1995) peak exposure infected animals entering food supply (rich of protein) appearance of symptom in human by variant form of Creutzfeldt-Jakob disease (vCJD) incidence after five years (1995-2000) that mean good outcome in ten years overall, 400000 infected animals out of 40 exposure human/1-12 years.

**Discussion**

Iraq stopped the import of a consignment of imported beef from Brazil in 2012 and because it was questionable in the presence of BSE infection in the region, as well as periodic inspection on the herds of sheep, cows and feed use Computer Hotline national product of feedstuff (CDC: 2012). Feed supply strengthened the 1997 rule in 2008 by prohibiting the use of the highest risk cattle tissues in ALL animal feed. These high risk cattle materials are the brains and spinal cords from cattle 30 months of age and older, and the entire
carcass of cattle not inspected and passed for human consumption, unless the carcasses are shown to be from cattle less than 30 months of age, or the brains and spinal cords have been removed. To date, four cases of BSE have been detected in the United States. The first case was detected in 2003 in a cow imported from Canada. Three cases have since been detected in U.S. born cattle, but laboratory evidence suggests that these cases had atypical strains of BSE, that is not the same strain that caused the large outbreak in the United Kingdom\textsuperscript{(16)}.

Viruses play a key role in the complex etiology of bovine respiratory disease (BRD). Bovine viral diarrhea virus 1 (BVDV-1) is widespread in Australia and has been shown to contribute to BRD occurrence. As part of a prospective longitudinal study on BRD, effects of exposure to BVDV-1 on risk of BRD in Australian feedlot cattle were investigated. A total of 35,160 animals were enrolled at induction (when animals were identified and characteristics recorded), held in feedlot pens with other cattle (cohorts) and monitored for occurrence of BRD over the first 50 days following induction. Biological samples collected from all animals were tested to determine which animals were persistently infected (PI) with BVDV-1\textsuperscript{(10)}.

There is one reported human cases of vCJD in the United States in a woman that appears to have acquired the illness from consumption of contaminated food when growing up in the United Kingdom. In the U. K., in France and one in Italy. Since 1986, more than 180,000 cases of BSE have occurred in the U.K. in cattle, particularly dairy cattle. BSE cases have also been identified in 20 European countries, Japan, and Canada. The feeding of rendered TSE-infected animal by-products to cattle is believed to have caused the epidemic of BSE. There is one reported case of BSE in the U.S. which appears to be the result of importing cattle from Canada that may have been exposed to feed which contained meat and bone meal from rendered cattle\textsuperscript{(11)}. Research indicates that the first probable infections of BSE in cows occurred during the 1970's with two cases of BSE being identified in 1986. BSE possibly originated as a result of feeding cattle meat-and-bone meal that contained BSE-infected products from a spontaneously occurring case of BSE or scrapie-infected sheep products. Scrapie is a prion disease of sheep. There is strong evidence and general agreement that the outbreak was then amplified and spread throughout the United Kingdom cattle industry by feeding rendered, prion-infected, bovine meat-and-bone meal to young calves\textsuperscript{(15)}.

The BSE epizootic in the United Kingdom peaked in January 1993 at almost 1,000 new cases per week. Over the next 17 years, the annual numbers of BSE cases has dropped sharply; 14,562 cases in 1995, 1,443 in 2000, 225 in 2005 and 11 cases in 2010. Cumulatively, through the end of 2010, more than 184,500 cases of BSE had been confirmed in the United Kingdom alone in more than 35,000 herds.

Such an effort could help us to describe the diversity of microbial agents to which our species is exposed to characterize animal pathogens that might threaten us in the future; and perhaps to detect and control a local human emergence before it has a chance to spread\textsuperscript{(12)}.

Monitoring should focus on people with high levels of exposure to wild animals, such as hunters, butchers of wild game, wildlife veterinarians, workers in the wildlife trade, and zoo
workers. Such people regularly become infected with animal viruses, and their infections can be monitored over time and traced to other people in contact with them. One of us (N.D.W.) has been working in Cameroon to monitor microbes in people who hunt wild game, in other people in their community, and in their animal prey (Wolfe et al., 2004). The study is now expanding to other continents and to monitor domestic animals (such as dogs) that live in close proximity to humans but are exposed to wild animals through hunting and scavenging. Monitoring of people, animals, and animal die-offs (Kuiken et al., 2005) will serve as an early warning system for disease emergence, while also providing a unique archive of pathogens infecting humans and the animals to which we are exposed.

Recommendations:
1 - There are no ways to detect the process, at the present time, but by screening tests early detection way.
2 - Prion prions in the form of abnormal resistance to most of the thermal and chemical treatments, this way of prepare diet of animals.
3 - The particular food manufacturing processes do not lead to a significant reduction in prion infection through exclusion.
4 - There is no known way of readjustment contaminated foods. To protect food key is to get the beef and meat by-products from animals infected with mad cow disease and protect against contamination of food with high-risk tissues, especially the brain tissue and spinal cord.

Reference
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