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Mini Review

On a Simple Model for Creutzfeldt Jakob Disease - @

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ABSTRACT

Creutzfeldt Jakob Disease (CJD) is a fatal disease which is transmitted by the ingestion of infectious materials (mainly BSE-contaminated beef). Here a simple mathematical model of its progress is given.

Transmissible Spongiform Encephalopathies (TSE's), also known as prion (proteinaceous infectious particles) diseases, [1,2] are a group of fatal brain diseases that affect both animals and humans. Infective prions propagate by refolding into abnormal structures which cause the conversion of normal protein molecules into the abnormally structured forms. These prions are resistant to inactivation by conventional decontamination methods. They also resist all routine sterilization procedures commonly used in health care facilities. Because of the unconventional transmissions of these agents, they are of particular concern for public health.

Variant Creutzfeldt Jakob Disease (vCJD) is a human form of Bovine (Transmissible) Spongiform Encephalopathy or mad cow disease. Variant CJD is an infectious disease typified by long incubation periods and asymptomatic infections [two factors making epidemiological investigations particularly difficult. Two modes of infection of vCJD have been identified:

PRIMARY INFECTIONS

Associated with the ingestion of infectious materials (mainly BSE-contaminated beef).

SECONDARY INFECTIONS

Associated with receiving blood from an infected blood pool (particularly through transfusions) and with the use of infected surgical instruments. Motivated by realizing that the interaction between wrongly folded prions and the healthy ones is similar to the interaction between infected persons with infectious disease and healthy ones the following model is presented:

Let x be healthy (properly folded) prions and y be infected (wrongly folded) ones. Then their interaction can be modeled by the simple equations:

$$dx/dt = ax(1-x) - bxy/(x+y)$$

$$dy/dt = bxy/(x+y) - y \quad (1)$$

Which means the change rate of both healthy (x) and infected (y) prions. The constants a, b are positive constants which represents the growth rate of healthy prions and the contact between healthy and infected prions respectively. We rescaled other parameters.

Only equilibrium solutions with positive x are considered hence we get the healthy equilibrium ($x = 1, y = 0$) and the diseased one where both x and y are positive:

$$X = 1 - (b-1)/a, y = (b-1)x \quad (2)$$

Hence a necessary condition for the existence of the diseased state is:

$$a > b-1 > 0 \quad (3)$$

It is stable if

$$a > 2(b-1) \quad (4)$$

Despite being a simple model, it indicates the importance of the parameter a (the growth rate for the healthy prions), for the presence of the disease.

Since CJD works in the brain it is highly likely that networks will be used to describe it [3,4]. In order to establish model (1) on homogenous networks, we assume two hypotheses:

Homogeneity

The network is homogeneous. For simplicity, assume that each node has degree k is the average connectivity in the network neglecting the heterogeneity of the node degrees.

Homogenous mixing

The infection strength is proportional to the population density.

The meaning of these two assumptions is that every prion has the same average connectivity to other prions.

Combining the above hypotheses, the proposed novel model is

$$dx/dt = a x (1-x) - b(k) xy/(x+y)$$

$$dy/dt = b(k) xy/(x+y) - y \quad (5)$$

A more realistic model will require inhomogeneous networks but we refer this to future work.

REFERENCES

1. Al-Zoughool M, and El Saadany S. Mathematical Models for Estimating the Risk of vCJD Transmission. Proceedings of the OCCAM-Fields-MITACS Biomedical Problem Solving Workshop. 2009. <https://goo.gl/YTtHA4>
2. Creutzfeldt Jakob Disease. Wikipedia. <https://goo.gl/wBK6Rd>
3. Yao Hua, Lequan Minab, Yang Kuangc. Modeling the dynamics of epidemic spreading on homogenous and heterogeneous networks. *Applicable Analysis*. 2014; 94: 2308-2330. <https://goo.gl/37E2FT>
4. Suzanne S Sindi. Mathematical Modeling of Prion Disease. INTECH. 2017. <https://goo.gl/CqCpkA>