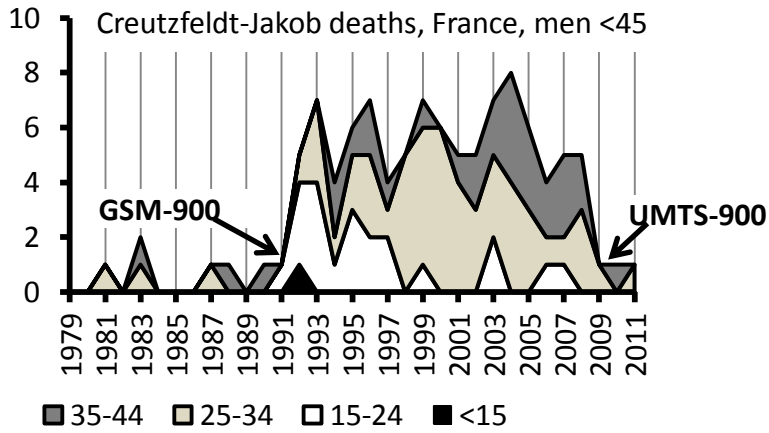
# Interaction of prion diseases with electromagnetic fields.

This summary is limited to Creutzfeldt-Jakob disease (CJD) in the less than 44 years, and to Bovine Spongiform Encephalopathy (BSE). These diseases (in the above age category for CJD) were very rare before the introduction of radio frequency emissions in the 900 MHz band. The very strong changes which affected these diseases in France and the UK, and the fact that these diseases reacted to changes within the 900 MHz band, do facilitate the analysis and understanding of the problem.

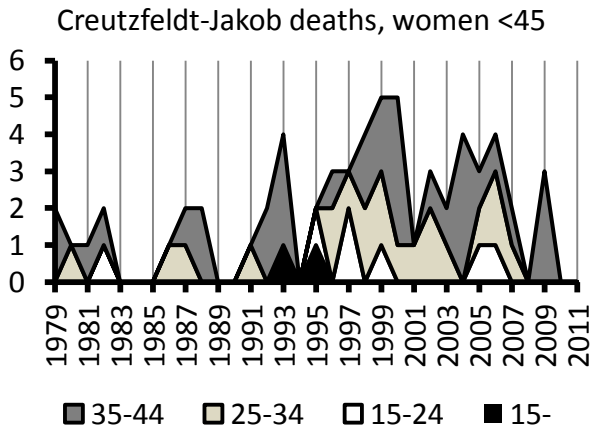
## Creutzfeldt-Jakob disease (CJD) in the younger population in France

Deaths by Creutzfeldt-Jakob disease in France in men less than 45 years old were very rare prior to 1990. They increased brutally in 1991/92 (onset of GSM-900) and decreased brutally in 2009 (onset of UMTS-900).

In 1991/1992 the first deaths were amongst the youngest (less than 25 years old). This can be explained by a faster renewal of immune cells (faster production of T cells by the thymus) yielding a faster reaction to the onset of GSM-900 (a reaction similar to a pro-auto-immune effect).




In women the change is less contrasted. Some differences are explainable by a temporary effect analog to an anti-auto-immune effect at onset of networks or during accelerated increase of network coverage. In particular, the final stopping of abnormal deaths is shifted by one year.




**This yields one hypothesis: the onset of a GSM-900 network causes an abnormal increase of CJD cases MCJ and the onset of a GSM-900 network stops these abnormal cases.**

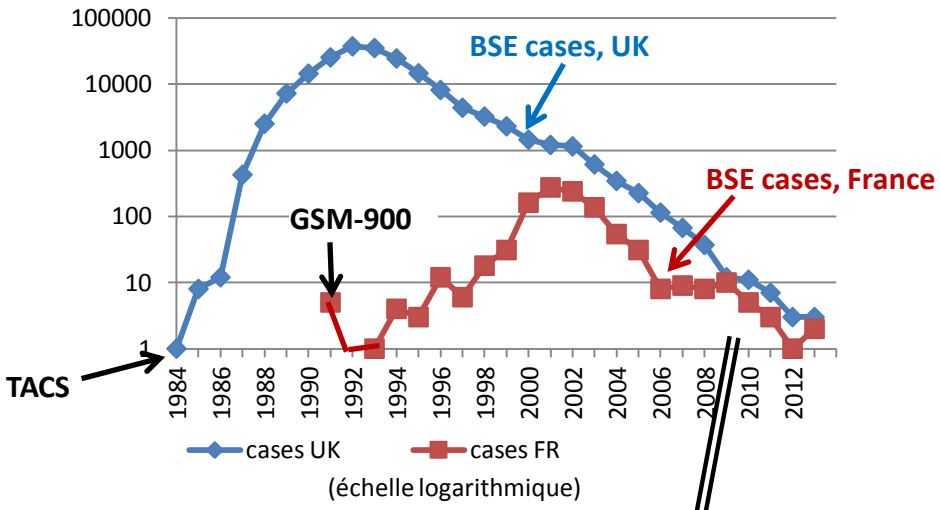
**Do we find such phenomenas elsewhere ?**

## Bovine Spongiform Encephalopathy (BSE)

Bovine Spongiform Encephalopathy (BSE) is the equivalent in cows of Creutzfeldt-Jakob disease in men (CJD).

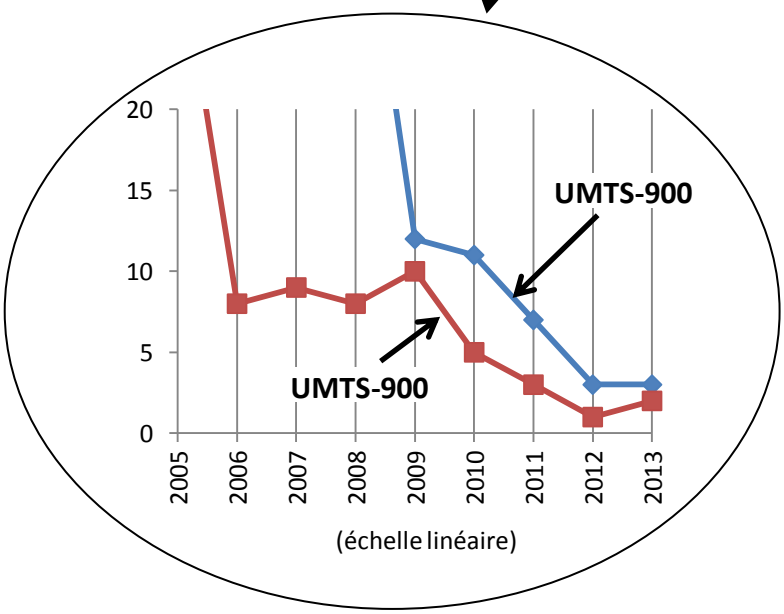
The BSE epidemic in the UK was started by the onset of TACS, an analog telephone network using the same frequencies as GSM-900 and using variable frequency carriers as GSM does – a network which is near equivalent to GSM in a physical point of view.

The BSE epidemic in France was started by the onset of the GSM-900 network. The corresponding frequencies were unused before..




Both epidemics were stopped by slaughtering of diseased animals and suppression of prions in cow's meals. But after the end of the epidemic phase, a largely constant number of annual cases remained, which was more visible in France because it lasted several years.

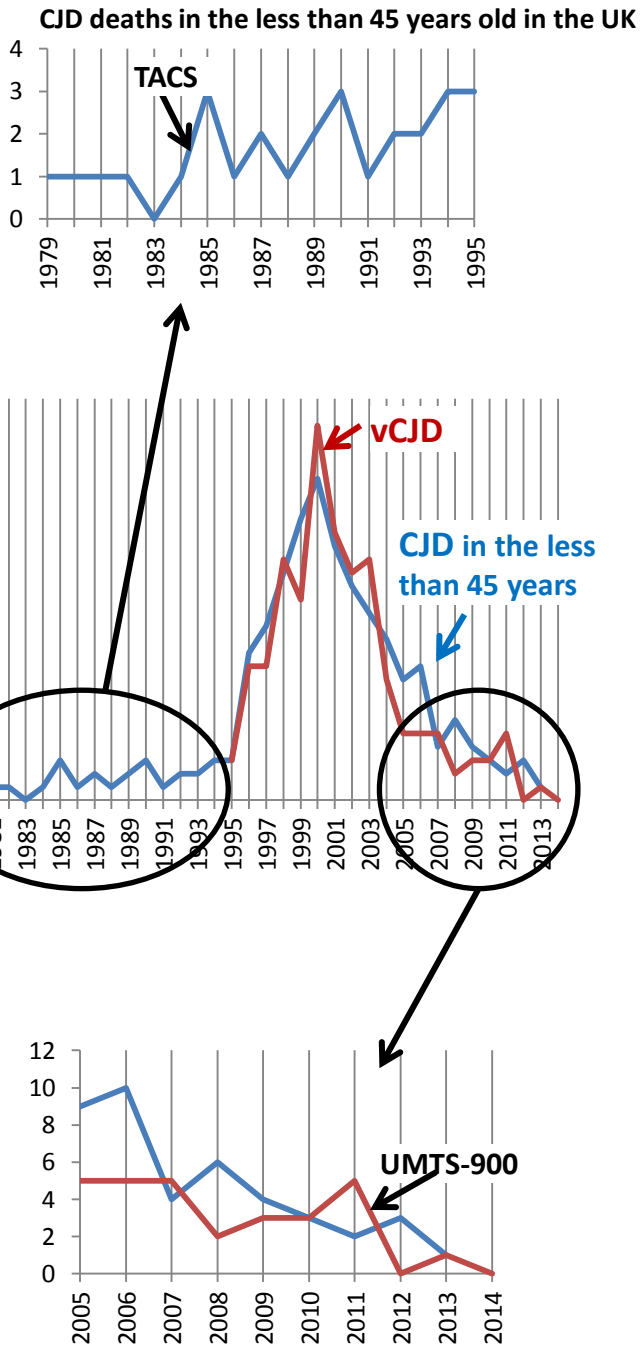
The stabilized number of cases was strongly reduced at the onset of UMTS-900 in both countries.




**The BSE epidemiology confirms the conclusions: the epidemic was triggered by the onset of TACS/GSM-900 and the number of yearly deaths was finally reduced by the onset of UMTS-900. This took place independently in France and the UK at two different dates.**

## CJD and variant CJS (vCJD) in the United Kingdom.

In 1984 at onset of TACS, CJD deaths increased, but non-significantly. Maybe the difference as compared to France is due to a different way of eating meat: prion transmission is probably less common from well-cooked meat.




Then an epidemic phase started. Most deaths of younger persons were due to variant CJD.

vCJD deaths then stabilized from 2005 onwards and decreased strongly in 2012 following the onset of GSM-900.
The timing of the end of abnormal vCJD deaths in the UK is the same as the timing of the end of CJD in France in the less than 45 years old (men and women together) : one year after commercial opening.

**The near cessation of abnormal vCJD deaths following the introduction of UMTS-900 in the United Kingdom confirms the hypothesis,** and the reaction of CJD during introduction of TACS is compatible with the hypothesis although non-significant. The factors that started and ended the epidemic phase remain ill-understood.

## What can we deduce from the facts?

**1- TACS and GSM-900 cause an abnormal increase of CJD and BSE cases.**

**2- UMTS-900 stops the abnormally high number of cases of vCJD, CJD andBSE.**

**3- Both effects take place at very low power.** In 1991/1992 the average exposure to GSM-900 of the French population, and moreover of the French cows, was very low. In 2009, the average exposure to UMTS-900 was against very weak.

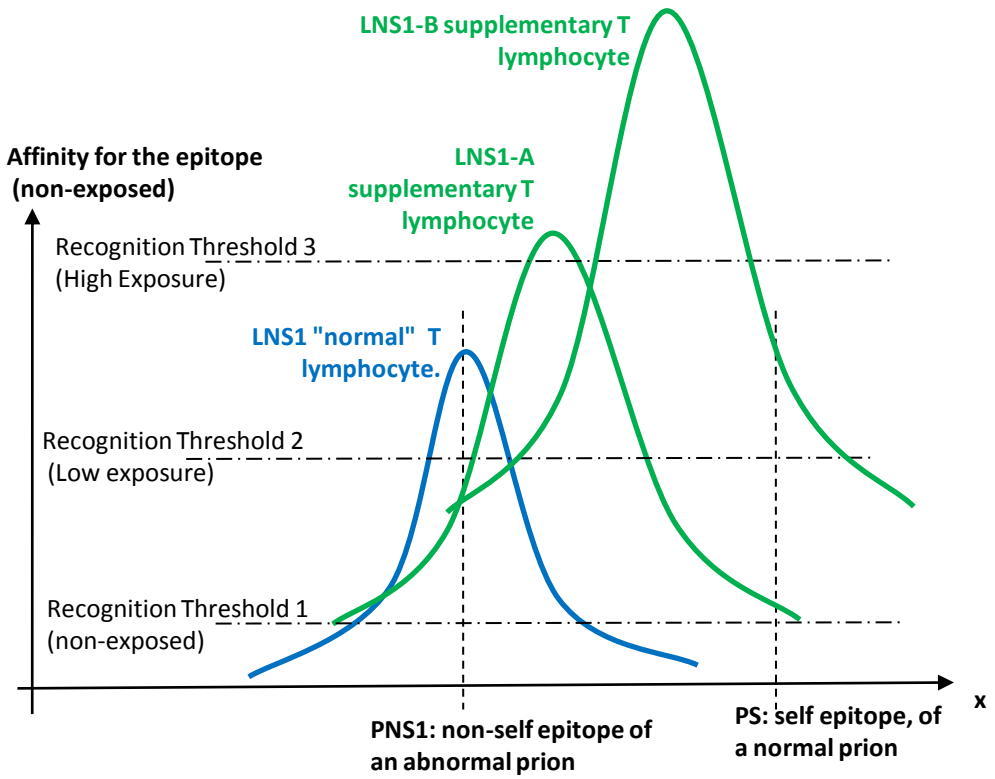
**4- Effects depend on modulation:** GSM-900 and UMTS-900 have opposite effects but occupy the same bands and differ only through their modulation.

## CJD: a simplified description.

Creutzfeldt-Jakob disease (CJD) is caused by a protein (prion) which is normally present in the organism, but changes its shape to adopt an abnormal and pathogenic conformation. Then the multiplication of abnormal prions causes the disease.

Prions pass from a normal to an abnomal conformation when entering certain cells. When the immune system attacks cells that comprise abnormal prions, these cells allow both normal and abnormal prions to leak in the intercellular areas. Re-entry of these prions in neighboring cells multiplies the number of abnormal prions and keeps the immune reaction going. It is a chain reaction in which direct damage is caused by immune system attacks on infected cells and in which each attack of an infected cell multiplies the numbers of other infected cells.

## CJD: its interaction with electromagnetic waves




Epitopes (« pieces » of antigens which are directly recognizd by lymphocytes) form a discrete space. The above continuous representation is symbolic, but it makes it easier to understand what takes place.

Lymphocytes that recognize the PS epitope of a normal prion are normally eliminated by the thymus. The disease is caused when enough abnormal prions are present and a lymphocyte (LNS1 on the figure) recognizes the PNS1 antigen of the abnormal prion. It attacks infected cells and triggers the chain reaction yielding infection and destruction of cells.

The presence of an electromagnetic wave which alternates between low exposure and non-exposure at certain frequencies (such as GSM downlink which regularly switches frequency) allows supplementary lymphocytes LNS1-A to survive thymus selection during low exposure periods. These supplementary lymphocytes have their curves shifted right because they recognize the PS epitope in the absence of exposure (the affinity curve is above the recognition threshold 1 for epitope PS). The recognition of epitope PS by lymphocyte LNS1-1 during non-exposed periods normally yields elimination of LNS1-A by regulator T lymphocytes Treg. But since LNS1-A recognizes epitope PNS1 more strongly than epitope PS, it tends to first recognize PNS1, which yields its replication after a primary immune reaction. The replication speed of LNS1-A is then faster than the speed of its destruction by Treg lymphocytes, which feeds the chain reaction.

A stronger temporary exposure yields LNS1-B lymphocytes which affinity for PS is higher than their affinity for PNS1, so that they are more easily eliminated in a non-exposed situation by regulatory lymphocytes Treg and so that they do not cause a chain reaction. A stronger exposure does not significantly contribute to CJD.

A (low) substantially constant exposure (similar to UMTS downlink) prevents the recognition of epitope PNS1 by the supplementary T lymphocyte LNS1-A because the affinity of LNS1-A for PNS1 is less than the recognition threshold 2. Therefore this low but constant exposure stops the chain reaction.

**On this basis, it is thus logical that CJD reacts to TACS/GSM at low exposure without a stronger exposure causing a stronger chain reaction, and it is also logical that abnormal CJD deaths stop when a weak UMTS exposure occurs, in both cases for the same wavelengths to which the same lymphocytes react.**