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医薬品　研究報告 調査報告書

識別番号・報告回数			報告日	第一報入手日	新医薬品等の区分	厚生労働省処理欄
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販売名(企業名)	－					

研究報告の概要	使用上の注意記載状況・その他参考事項等
CJD伝播管理の専門家委員会(CJDIP)は、汚染された血漿由来製剤による変異型クロイツフェルト・ヤコブ病(vCJD)のリスク管理に関する勧告を発表した。現在まで英国で9人の供血者がvCJDを発病したことが知られており、これら供血者より23件の血漿供与が行われた。その血漿は第Ⅷ因子、第Ⅸ因子、アンチトロンビン、静注用免疫グロブリン、アルブミン、筋注用ヒト免疫グロブリンおよび抗Dグロブリンの製造に用いられたが、これまでのところ受血者にvCJDが伝染したとの記録はない。2004年7月に2例目の輸血によるvCJD感染疑い例が確認され、血液による感染の懸念が大きくなっている。供血後vCJD発病者由来の血漿由来製剤受血者の一部については、他者にリスクをもたらす可能性があることから公衆衛生予防措置(血液や臓器、組織などを供与しないこと、外科手術、歯科治療が必要な場合は医師に知らせることなど)が必要と勧告した。 また、公衆衛生予防措置を要する可能性に応じ、汚染された血漿由来製剤のバッチを「高、中、低」に分類すると共に、vCJDリスクを有する患者(出血性疾患、原発性免疫不全などの血漿由来製剤による治療を定期的に受ける人)には、講じる必要のある予防措置を通知する。なお、筋注用免疫グロブリン(Rh陰性の妊娠女性に投与される抗Dグロブリン、A型肝炎予防のための人免疫グロブリンなど)を投与されたことのある人々はリスクがないと考えられ、措置を講じる必要がないとしている。	

報告企業の意見	今後の対応
異常プリオン蛋白を含む可能性のある原料血漿由来の製剤からのvCJD伝播の報告はないが、供血後にvCJDを発症した人由来の製剤投与を受けた人の一部については血液等の提供をしないようにする等を勧告している。 なお、プリオン蛋白については、血漿分画製剤の製造工程で除去できるとの考え方がある。	今後もvCJDに関連する情報収集に努めていく。

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News

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Next update due: **30 September 2004**

Variant Creutzfeldt-Jakob disease and plasma products: implementation of public health precautions in the UK

The CJD Incidents Panel (CJDIP), an expert committee set up to advise on the management of "incidents" of potential transmission of CJD between patients, has issued recommendations on the management of variant Creutzfeldt-Jakob disease (vCJD) risk from implicated plasma products.

To date, nine UK plasma donors are known to have developed vCJD. Collectively, they made 23 plasma donations. The donated plasma was used to manufacture factor VIII, factor IX, antithrombin, intravenous immunoglobulin G, albumin, intramuscular human normal immunoglobulin, and anti-D.

The potential risk of vCJD infection following treatment with any implicated plasma products, on top of the risk from dietary exposure to the BSE agent, is very uncertain. So far, there are no recorded instances of vCJD being spread through surgery, nor have there been any cases among recipients of plasma products sourced from individuals who later developed vCJD. In December 2003, the death of a person from vCJD some years after receiving a blood transfusion from a donor who themselves died of vCJD, was announced. In July 2004 a second probable case of transfusion-associated vCJD infection was identified. These two events have increased concern about the potential infectivity of blood and plasma products

Public health precautions against vCJD
The CJDIP now recommends that certain special public health precautions need to be taken for some recipients of UK sourced plasma products that were manufactured using donations from individuals who subsequently developed vCJD. This is in order to reduce any possible risk of iatrogenic transmission of vCJD (*ie*, disease or infection acquired via a healthcare setting).

The CJDIP has used a vCJD blood risk assessment <http://www.dnv.com/consulting/news_consulting/RiskofInfectionfromvariantCJDinBlood.asp> together with information on how the particular batches of plasma products were manufactured, to assess the potential levels of infection that patients were exposed to.

The CJDIP advises certain special public health precautions need to be taken for recipients of UK sourced plasma products who have been exposed to a 1% or greater potential additional risk of vCJD infection as these patients could pose a risk to others in defined circumstances. These "at risk" patients are asked:

- not to donate blood, organs or tissues, and
- to inform their clinician if they need medical, surgical or dental treatment, so that infection control precautions can be taken <http://www.advisorybodies.doh.gov.uk/acdp/tseguidance/> to reduce any possible risk of spreading vCJD, and to consider informing their family, in case the patient needs emergency surgery in the future.

The CJDIP has categorised each batch of implicated plasma products according to the likelihood that special public health precautions need to be taken as follows:

- **High:** the amount of potential infectivity in product batches was high enough to warrant special public health precautions following the administration of a very small dose. These batches should be traced, the recipients advised of their exposure and asked to take special public health precautions.

- **Medium:** substantial quantities of the material in question would need to have been administered to warrant special public health precautions. Efforts should be made to trace these batches and assess the additional risk to individual recipients to determine if special precautions should be taken.

- **Low:** the potential additional risk to recipients is considered negligible. These batches do not need to be traced and the individual recipients do not need to be informed.

This categorisation is based on very cautious assumptions, and the uncertainties underlying the assessment of 'risk' are great. The CJDIP guidance is to limit any possible iatrogenic human-to-human transmission ofvCJD. It should **NOT** be interpreted as an estimate of an individual patient's additional risk of developing vCJD, which is uncertain, and likely to be very low.

Informing patients

The patients who may be affected include some patients with bleeding disorders, primary immunodeficiency (PID), and with other conditions, who may include, for example, patients with secondary immunodeficiencies, certain neurological and autoimmune conditions, plasma exchange recipients, patients with severe burns, and those with some other conditions requiring critical care.

Patients who are 'at-risk' of vCJD for public health purposes are being contacted by their doctors and informed of the precautions they will need to take.

The **MAIN MESSAGES** for patients are:

1. **ALL** people treated regularly with plasma products , *eg*, patients with bleeding disorders or primary immunodeficiency (PID) are being contacted by the specialist doctor responsible for their care.

2. Hospitals will trace other people who received implicated plasma products , *eg*, for emergency treatment of severe burns) and arrange to have their exposure assessed. Individuals will be contacted **ONLY IF THEIR EXPOSURE IS IDENTIFIED AND WARRANTS FURTHER ACTION.** The process of traceback and assessment may take a number of weeks, and, where treatment took place a number of years ago, it may not be possible to find out who received the implicated plasma products.

3. People who have received intramuscular immunoglobulins (eg anti-D for Rhesus negative pregnant women; human normal immunoglobulin for travel prophylaxis for hepatitis A) are **NOT** considered to be 'at-risk' and no action is needed.

Sources for Additional information

The Health Protection Agency's (HPA) CJD section at the Communicable Disease Surveillance Centre (Colindale) is co-ordinating the patient notification in England, Wales, and Northern Ireland. The Scottish Centre for Infection and Environmental Health (SCIEH) is co-ordinating this notification in Scotland. Background information about vCJD with useful links is available from their websites:

HPA: <http://www.hpa.org.uk/infections/topics_az/cjd/menu.htm>.

SCIEH: <http://www.show.scot.nhs.uk/scieh>.

別紙様式第２－１

医薬品　研究報告 調査報告書

識別番号・報告回数			報告日	第一報入手日	新医薬品等の区分	厚生労働省処理欄
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研究報告の概要

シカおよびヘラジカなどの動物の伝達性海綿状脳症（TSE）は、公衆衛生上の潜在的脅威となる可能性も考えられる慢性消耗病（CWD）で、米国内に多く存在する。コロラド州北東部およびワイオミング州南東部の隣接地域では、この数十年にわたってCWDの流行が報告されているが、人間における症例は発見されていない。
一方、BSEに汚染された牛製品の摂取を介して人間にBSE病原体が感染し、変異型CJDを引き起こすことを示唆する強力な疫学的証拠および研究室レベルの証拠が存在するが、英国におけるBSE感染牛が100万頭以上と推定されることから、これまでに報告されている変異型CJD例数は比較的少なく、「種間障壁」によって人間における変異型CJD発病が大きく阻止されてはいるものの、必ずしもその阻止が完全でないことを示唆している。変異型CJDは、比較的若い人々に発現することに加え、特定の遺伝子プロフィールを持つ一部の人々に限られるように推察される。一般集団の約3分の1がこのBSEにかかりやすいカテゴリに属することを、複数の研究が示唆している。
CDCはCWDの人間への感染可能性を監視するため、シカおよびヘラジカの肉を食したハンターにおけるCJD群発例を調査してきたが、2001年発表の調査の一つには、野生のシカやヘラジカの肉を食べた28歳から30歳の大変若いCJD患者3名についての調査が含まれていた。この3例に関する調査では、CWDとこれらの患者におけるCJDとの因果関係に関して確証は得られなかった。国立衛生研究所のある研究室で行われた実験では、ヒトプリオンタンパク質がCWD関連プリオンによって病原性プリオンタンパク質に変換されることが証明されている。この知見およびBSEの人間への感染は、人間がCWD病原体に感染する可能性を示唆している。
CDCは、変異型CJDおよび人間におけるCWDといった新たな疾患の発現を調べるため、これからも引き続き関係機関と協力して監視活動を実施、強化していく予定である。

報告企業の意見	今後の対応
CWD、vCJDの病原物質とされているプリオン蛋白については、有用な検査方法がないため、採血時の問診、Look Backシステムに頼らざるを得ないが、血漿分画製剤の製造工程で除去できるとの考え方がある。	今後もCWD、vCJDの発生状況、プリオン蛋白除去等の安全対策に関する情報収集に努めていく。

使用上の注意記載状況・その他参考事項等

ProMED情報20040409-0060により入手したCWD、vCJD情報のもと文献。

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CDC's Role in Monitoring for Transmissible Spongiform Encephalopathies

Statement of Julie L. Gerberding, M.D., M.P.H.
Director Centers for Disease Control and Prevention
U.S. Department of Health and Human Services

Before the
Committee on Appropriations
U.S. Senate
February 24, 2004

Good morning, Mr. Chairman and Members of the Committee. I am Dr. Julie L. Gerberding, Director of the Centers for Disease Control and Prevention (CDC). Thank you for the invitation to participate in this timely hearing on a critical public health issue: bovine spongiform encephalopathy (BSE) and its potential impact on human health. In my testimony today, I will describe CDC's surveillance activities for Creutzfeldt-Jakob disease (CJD), variant CJD which is believed to have resulted from consumption of BSE-contaminated cattle products, and the possible occurrence of chronic wasting disease in humans.

Transmissible Spongiform Encephalopathies
Bovine Spongiform Encephalopathy (BSE)–Cattle BSE is a progressive, fatal neurologic disorder of cattle and is classified as one of the transmissible spongiform encephalopathies (TSEs), a group of diseases of animals and humans believed to be caused by abnormally folded proteins called prions. BSE was first identified in the United Kingdom in 1986 where it had been causing a large outbreak among cattle. Although controversy exists about the origin of BSE, there is general agreement among scientists that feeding cattle meat-and-bone meal produced from rendered carcasses of BSE-infected cattle is the primary route of BSE spread and greatly amplified the BSE outbreak. Since 1986, BSE cases have been identified in 20 European countries, Japan, Israel, Canada, and in December 2003 in an imported cow in the U.S. state of Washington.

Chronic Wasting Disease (CWD)–Deer and Elk Another animal TSE that is prevalent in the United States which could potentially pose a public health threat is chronic wasting disease (CWD) of deer and elk. The endemic occurrence of CWD for several decades has been reported in a contiguous area in northeastern Colorado and southeastern Wyoming. Since 2000, new foci of CWD among free ranging deer and elk were reported in Illinois, Nebraska, New Mexico, South Dakota, Utah, Wisconsin, and non-endemic portions of Colorado and Wyoming. No human cases of CWD have been identified.

Creutzfeldt-Jakob Disease (CJD)–Humans
In contrast, strong epidemiologic and laboratory evidence indicates that the BSE agent has been transmitted to humans via consumption of BSE-contaminated cattle products, causing a newly recognized variant form of CJD. The occurrence of the first 10 variant CJD cases and their possible causal link with BSE was first announced in the United Kingdom in 1996, about 9 years after the identification of BSE. Since then, as of February 2004, a total of 156 variant CJD cases have been identified worldwide; 146 of these were reported from the United Kingdom, 6 from France, and 1 each from Canada, Ireland, Italy, and the United States. Except for the variant CJD cases in France and Italy, all the other cases, including the cases in Canada and the United States, are believed to have acquired variant CJD as a result of exposure to BSE in the United Kingdom. The number of variant CJD cases reported to date is relatively small compared with an estimated 1 million or more cattle infected with BSE in the United Kingdom alone, indicating that a "species barrier" provides substantial but incomplete protection to humans against development of variant CJD.

The variant form differs from what is termed the "classic form" of CJD which also occurs in humans. Classic CJD is the most common TSE in the human population. It primarily occurs in a sporadic form with no recognizable pattern of transmission but also as a familial disease resulting from inheritable defective genes. It has also been associated with receipt of contaminated human growth hormone and dura mater and corneal grafts and with exposure to contaminated neurosurgical equipment. The variant form differs from the classic form of CJD also in the age distribution of affected patients, duration of illness, clinical manifestation and course, and most importantly the pathologic lesions in the brain and biochemical characteristics of the agent. Characteristically, variant CJD patients are unusually young with over half of the patients dying before 30 years of age and over 98% before 55 years of age. In contrast, classic CJD commonly develops in patients in their sixth and seventh decade of life, and about 90% of classic CJD patients die at 55 years of age or older. Patients with variant CJD often have prominent behavioral or psychiatric manifestations and painful sensory symptoms, with neurologic signs such as involuntary movements and gait disturbances being delayed for several months after illness onset. The typical electroencephalographic findings observed in classic CJD patients are absent in patients with variant CJD. A characteristic MRI finding, demonstrated in over 75% of variant CJD patients, is considered to be very supportive of a variant CJD diagnosis in the appropriate clinical context.

In addition to selectively affecting younger people, the occurrence of variant CJD seems to be limited to a segment of the general population that have a particular genetic profile. Studies have indicated that only about one-third of the general population falls under this category of BSE susceptibility. Although the diagnosis of variant CJD or classic CJD can often be strongly suspected on the basis of clinical manifestations and radiologic and electroencephalographic studies, confirmatory diagnosis of both of these diseases requires testing of brain tissue obtained after a brain autopsy or biopsy.

CDC Role
CDC's primary role related to BSE is to monitor for the occurrence of variant CJD possibly resulting from exposure to BSE within the United States or due to travel to other countries such as the United Kingdom where more cases of BSE have been identified. CDC also investigates CJD cases or clusters in which a plausible external source of infection is

suspected. A foundation of CDC's disease surveillance and outbreak investigation activities is its relationship with and support of state and local health departments that are often the first to encounter newly emerging human diseases or changes in the epidemiology of recognized human diseases. These relationships complement those that CDC maintains with health care providers and other healthcare institutions and are designed to maximize the likelihood that a sentinel event will be detected, whether in an expected or an unexpected location.

CJD Surveillance

After the identification of variant CJD in the United Kingdom in 1996, CDC enhanced its CJD surveillance activities to monitor the occurrence of the disease in the United States and monitor the emergence of other new forms of the disease among the U.S. population. CDC utilizes multiple surveillance mechanisms to track the occurrence of human TSEs. These mechanisms include: 1) periodic review of national mortality database; 2) investigation of individuals who have died from CJD and are under 55 years of age; 3) establishment and support of the National Prion Disease Pathology Surveillance Center; 4) active collaborative surveillance of TSEs in special populations (e.g., recipients of human growth hormone); and 5) investigation of patients suspected with a TSE reported to CDC by health care providers (e.g., suspected variant CJD cases).

One of the major ways by which CDC monitors the occurrence of CJD is through periodic review of the national multiple cause-of-death data compiled by the National Center for Health Statistics. Review of death certificate data enables CDC to more efficiently monitor any changes in the epidemiology of CJD in the United States. Because patients with CJD invariably die of the disease and usually within 1 year of illness onset, death certificate surveillance of CJD can be used to estimate the incidence of the disease. Through analysis of death certificate data, CDC determined that the rate and characteristics of CJD deaths identified in the United States during 1979 through 2001 were stable over time and were not at an unexpected level. During the 23-year surveillance period, 5473 deaths attributed to CJD were identified in the United States. The average annual age adjusted CJD death rate during 1979-2001 was 1.06 cases per million population. The CJD death rate has been relatively stable during the surveillance period particularly after 1985. Most cases of CJD in the United States occur in persons in their sixth and seventh decade of life, and unlike in the United Kingdom, where the occurrence of variant CJD resulted in an appreciable increased occurrence of the disease in persons less than 30 years of age, no such increase has been identified in the United States.

To effectively monitor the possible occurrence of variant CJD, CDC routinely investigates the illnesses of suspected or physician-diagnosed CJD patients under 55 years of age by reviewing their clinical and pathologic records. This investigation is conducted in collaboration with state and local health departments and takes advantage of the differences in age distribution of variant CJD from that of classic CJD patients by targeting the age group known to be at an increased risk of BSE transmission. Patients who have died of CJD before 55 years of age are identified from multiple sources, including review of mortality data and reports by clinicians and state and local health departments. To date, CDC has completed review of the clinical or pathologic records of 185 CJD patients under 55 years of age who died since 1994, and none of the patients have evidence of variant CJD.

In addition, during 1996 and 1997, in collaboration with the American Association of Neuropathologists, CDC established the National Prion Disease Pathology Surveillance Center which is located at the Division of Neuropathology of Case Western Reserve University in Cleveland, Ohio. CDC continues to support and monitor the activities of this pathology center. In fiscal year 2003, CDC provided approximately $2.5 million to support the activities of the center. The pathology surveillance center makes available state-of-the-art human TSE diagnostic services to U.S. physicians and state and local health departments. It periodically contacts neurologists and pathologists across the United States informing them about the need to arrange for autopsy of suspected CJD cases and urging them to utilize the free services provided by the center. During its existence since 1996, the center has examined brain tissues from over 1200 patients but did not identify any domestically acquired variant CJD case in the United States. Slightly over half of the cases examined by the center are confirmed with a TSE, most of them with sporadic CJD. Through analysis of brain tissues of patients suspected with TSEs, the center continues to define and characterize the normal background occurrence of human TSEs to help recognize any emerging forms of the disease. It specifically searches for the occurrence of variant CJD and any other form of TSE with potential link with CWD of deer and elk. In addition, the pathology surveillance center with support from CDC and the Food and Drug Administration (FDA) conducts applied laboratory research by developing appropriate animal models to characterize the susceptibility of humans to infection by the CWD agent.

CDC's multi-pronged surveillance mechanisms have not identified domestically acquired variant CJD in the United States. In 2002, CDC reported the occurrence of the first variant CJD case in the United States in a 22-year-old British woman who is a resident of Florida. The patient was born in the United Kingdom and raised there during the BSE outbreak and is believed to have acquired variant CJD from consumption of BSE-infected cattle products in her native country. This patient is still alive, and is receiving medical care in Florida.

The major challenge to CDC's national surveillance of CJD and other emerging forms of the diseases is lack of autopsy brain specimens in some cases of CJD. Because availability of brain specimens permits confirmation of the diagnosis and allows for effective monitoring of newly emerging forms of the disease such as variant CJD, CDC places a high priority in increasing the number of autopsies in suspected and physician-diagnosed patients with TSEs. To understand the problems associated with lack of autopsies, CDC recently collaborated with the California State Department of Health Services and the New York State Department of Health on a survey of neurologists and pathologists to determine the barriers for autopsy performance in CJD patients. The survey findings indicate that cost of autopsy, infection control concerns by pathologists, and lack of family consent may be some of the barriers to autopsy performance. CDC is collaborating with state and local health departments, the National Prion Disease Pathology Surveillance Center, and the national Creutzfeldt-Jakob Disease Foundation to address these autopsy barriers and increase the number of brain specimens available for effective surveillance of emerging forms of human TSEs. The increasing number of brain specimens referred to the National Prion Disease Pathology Surveillance Center during the last several years may indicate some improvement in the number of suspected CJD cases that are being autopsied. However, some challenges still remain.

CWD Investigations

To monitor the possible transmission of CWD to humans, CDC has investigated several CJD case clusters among hunters who consumed deer and elk meat. One such investigation, published in 2001, included an investigation of three unusually young patients aged 28 to 30 years of age with CJD who consumed deer or elk meat they harvested from the wild or provided by a family member. Our investigation of the three cases did not provide any convincing evidence for a causal link between CWD and the CJD illness in the patients. Recently, CDC also investigated the illnesses of three hunters who participated in wild game feasts and subsequently died of neurologic illnesses. The wild game feasts were regularly held in a cabin in Wisconsin and were hosted by one of the decedents who was a lifelong

hunter and consumed venison frequently. Clinical and pathologic investigation of the illnesses of the three patients indicated that two of the patients did not die of a TSE illness. The one patient confirmed with CJD had an illness consistent with other classic CJD patients with no history of venison consumption. In addition, this patient was unlikely to have consumed CWD-infected venison because venison and other game that was served during the feasts did not originate from known CWD-endemic areas, and the man participated in the feasts only once. Although CDC's investigations to date have not identified strong evidence for a causal link between CWD and human illness, the conversion of human prion protein to the disease causing form by CWD-associated prions has been demonstrated in a cell-free experimental study performed at a National Institute of Health laboratory. This finding and the transmission of BSE to humans indicate that humans may not be completely protected from infection by the CWD agent. To monitor for this possibility, CDC is collaborating with the Wyoming Department of Health and the Colorado Department of Public Health and Environment to determine any unusual increase in the number of human TSEs among hunters who hunted deer and elk in areas where CWD was known to be endemic for several decades.

Acquired TSEs

In collaboration with other government and private institutions, CDC also conducts several follow up studies of special populations deemed to be at a potentially increased risk of developing acquired TSEs. One of these studies was initiated in the 1980s to monitor the occurrence of CJD among patients known to have received cadaveric-derived human growth hormone. Before 1985, this hormone was derived from pituitary glands obtained from human cadavers and was inadvertently contaminated when pituitary glands from donors who had died of CJD were included in the batch processing. As of February 2004, a total of 26 human growth hormone-associated CJD cases have been identified among about 8000 recipients included in the follow-up study. A second follow-up study that is being conducted in collaboration with the American Red Cross relates to the possible transmission of CJD to patients known to have received blood components obtained from donors who, subsequent to their donation, died of CJD. As of February 2004, 331 recipients of blood components obtained from 25 "CJD donors" were enrolled in the follow-up study. Although some of the recipients died of the underlying condition that led them to receive blood, none of the 116 recipients who were followed for at least 5 years developed CJD. The median follow-up time was 9½ years, and in some cases, the follow up lasted for over 20 years.

Conclusion

In summary, two animal TSEs are believed to potentially pose a risk to the U.S. population. One of these, BSE of cattle with documented evidence of transmission to humans, has now been detected in Canada and the United States. Although the measures taken by the U.S. Department of Agriculture are expected to reduce the risk of exposure of the U.S. population to BSE, there is a possibility that domestically acquired variant CJD may appear in the United States. However, this possibility is believed to be extremely small. Another animal TSE that could pose a risk to the U.S. population is CWD of deer and elk. Although the occurrence of CWD seems to be geographically limited to certain areas of the United States, recent evidence suggests that new foci of infection may have emerged. An ongoing concern exists about the possibility that human exposure to the CWD agent may increase with the increasing spread of the disease to new geographic areas. CDC will continue working with our partners to conduct and enhance surveillance mechanisms to monitor the occurrence of classic CJD in the United States and to monitor the occurrence of emerging forms of the disease, including variant CJD and CWD in humans.

I will be happy to respond to any questions you may have.

For more information about this topic contact:
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