

ARTICLE

Mad cow disease

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ABSTRACT The correct name for Mad Cow Disease is Bovine Spongiform Encephalopathy (BSE) and I will use this abbreviation throughout the text. BSE was first detected in England in 1985. Since then millions of cattle have been slaughtered either because they were infected or for precautionary measures. Payment for compensation to the farmers has cost the UK Government some Euro 6 billion. Although the UK has been the main site for BSE other countries have had BSE and all countries are forewarned. It seems certain that BSE has been transmitted to humans which has emphasised the necessity to protect the public from further infections. The many measures taken will be described. Basic research continues in an attempt to understand the science behind the advent of BSE but there remain many puzzling aspects. Some success has been achieved in identifying infected animals before the clinical symptoms appear. Naturally many professional people have been criticised for the spread of BSE and interesting lessons are being learnt from the links between politicians and scientists. There is little doubt that the standing of scientists in the public eye has been detrimentally affected by BSE.

Acta Biol Szeged 50(3-4):89-95 (2006)

KEY WORDS

BSE
Creutzfeldt-Jakob disease
prion
scrapie

It is many years since I first lectured on this subject which now has a mammoth bibliography and indeed publications reporting new developments appear daily. This, then, is merely an attempt to provide the background on which opinions about food safety should be based. After a brief introduction I will describe the epidemiology of BSE, then summarize the basic science and the gaps in our knowledge concerning the infectious agent; I will then summarize the precautions that have been taken and end with the impact of BSE on the reputation of the scientists and their interaction with the politicians. I should mention my credentials, or lack of them, for I have retired from experimental work for many years. My research interests were on the biosynthesis of animal proteins so I have for long been interested in the disease of sheep known as “scrapie” and this has led to an interest in “BSE”. We have many experts on the subject at University College London and I have endeavoured to keep abreast of what has become a major field of international research. I will not provide an extensive list of references but for background information the recent book edited by Prusiner (2004) should be referred to.

Epidemiology

The situation concerning the occurrence of the transmissible spongiform encephalopathies (TSE) in the early 1980’s is summarised in Table 1.

Scrapie in sheep is characterised by an irritation of the skin caused by damage to the neuronal cells, which causes the

animals to rub against a fence or wall in the later stages of the clinical condition, hence its name. While there are sporadic outbreaks of scrapie in many countries, including Europe and some Americas, it is no longer present in Australia or the USA, some breeds being very resistant. Scrapie could be transmitted to other sheep by intracranial injection of infected brains but more significant was the transmission to other species such as goats. After the first passage to a goat there is a long incubation period. This so-called “species barrier”, whereby the clinical symptoms of the disease take longer to emerge when the recipient is of a different species from that of the donor, is an important characteristic of the disease. The length of the incubation period is related to the evolutionary gap between donor and recipient. In 1961 it was shown that the infection could be transmitted to mice with a much shorter incubation period and more certain outcome and they, therefore, became a favourite test animal. The Syrian Golden Hamster has also proved to be a useful experimental animal particularly in the hands of Prusiner in San Francisco.

Table 1. The occurrence of transmissible dementias.

Species	Name of dementia
SHEEP	Scrapie
HUMANS	Kuru in New Guinea Creutzfeldt-Jakob Disease (CJD) Gertsman-Straussler-Scheinker (GSS) Fatal familial insomnia (FFI)
COWS	Bovine Spongiform Encephalopathy (BSE)

Accepted Dec 15, 2006

Table 2. The chronology of the BSE Epidemic.

Date	Event
1985 April	First clinical observation
1986 November	Disease identified as BSE
1987 December	Meat and Bone meal (MBM) implicated
1988 July	MBM feed banned
1988 August	All diseased cattle slaughtered
1989 February	Risk to humans "remote", Southwood Committee
1989 November	Offal banned for consumption
1992	BSE epidemic peaks at 36,681 cases in year
1995 November	Deaths of 3 young patients
1996 March	Suspected link between BSE and nvCJD
1996 July	Controls on slaughter of sheep

In humans Zigas and Gajdusek discovered a disease named "Kuru" among the Fore tribe in New Guinea. They showed that this was spread as a result of a cannibalistic feast involving ritual consumption of their dead relatives. The explanation of Kuru was that by chance someone suffering from CJD, which as I will explain is a nervous disease occurring sporadically all over the world, appeared among the Fore people. In view of the work with scrapie, Gajdusek inoculated chimpanzees and other primate species with suspensions of Kuru brains. The chimpanzees succumbed after about 1.5 years. Gajdusek was awarded a Nobel prize in 1976.

Until 1985 there were probably not many biochemists in the UK who were more than vaguely aware that sheep tended to suffer from scrapie. Moreover, since the disease had existed in the UK for some 200 years, and no one had suggested that it could be transmitted to humans, the subject was hardly a prominent one for research. Nevertheless, the British Government did continue to finance research at a modest level in order to unravel the nature of the infective agent, often described as a "slow virus", and similar work was pursued in other countries, especially the USA.

All this changed following the report in 1985 from a farm in Kent that they had a cow that had difficulty in walking and suffering from what is now called "Mad Cow Disease". After a delay of about a year before the full significance of the finding became apparent the situation changed dramatically. The disease became known as "Bovine Spongiform Encephalopathy" in view of the spongy appearance of sections of the brains of the dead animals.

The chronology of the epidemic of BSE

The number of cases of BSE rose dramatically and investigations to study the cause were set up. The critical dates in the unravelling of the epidemic are given in Table 2.

Suspicion centred on a high protein dietary supplement prepared from meat and bone meal (MBM) from the scrapings and offal of cattle and other animals that were not suitable for feeding to humans. MBM was particularly used for feeding to

high milk-yield dairy cows and it was a common practice in many countries. The traditional way of preparing MBM was to extract the fat with hot organic solvents to give a protein rich product and tallow. Steam treatment was then applied to recycle the organic solvents. Around 1980 an increase in fuel prices made the organic solvent extraction uneconomic and it was omitted. Because of the rise in the price of soya, MBM became more important as an animal feed.

Report of the Southwood Committee

As a result of the suspicion that BSE was caused by the feeding of MBM a ban on its use as a feed for cattle and sheep was rapidly instituted and in 1988 a working party under Professor Southwood was set up. They confirmed in 1989 the suspicion concerning MBM, and recommended that all affected cattle be slaughtered. At that time it was suspected that the trouble was caused by scrapie from infected sheep being transmitted to cattle via MBM but as I will show this is now questionable. As I have said, scrapie had been known for some 200 years and had never been shown to be transmitted to humans. On this basis the Southwood Committee reported that although transmission of BSE to humans was possible and could not be ruled out the chances of it happening were "remote". The British Government eagerly accepted this advice since otherwise the whole dairy industry was in danger. The politicians assured the public that beef was safe to eat especially since measures had been taken to ban the use of MBM. Soon after, the human consumption of brain, spinal cord and other offal was banned. Because the chance of transmission to humans was regarded as remote, it is clear that some of the recommended precautions were regarded merely as "window dressing" and were not fully implemented.

The number of cases of BSE rose dramatically and it is estimated that about 1 million cattle have been infected in the UK and of these about 750,000 were fed to humans in the period 1985-95. Although the incidence of BSE has been greatest in the UK there have been many cases in Switzerland and Portugal and a few cases in many other countries. In addition to the slaughter of animals showing symptoms there has been a ban on the human consumption of cattle over the age of 30 months since the symptoms of BSE mainly occur in older cattle. Such apparently healthy animals in affected herds have been slaughtered and many millions of cattle will have been slaughtered. The British Government has had to pay compensation to the farmers which has cost more than four billion pounds sterling. During this period some 25 million cattle will have been killed for food so the infected animals represent 3% of the total. The measures taken to control BSE in cattle have in general been successful and it is slowly dying out, but more slowly than originally predicted. The more recent figures are shown in Table 3.

The slow decline, with some cases occurring in cattle born after the complete ban on the feeding of MBM in 1996, is a

Table 3. Recent incidence of BSE in cattle in UK.

Year	Number of reported cases
1997	4309
1998	3178
1999	2254
2000	about 2000
2003 Jan.-May	81
2004 Jan-May	38

puzzle and has been attributed by some people to transmission from mother to calf before clinical symptoms have arisen but tests on 23,600 calves born to BSE-infected cattle have produced no evidence of this transmission.

Transmission of BSE to other species including man

After 1987 cases of BSE appeared in various other species of animals particularly various ungula in the zoos and also a few cases in cats. The incidence in cats caused particular concern for it was the first case of transmission to a meat eating animal rather than a herbivore. This strengthened the concern about the possible transmission to humans.

Creutzfeld-Jakob Disease

Apart from Kuru, three different kinds of CJD are recognised. First, there is "sporadic" which is the most common and affects individuals, usually in later life, for no known reason. Those affected reside worldwide and there has been no evidence of a recent epidemic in the UK. Second, "familial", this is an autosomal dominant disease which is rather rare. A good deal of genetic work has been done to understand this. Third, "iatrogenic" which has been caused by the administration of growth hormone preparations, made from a pool of 3000 cadaver brains in the USA, to children with problems of growth. There has been the emergence of CJD in children as a result of such treatment in several countries. The use of recombinant growth hormone has replaced pituitary extracts.

Later a further kind of CJD was to emerge as indicated in Table 4.

In August 1995 the first young person, then aged 19, died of CJD followed by several others who were aged about 29.

Table 4. Comparisons between sporadic and new variant CJD.

	Sporadic CJD	New variant CJD
Mean age at onset	65 years	26 years
Mean duration of illness	5 months	13 months
Presenting features	Rapidly progressive	Anxiety Depression Dementia
Codon 129 genotype	80% Met/Met	100% Met/Met

The brains of these patients had a pathology which closely resembled that of cattle with BSE and was quite different from those who had died of sporadic CJD. In March 1996 when 10 young people had died of CJD the British Government took the advice of their scientific advisory committee that we were witnessing the emergence of a "new variant" of CJD (nvCJD) where the duration of the illness at 13 months was long compared with sporadic CJD at 5 months. It seemed likely that this had arisen by the transmission of BSE as a result of the consumption of infected beef. The long incubation period of nvCJD, probably of at least 5-10 years, is based largely on the assumption that the greatest chance of people eating infected beef was between 1980 and 1988 when the ban on MBM was instituted. The best present estimates of the maximum number of cases likely to occur is 136,000 but this would imply an incubation period of 60 years which is unlikely but possible. Fortunately, so far the worst predictions have not been fulfilled since the total number of cases in the UK over the last 9 years is 147 but I will indicate later why this number may rise. Nearly all the cases are in the UK, but there have been 6 in France and 1 each in Italy, Canada, Ireland and the USA. Nearly all of the infected people had been in the UK between 1980 and 1996.

The possible origin of BSE

I have indicated that initially the cause of BSE was thought to be the transfer of scrapie to cattle. However, scrapie cannot be transferred to cattle by feeding BSE but BSE can be transferred to sheep by cranial injection. Unlike ungulates and feline species the symptoms of BSE in sheep are similar to scrapie so it has been suggested that perhaps BSE has been misdiagnosed for scrapie (Kao et al. 2002). However, there has been no marked increase in the number of cases of scrapie since 1980 and there is no incidence of the infection of wild sheep by BSE. BSE in cattle and nvCJD in humans bear the hallmarks of BSE rather than scrapie. The symptoms of mice infected with BSE and scrapie can be differentiated so such experiments are in hand but conclusions cannot yet be reached for there is a long incubation time. Thus it now seems more likely that BSE arose, probably on more than one occasion, as a result of an unusual metabolic event in a cow which led to the formation of a biologically active prion. This did not involve a mutation in the suspected infective agent but it may have been a mutation in another protein that influenced the formation of the agent. It has been conjectured that before the method of preparation of MBM was changed the infective agent would have been destroyed before being fed back to cattle but this view is controversial. It seems certain that the feeding of MBM was to blame but the reason is still not clear.

The nature of the infective agent

Following the discovery in 1961 that the scrapie agent could

Table 5. Polymorphism of prion protein at position 129.

The distribution in Caucasians is:	
Met/Met	37%
Met/Val	51%
Val/Val	12%

be transmitted to mice, rapid progress was made in determining the characteristics of the infective agent. The agent was resistant to formalin and seemed to have the size of a small virus, e.g. the picornavirus. However, the results of irradiation indicated that the scrapie agent was much smaller than any known virus and corresponded more closely to plant viroids which only contain RNA but unlike them the agent was resistant to nucleases. These were major findings and to the present day no one has been able to demonstrate that a nucleic acid is associated with the infectivity of the scrapie agent. Another important characteristic of the scrapie agent was its resistance to heat. Thus infectivity was retained even if the brain was boiled but infectivity was destroyed at very high temperatures.

Prusiner in California worked with Syrian Golden Hamster adapted scrapie where the incubation period was shorter than in the mouse and the amount of scrapie agent in clinical brain was at least tenfold greater. He came to the conclusion that the main component of the scrapie agent was a hydrophobic protein which polymerised easily. He coined the name "prion" (proteinaceous infective particle) and subsequently isolated protein from scrapie brain which he claimed was the major prion protein. Thus emerged the so-called "protein only hypothesis" for which Prusiner was in 1997 awarded a Nobel Prize. This was a very unexpected claim that the infectivity could be due merely to a protein for we had always implicated a nucleic acid with infectivity.

Another characteristic of the scrapie agent is that it is partially resistant to breakdown to amino acids by Proteinase K with no loss of infectivity. Proteinase K is a fungal enzyme much favoured by protein chemists. This finding has been linked to the fact that in the transmission of BSE the active agent is not destroyed in the gastrointestinal track and rumen of sheep and cattle and that the agent is resistant to the proteolytic enzymes therein.

The structure of prions

I realise that the audience may have only a limited knowledge of biochemistry so I will speak in rather general terms. Proteins consist of chains of amino acids linked together. There are 20 different amino acids in the chain. The chains are twisted and coiled (tertiary structure) similar to that of the wire in the traditional electric light bulb. The order of the different amino acids in the chains (the primary structure) determines the biological properties of the protein. Prusiner determined the primary structure of his prion, designated

PrP^{sc}. A protein with the same primary structure was found to be present in many different tissues of the body, designated PrP^c. The function of this protein is unknown but seems to be harmless. The difference between PrP^c and PrP^{sc} resides in a difference in the tertiary structure. Experimentally it can be shown that the infective process involves the conversion of PrP^c to PrP^{sc}. A crucial experiment in support of the "protein only hypothesis" would be to demonstrate the conversion of PrP^c to an infectious protein in the test tube. This has not been possible. Proteins with similar properties to the animal prions have been found in yeast and much effort is being devoted to understand the conversion process using yeast as a model.

I should emphasize that there are many aspects of the basic science and pathology of the TSE's that are as yet not understood (May et al. 2004).

The effect of mutations and polymorphism of prions

A lot of work has been done on the effect of mutations in PrP^c on the incidence of CJD. There is no doubt that familial CJD is associated with certain mutations and in sporadic CJD they make some difference to susceptibility. Of more significance is the effect of polymorphisms in PrP^c at position 129 of the protein as mentioned before and shown in Table 5.

Since virtually all the cases of nvCJD, so far found except one, are homozygous for Met-129 it suggests that such polymorphisms have an influence on the ease of conversion of PrP^c to PrP^{sc}. If this be so then it may be that those with Met/Val will emerge with nvCJD after a longer incubation period.

Diagnosis of BSE and the preclinical identification of PrP^{sc}

There is now an enormous effort being made internationally to find a reliable method for diagnosing the presence of PrP^{sc} in organs from both cattle, sheep and humans. Since such methods will provide considerable profits to the originator it is not always easy to gain access to the details or the results. It seems highly likely that one or more reliable methods will be demonstrated in the near future. While it is possible to prepare an antibody to prions it has proved very difficult to obtain one that is specific for PrP^{sc}. PrP^{sc} can be differentiated from PrP^c by dependence on the resistance of the prion to Proteinase K, so that antibody can be used to detect the residue. The European Union now tests all cattle over 24 months old using a method devised by the Zurich group. The Japanese test all cattle irrespective of age. The latest report I have is that since 1987 1200 French cattle have tested positive. It seems that the methods are useful in detecting animals with the disease shortly before the clinical symptoms appear. The antibodies used in these tests are very expensive. A similar method is used to detect the presence of PrP^{sc} in appendices and tonsils of patients. In the latest report there were 3 positives out of 12,500 specimens tested (Hilton et al. 2004). If this ratio

were repeated across the UK then 3,800 people would carry the infective prion. So far only 3 infected Britons have died in 2004 with 5 still alive.

Precautionary Measures

Although the vast majority of cases of both BSE in cattle and nvCJD in humans have occurred in the UK, cases of BSE infected cattle have appeared in other countries, such as France, Switzerland and Portugal. The USA and Canada hoped that they were free of BSE but there have now been reports of a very few cases in those countries. One worry has been that in many countries farmers who detect that an animal has difficulty in walking, called downers, send them off to the slaughter house so the number of cases may be more than those recorded. The conclusion must be that the situation calls for international collaboration.

Action on the farm and at slaughter houses

There seems little doubt that the original source of infection was MBM and so care must be taken that imports of this feed must be checked. One mistake in the UK was to allow the feed to continue to be fed to chickens and pigs until 1996 long after it had been banned for feeding to cattle. It seems likely that some food was diverted to cattle and no doubt the vessels used were contaminated. Since the symptoms of BSE usually appear only in older cows, there has in the UK, been a ban for 8 years on allowing cattle over the age of 30 months to enter the food chain. Some 700,000 cattle a year are now being slaughtered in the UK as a result of this ban. It is claimed by the farmers that the ban should be lifted now that infected cattle can be detected before the clinical signs are apparent. Others argue that the detection methods are not yet sufficiently proven. Ideally the natural history of every cow should be recorded so that if an animal is detected with BSE it's movement will be known and precautions can be taken. The most likely site of contamination is the spinal cord and so measures must be supervised to ensure the careful removal of this tissue in the slaughter house. Such procedures are more difficult in sheep but to be on the safe side sheeps' brains re excluded from the food chain in the UK and France.

Use of surgical instruments

As mentioned already the infective agent proved resistant to normal methods of sterilization. Charles Weissmann has reported on experiments that he is undertaking to determine whether surgical instruments could account for the transmission of infectivity (Flechsig et al. 2001). He has shown that a stainless steel instrument used in human brain surgery for a patient suffering from CJD can transmit the disease to another patient even though the instrument was sterilized with formaldehyde. Working with mice it seems that a mere 5 min exposure to an infected brain is enough to infect the instru-

ment. Formaldehyde sterilization merely causes the protein to become cross-linked. In spite of this, and the apparent tight binding, the protein can create an infective agent probably by causing the conversion of PrP^c to PrP^{sc}. It has been recommended that disposable instruments should be used in brain surgery and for the removal of tonsils. Concern is now being expressed about dental surgery involving the removal of infected roots. While formaldehyde is ineffective, the agent is thought to be inactivated either by heating at 132°C for 4.5 h or by the use of M-NaOH. Dental surgeons are being advised to use 4% hypochlorite. The fear is that there may be a bank of human carriers passing on the infection to others.

Blood transfusion

Although the neuropathologies do not have an immunological pathogenesis there is evidence that the immune system is important for transporting infection from the periphery to the brain (Aguzzi 2001). Thus mature B cells are essential for prion accumulation in the spleen. Infectivity in the spleen is associated with B and T lymphocytes and with the stromal fraction which contains follicular dendritic cells (FDCs). It seems that the infective agent is synthesized in mature FDCs and is transferred to splenic lymphocytes which are in intimate contact with FDCs. These results have given cause for concern about blood banks. Recently PrP^{sc} was found in the spleen of a patient who died from an unrelated cause but had received blood from a donor who later developed nvCJD. In this case the polymorphism at position 129 differed from that of the other patients who died from nvCJD. The content of white cells from blood can be reduced before administration (leucodepletion), but this is difficult and expensive. Blood taken from donors in the U.K. will not in future be pooled. (In the USA people who have lived in the UK for more than 6 months are excluded as blood donors.) In all 15 blood donors in the UK have gone on to develop nvCJD. At least 48 people have received possibly contaminated blood and 17 are still alive. One person appears to have died as a result of blood transfusion. People who have received blood are being eliminated as blood donors.

Blood products

It has been traditional to prepare essential products from pooled blood. Very large quantities of Serum Albumin are used throughout the world. As a result of BSE such products are banned for export from the UK. Fetal calf serum is also used in the preparation of many vaccines and this has also caused concern. Another product, Gelatin, is used extensively in medical preparations and is prepared from cattle bones. Tests have indicated that the prions would be destroyed during preparation but it is also no longer sourced from the UK (Grobben et al. 2004).

Possible therapies

So far all therapies have failed. The Koreans in December 2003 announced the birth of four cloned calves lacking prions. Potentially this represents a way of ensuring that cattle do not have BSE but the expense of producing herds of cattle lacking prions is formidable. Moreover, we do not yet know the biological properties of PrP^c.

Interactions between journalists, politicians and scientists

I have explained some of the many scientific surprises that have arisen as a result of the BSE crisis that has cost the UK government more than 4 billion pounds sterling alone in compensation to farmers and caused much disquiet in agriculture. The public were surprised that meat in the form of MBM should have been fed to herbivorous cattle and even more surprised that sheep with scrapie entered the food chain. It seems that not even the farmers knew the origin of MBM, merely that it was a high protein supplement fed to dairy cows. It is good if we have learnt to be more enquiring. The scientists have also been criticised for misleading the public in saying that the transfer of BSE to humans was only a remote possibility. There is no doubt that the reputation of scientists has been badly affected by the BSE saga and we know that the story is not at an end. The experience with BSE has increased the resistance of the public to biotechnology in general.

Journalists rightly reflect the concerns of the public and seek the views of the scientists. They understandably relish straight answers. If a scientist is asked "is it safe to eat beef?" the scientist is unlikely to give a straight answer, Yes or No, but will wish to introduce qualifications. The matter of risk factors may well be referred to. This is an area little understood by the public for the risk they are willing to take depends on the pleasure they derive from the activity. The scientists may mention that while it is very regrettable that some 150 people have died from infection with the BSE agent the numbers are very small compared to the once threatened disaster and the number of people killed on the road. There must be plenty of people who have declined to eat beef but continue to smoke cigarettes.

Lord Waldegrave, a former Cabinet Minister in the British Government, has recently written an article "When scientists advise politicians" and several times refers to the problem of BSE. He correctly emphasises the provisional nature of scientific conclusions and being aware that scientists change their minds, as I have indicated in this article. It is important too, he writes, that Ministers should listen to dissident scientists who, although they may be bad scientists, happen to have got something right. It is not surprising that politicians, having consulted the scientists, have difficulty in conveying the answers to the public, especially when the outlook for a major industry may be affected by their words.

I suspect that the public thinks that the scientists under-

stand the basic science underlying the BSE crisis and that their hedged answers to questions are a cover up. I hope that I have shown that this is not so and that the scientists involved in research on BSE often are dealing with the world of the unknown. For such reasons alone I hesitate to apportion blame among those involved, both scientists and politicians. For more details about this matter the Phillips Report (www.bseinqury.gov.uk/report) should be consulted

Acknowledgements

This study was presented on the NATO Advanced Research Workshop on "Food Safety and Security" held between 13-15 September, 2004, at Lake Issykkul, Kyrgyzstan. The workshop was funded by NATO. Co-directors were Prof. Dr. A. Aldashev, National Academy of Sciences of the Kyrgyz Republic, and Prof. Dr. L. Erdei, University of Szeged, Szeged, Hungary.

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Obituary

Peter N Campbell (1921-2005)

Professor Peter N. Campbell passed away on February 7 2005. He was Chairman of the Department of Biochemistry at the University of Leeds, 1967-1975, and Director of the Courtauld Institute of Biochemistry, Middlesex Hospital Medical School, 1976-1987. Since retiring he has been located at University College London. There he was concerned with the 'Scientific Apparatus Recycling Scheme' for FEBS and with the UNESCO 'Molecular Cell Biology Network' in support of biochemists in Africa. He also worked for the Association of Researchers in Medicine & Science. He played major roles in the Federation of European Biochemical Societies (FEBS). Peter Campbell is the author of a large number of scientific papers. He has been Editor of the *Essays in Biochemistry* series (1965-1985) and Editor-in-Chief, *Biotechnology* and

Applied Biochemistry from 1981 to 1995. He also edited or authored a number of books, including the Oxford Dictionary of Biochemistry and Molecular Biology.

Peter Campbell travelled intensively in the name of several organizations. His activities as a scientific voyager are summarised in his book "A Biochemical Foreign Correspondent- Stories and Impressions from Around the World" which appeared in 2003. We, the participants in the NATO Advanced

Research Workshop on "Food Safety and Security" held between 13-15 September, 2004, at Lake Issykkul, Kyrgyzstan, have had the luck to enjoy his brilliant personality. We all deeply regret the loss of a very valid scientist and teacher.

Extracted from the obituary written by Angelo Azzi, Secretary General of SFRR International and Chairman IUBMB Publication Committee and modified by László Erdei.