VIRGINIA
EPIDEMIOLOGY BULLETIN

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Transmissible Spongiform Encephalopathies (TSEs)

A little over one year ago, on December 25, 2003, the US Department of Agriculture (USDA) confirmed the diagnosis of bovine spongiform encephalopathy (BSE, or "mad cow disease") in a single dairy cow in Washington state.[1] As a result, the USDA expanded and intensified its national testing program for BSE. From June 1, 2004, to January 2, 2005, 167,476 cattle were tested. Not a single positive animal was found.[2]

In May 2004, the New Jersey Department of Health and Senior Services (NJDHSS) and the Centers for Disease Control and Prevention (CDC) reported on a suspected cluster of Creutzfeldt-Jakob disease (CJD) cases (including one Virginia resident) reportedly linked to the Garden State Racetrack in Cherry Hill, New Jersey. Although there was public concern that these deaths might have resulted from the consumption of meat contaminated with the agent that causes BSE, the study did not find a significantly elevated number of cases of CJD or a common etiology for the cases of CJD that were identified.[3]

However, incidents like these reinforce the need for healthcare providers to be aware of the previously low-profile group of diseases known as transmissible spongiform encephalopathies (TSEs). This article is part of the Virginia Department of Health's efforts to improve surveillance for TSEs in Virginia to better understand the disease as well as to detect any changes in disease patterns.

In This Issue:

Overview of TSEs

TSEs are a group of relatively rare progressive neurodegenerative disorders that affect both humans and animals. They are distinguished by long incubation periods, characteristic spongiform changes of the brain associated with neuronal loss, and the lack of a detectable immune or inflammatory response. Unconventional agents termed prions (or proteinacious infectious particles), abnormal isoforms of a normal cellular protein encoded by a gene on chromosome 20, are considered the etiologic agent by most scientists.[4] An alternative hypothesis, the virino model, proposes that the agent consists of a small nucleic acid, and that this molecule is protected by the host prion protein. A third theory proposes that TSEs are caused by conventional viruses. However, to date no infection-specific nucleic acid has been detected in cases of TSE.[5]

Known TSEs include:

- Creutzfeldt-Jakob disease (CJD), New Variant CJD (vCJD), Kuru, Gerstmann-Straüssler-Scheinker (GSS), and Fatal Familial Insomnia (FFI) in humans;
- Scrapie in sheep and goats;
- Bovine Spongiform Encephalopathy (BSE) in cattle;

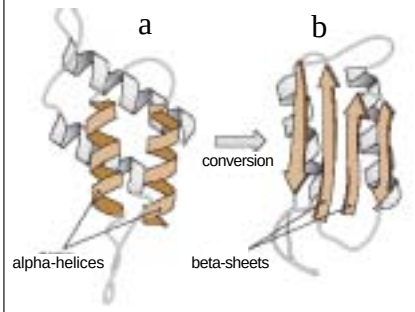


***Figure 1**. (a) A healthy cellular prion protein contains only alpha-helices. (b) The pathogenic isoform with alpha-helical structures converted to beta-sheets.*[6]

- Chronic Wasting Disease (CWD) in deer and elk;
- Exotic ungulate encephalopathy (EUE) in nyala and greater kudu;
- Transmissible Mink Encephalopathy (TME) in mink; and
- Feline Spongiform Encephalopathy (FSE) in cats.

While there is no evidence of contact or aerosol transmission for most prion diseases, they are infectious under some circumstances, such as direct central nervous system inoculation or consuming contaminated tissue. Prions are also characterized by extreme resistance to conventional inactivation procedures including irradiation, boiling, dry heat, and chemicals (formalin, alcohols). And while prion infectivity in purified samples is diminished by prolonged digestion with proteases, prion inactivation requires 1N NaOH, 4M guanidinium hydrochloride or isocyanate,

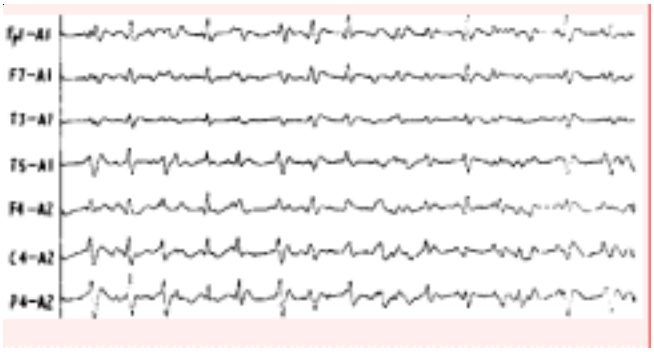


***Figure 2**. EEG typical of CJD, with diffuse one-hertz triphasic waves.[7]*

sodium hypochlorite (2% free chlorine concentration), or steam autoclaving at 132°C for 4.5 hours.[8]

Creutzfeldt-Jakob Disease (CJD)

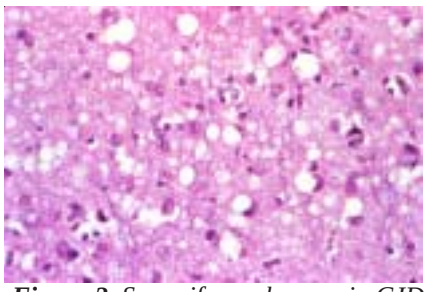


***Figure 3**. Spongiform changes in CJD*

The prototype TSE in humans, Creutzfeldt-Jakob disease (CJD), is a rapidly fatal dementing illness that occurs worldwide. Although 10% of cases are due to autosomal dominant inheritance (familial CJD), most cases (>90%) occur unpredictably in the population (sporadic CJD). Less than 1% of cases are the result of iatrogenic transmission (e.g., associated with receipt of contaminated human pituitary growth hormone, human pituitary gonadotrophin, dura mater grafts or corneal tissue, or exposure to contaminated neurosurgical equipment).[4,9] However, all six of the cases linked to the use of contaminated equipment occurred before the implementation of sterilization procedures currently used in health care facilities. No such cases have been reported since 1976.[9]

The incubation period of CJD can range from one year to more than 30 years.[10] Prodromal symptoms may include headache, anorexia, behavioral changes, and depression.[7] Initial neurologic signs can include progressive memory loss over weeks to months, as well as vertigo, blurred vision, visual field defects, or diplopia. Mental deficits rapidly lead to global dementia, as well as self-neglect, apathy, or irritability. Some patients complain of easy fatigability, somnolence, or insomnia. Other abnormalities of higher cortical function (e.g., aphasia, apraxia, dyslexia, dysgraphia, agnosia, left-right disorientation, unilateral neglect) may occur.[10]

Myoclonus, often provoked by sensory stimuli, usually appears within the first six months of symptom onset. Cerebellar disturbances occur and corticospinal tract involvement (such as extensor plantar reflexes, clonus, and hyperreflexia) appear. Signs of basal ganglia involvement, such as hypokinesia, dystonic posturing, cogwheel rigidity, tremor, and choreoathetoid movements, may develop. In the final stage the patient loses all mental and physical functions. The patient may lapse into a coma and usually dies from an infection, such as aspiration pneumonia, precipitated by the bedridden, unconscious state.[10]

Neuropathologic evaluation, particularly by immunohistochemistry or Western blot, is currently the most definitive method to diagnose human prion diseases. In terms of treatment, although some medications can help relieve jerking movements and unsteadiness (e.g., clonazepam) and the possible benefits of quinacrine are under evaluation, there is no cure for TSEs and they are invariably fatal.[6,8]

US CJD Epidemiology

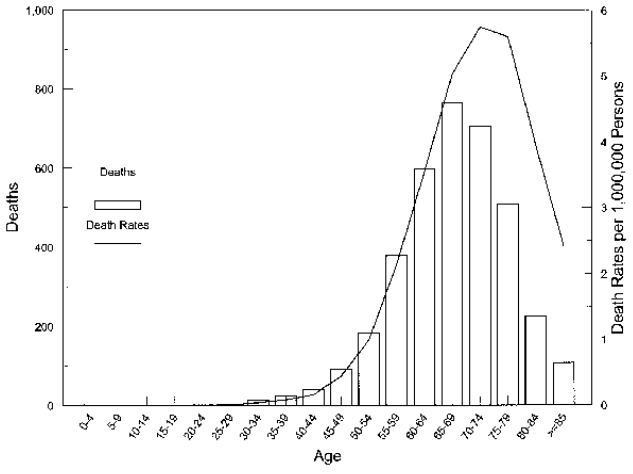
In 1996, Holman et al. examined trends in the incidence of CJD in the United States. From 1979 through 1994, CJD was recorded as the cause of 3,642 deaths. The average age at death was 67 years. Approximately 98% of the deaths were among persons 45 years of age or older, and 80% of the CJD deaths were among persons 60 years of age or older (Figure 4).[4]

The average annual age-adjusted death rate during the study period was 0.95 deaths per million persons in the US. This is consistent with published estimates of the crude incidence worldwide of one case per million persons. The age-adjusted death rate of male patients was slightly higher than that of female patients, and most (95.2%) deaths were among whites.[4]

Virginia CJD Epidemiology

CJD is currently a reportable condition in Virginia only if the ill person is less than 55 years of age; therefore data on this condition are very limited. However, a review of death certificate data in 2004 found a total of 72 cases of CJD from 1986-2003 (annual range: 0 to 10 cases). This suggests an overall incidence of CJD of 0.62 per million persons over the 18 year period. Among the five regions in Virginia, the Northern region had the lowest incidence (0.29/1,000,000 persons/year), while the Eastern region had the highest (0.96/1,000,000 persons/year). The rates in the 35 health districts (using 2002 populations) varied from 0/1,000,000 persons/year to 2.47/1,000,000 persons/year (Figure 5). However, due to the very small numbers of cases of CJD that were re-

***Figure 4**. Creutzfeldt-Jakob disease deaths and death rates by age group, United States, 1979 through 1994.[4]*



ported, these rates are considered unstable.

The average age at death of CJD cases in Virginia from 1986 through 2003 was 70 years (range: 42-94 years). More cases occurred in females (64%) than males. The disease predominantly occurred in whites (89%) with 8% of cases in blacks and 1% of cases in Asians. Therefore, the overall pattern of CJD in Virginia appears comparable to the US pattern.

One limitation to our understanding of CJD in Virginia and the US is that surveillance mainly uses death certificate data. However, it is very likely that some cases do not get recorded. CJD is an uncommon disease and physicians who are not familiar with the signs and symptoms may mistake it for other conditions. Evidence of the under-reporting of cases comes from some European countries, where enhanced surveillance has found higher than expected rates due to increased awareness and better diagnosis.[11] In addition, one small study reported that as many as 13% of patients diagnosed with Alzheimer's disease were found upon autopsy to have actually had CJD.[12] Therefore, it seems likely that the current data underestimate the true incidence of CJD in Virginia and in the US.

Table 1: Clinical and pathologic characteristics distinguishing classic Creutzfeldt-Jakob disease (CJD) from variant CJD (vCJD)

Characteristic	CJD	vCJD
Median age at death	68 yrs	28 yrs
Median duration of illness	4-5 months	13-14 months
Clinical signs and symptoms	Dementia; early neurologic signs	Prominent psychiatric/behavioral symptoms; painful dysesthesias; delayed neurologic signs
Periodic sharp waves on electroencephalogram	Often present	Absent
'Pulvinar sign' on MRI*	Not reported	Present in >75%
Presence of 'florid plaques' on neuropathology	Rare or absent	Present in large numbers
Immunohistochemical analysis of brain tissue	Variable accumulation	Marked accumulation of PrP^{res}**
Presence of agent in lymphoid tissue	Not readily detected	Readily detected
Increased glycoform ratio on immunoblot analysis of PrP^{res}	Not reported	Marked accumulation of PrP^{res}
Genotype at codon 129 of prion protein	Polymorphic	Methionine/methionine

Source: Adapted from Belay E, Schonberger L. 2002. *Clin Lab Med*. 22:849-62.[14]

*Symmetrical hyperintensity of the pulvinar (posterior) nuclei of the thalamus compared to the anterior putamen on brain magnetic resonance imaging (MRI); in the appropriate clinical context, this signal is highly specific for vCJD.[15]
**Protease-resistant prion protein.

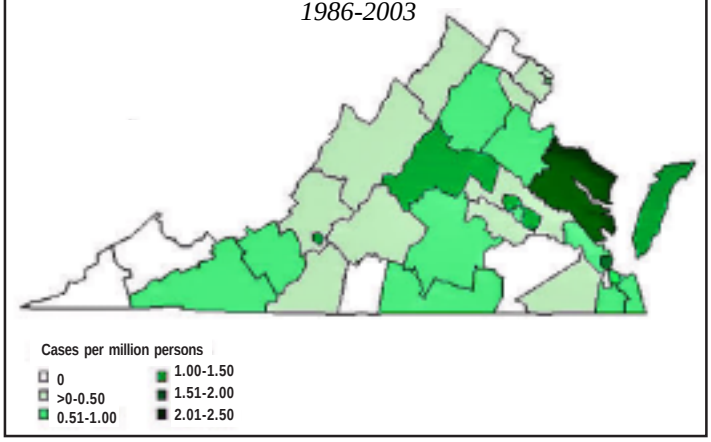
Bovine Spongiform Encephalopathy (BSE) and Variant CJD (vCJD)

Scrapie, an endemic spongiform encephalopathy of sheep and goats, has been recognized since the mid-18th century.[13] The widespread epizootic of BSE that was detected in 1986 in the United Kingdom (UK) may have originated from scrapie[4] as a result of contaminated rendered sheep carcasses that were fed to cattle. Since then cases of BSE have also been detected in many other countries as a result of imported contaminated live animals or livestock food supplements. While the incidence of new cases is decreasing in some countries (including the UK) in other countries (e.g., France, Portugal, Germany, Spain, and the Republic of Ireland) the incidence appears to be increasing. This is partly explained by improved diagnosis and active surveillance, but new infections may also be occurring.[13]

During the 10 years after the first cow with BSE was identified, there was no change in clinical or neuropathologic features of human cases of CJD in the UK. However, in 1994 a cluster of cases with a unique clinical picture began to appear. Termed "new variant of CJD" (vCJD), the epidemiology for the disease differs from "classic" CJD. For example, the median age at death is much younger (28 years, versus 68 years for classic CJD).[1,4] In addition, the median duration of illness before death is 13-14 months for vCJD patients, compared to 4-5 months for classic CJD (see Table 1).[1]

Clinical differences also exist between patients with vCJD and classic CJD. Patients with vCJD have prominent early behavioral or psychiatric manifestations and dysethesias (painful sensory symptoms), with neurologic signs such as myoclonus and extrapyramidal dysfunction delayed for several months after illness onset. The characteristic electroencephalographic pattern of periodic sharp waves observed in classic CJD patients is absent in patients with vCJD. In addition, more than 75% of patients with vCJD have a characteristic symmetrical hyperintensity of

Figure 5. *Creutzfeldt-Jakob Disease in Virginia 1986-2003*



the pulvinar (posterior) nuclei of the thalamus compared to the anterior putamen on brain magnetic resonance imaging (MRI) (the "pulvinar sign"); in the appropriate clinical context this is highly indicative of a vCJD diagnosis.[1,15] Variant CJD cases also have characteristic severe spongiform change, neuronal loss, and astrocytosis in the basal ganglia and thalamus, and a distinctive florid or "daisy" plaque (an amyloid core surrounded by "petals" of spongiform change in the cerebrum and cerebellum and protease-resistant prion protein (PrP^{res}) accumulation in high density shown by immunocytochemistry).[1,4,13] Prions are detected readily by immunohistochemical analysis in lymphoid tissues (e.g., appendix, lymph nodes, spleen, and tonsils) of vCJD patients, but not in classic CJD patients. Finally, all persons with vCJD (tested as of January 2004) have had methionine homozygosity at codon 129 of the prion protein gene, indicating that persons who do not carry this genotype (the majority of the general population) may have increased resistance to vCJD.[1]

Evidence to date indicates that there has never been a case of vCJD transmitted through direct contact of one person with another.[16] The main association is with the BSE epizootic in British cattle. For example, the first and only (as of January 2005) probable vCJD case in a US resident was diagnosed in 2002. This patient had grown up in the UK when the risk for human exposure to the agent of BSE was at its peak. Therefore, it is likely that this patient was exposed to the BSE agent one or more times during 1980-1992 before moving to the United States.[17]

Overall, the risk for acquiring vCJD from BSE-contaminated product appears to be low. For example, in the UK more than one million cattle were probably infected with BSE.[1] In contrast, the vCJD outbreak has shown only a modest increase in cases (as of December 1, 2003, a total of 153 cases of vCJD had been reported worldwide).[13,16] The disparity between the number of cases of BSE and the number of cases of vCJD suggests that the very small infectious doses cannot readily surmount the combined effects of a "species barrier" and a comparatively inefficient route of infection.[13]

Chronic Wasting Disease (CWD)

Chronic wasting disease (CWD) was first identified as a syndrome of captive mule deer in the late 1960s and was recognized as a TSE in 1978. Its only known natural hosts are mule and white-tail deer (*Odocoileus* species) and Rocky Mountain elk (*Cervus elaphus nelsoni*).[18]

Clinical manifestations of CWD include weight loss over weeks or months, behavioral changes, excessive salivation, difficulty swallowing, polydipsia, and polyuria. In some animals, ataxia and head tremors may occur. Most animals with the disease die within several months of illness onset, sometimes from aspiration pneumonia. In rare cases, illness may last for more than one year. In captive cervids, most cases occur in animals 2 to 7 years of age; however, the disease has been reported in cervids as young as 17 months and as old as 15 years of age. This disease can be highly transmissible within captive deer and elk populations, although the mode of transmission is not fully understood. Evidence supports lateral transmission through direct animal-to-animal contact or indirect exposure, such as though contaminated feed and water sources.[18]

Figure 6. *Positive CWD Areas in North America*

Although the original CWD-endemic areas were Colorado and Wyoming, the geographic distribution seems to be changing as a result of the natural movement of deer and elk as well as commercial movement of infected animals (Figure 6). For example, CWD in free-ranging cervids was first reported east of the Mississippi River in Wisconsin among white-tailed deer harvested in the 2001 hunting season.[18] As a result, the Virginia Department of Game and Inland Fisheries is conducting an extensive surveillance program. From 2002-2004 over 1,200 animals were tested from randomly selected hunter-harvested deer, target or suspect animal surveillance, and all captive deer mortalities. To date, CWD has not been detected in Virginia. In addition, measures to prevent the spread of CWD to Virginia include a ban on the importation or intrastate movement of cervids, strict controls over the deer farming industry, euthanizing and testing any illegal cervids, and a policy decision not to reintroduce elk into Virginia.[19]

A major concern with CWD is the possible transmission of the agent to domestic animals, such as cattle and sheep, which may come into contact with infected deer and elk or CWD-contaminated environments. If such transmissions were to occur, it could increase the extent and frequency of human exposure to the CWD agent. Passage of the agent through a secondary host could also alter its infectious properties, increasing its potential for becoming more pathogenic to humans.[18]

However, while CWD has been transmitted experimentally by intracerebral injection to a number of animals, it does not appear to occur naturally outside the cervid family. Domestic cattle, sheep, and goats residing in research facilities in close contact with infected cervids did not develop a prion disease. In addition, despite the decades-long endemicity of CWD in Colorado and Wyoming, the incidence of CJD and the age distribution of CJD case-patients in these two states are similar to those seen in other parts of the United States.[18] Finally, laboratory investigations of case-patients diagnosed with CJD have not indicated a causal link

to CWD.[18,20] These findings suggest that the risk of transmission of CWD to humans is very low.[18]

Unfortunately, the transmission of BSE to humans and the resulting cases of vCJD indicate that, provided sufficient exposure, the species barrier may not completely protect humans from animal prion diseases. Because CWD has occurred in a limited geographic area, an adequate number of people may not have been exposed to the CWD agent to result in a clinically recognizable human disease. However, the level and frequency of human exposure to the CWD agent may increase with the spread of CWD in the United States. More epidemiologic and laboratory studies need to be conducted to monitor the possibility of such transmissions.[18]

TSE Prevention

A wide range of efforts, from national to state to institutional level, have been developed in various countries to prevent the transmission of TSEs. In the US, these include:

- Preventing importation—severe restrictions are placed on the importation of live ruminants (e.g., cattle, sheep, and goats) and certain ruminant products from countries where BSE is/was known to exist;[16]
- Surveillance in humans and animals;
- Preventing amplification—the US Food and Drug Administration instituted a ruminant feed ban in June 1997 to prevent the transmission to other animals from infectious tissues;[16]
- Preventing blood-borne human transmission—a blood donor policy excludes donations from anyone who has lived in or visited European countries for a cumulative period of six months or more since 1980.[13] In addition, the American Red Cross will not accept blood donations from people who have ever received a transplant of high-risk tissue (e.g., dura mater) or from those who have a close blood relative who has had CJD;[21] and,
- Infection control—since the standard methods of sterilization are ineffective for prions, the World Health Organization (WHO) has developed CJD infection control guidelines. Details are available from the WHO website at http://www.who.int/emc-documents/tse/whocdscsraph 2003c.html.

While not an exhaustive list of prevention measures, this list suggests the wide scope of interventions that have been developed to monitor and prevent the spread of these diseases.

(continued on page 6)

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Conclusions

The need to better understand classic CJD, the emergence of BSE and the spread of CWD in the US all reinforce the need to consider TSEs in patients, regardless of age, who report with distinguishing neurological signs and symptoms. Although reporting CJD cases in Virginia is required only if the patient is less than 55 years of age, healthcare providers are encouraged to report all cases of CJD to their local health departments as this may help to identify cases of vCJD, as well as improve the understanding of TSEs in Virginia.

In addition, because testing brain tissue permits the most definitive diagnosis of all forms of CJD and identification of emerging forms of the disease, VDH encourages physicians to arrange for brain autopsies in all decedents with known or suspected CJD. The National Prion Disease Pathology Surveillance Center (NPDPSC) provides neuropathologic and biochemical diagnostic services free of charge to US physicians and state and local health departments. This includes testing for the presence of the 14-3-3 protein (a marker for some prion diseases when other conditions have been excluded) in cerebrospinal fluid (CSF), detecting mutations in prion protein genes, and histological and immunohistochemical examination for the prion protein in specimens.[1] Information about these services is available from NPDPSC at http://www.cjdsurveillance.com or from the CDC (telephone 404-639-3091). Protocols for autopsy, biopsy and other specimens (e.g., blood, urine, CSF), forms and shipping/mailing instructions for specimens are also available on the NPDPSC website.[1,3,17]

Submitted by: Christopher Novak, MD, MPH and Aileen Buckler, MD, MPH

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Office of the Chief Medical Examiner 2004 Annual Report

Under § 32.1-283 of the *Code of Virginia*, the Office of the Chief Medical Examiner (OCME) has the responsibility for investigating:

- any death from trauma, injury, violence, or poisoning attributable to accident, suicide or homicide;
- sudden deaths to persons in apparent good health or deaths unattended by a physician;
- deaths of persons in jail, prison, or another correctional institution, or in police custody;
- deaths of patients/residents of state mental health or mental retardation facilities;
- the sudden death of any infant less than eighteen months of age whose death might be attributable to Sudden Infant Death Syndrome; and
- any other suspicious, unusual, or unnatural death.

The Virginia Department of Health Office of the Chief Medical Examiner 2004 Annual Report summarizes the findings from the 5,821 deaths (10.1% of the estimated total deaths in Virginia) investigated by the OCME in 2003. The report includes detailed analyses of overall deaths by temporal, geographic and demographic factors, as well as manner of death. In addition, more specific analyses of deaths in children, deaths related to ethanol use, motor vehicle-related deaths, and drug-related deaths have been provided. Overall, this is the most detailed analysis of medical examiner cases ever produced in Virginia.

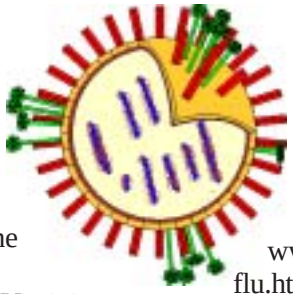
The full report from the OCME is available on-line at: www.vdh.virginia.gov/medexam/OCMEAnRpt04.pdf.

Did you know?

In Virginia in 2003, more deaths occurred on Saturdays than any other day of the week. The fewest number of deaths occurred on Tuesdays.

Flu Corner

Influenza Activity



Influenza activity was low in Virginia during October through early December 2004, but has increased steadily since mid-December. During the week ending January 22, 2005, influenza activity in Virginia was classified as Regional (activity greater than threshold in two or more regions AND either lab confirmed influenza in the affected regions within the past 3 weeks OR recent detection of a laboratory confirmed outbreak in two regions). Nationwide, fifteen states/territories reported widespread influenza activity, 17 reported regional activity, 10 reported local activity, and 11 reported sporadic activity. The Centers for Disease Control and Prevention (CDC) reported that during this same period the proportion of deaths attributed to pneumonia and influenza remained below the epidemic threshold.

By January 29, 2005, the Division of Consolidated Laboratory Services (DCLS) reported 56 confirmed cases of influenza (54 type A and 2 type B) by direct fluorescent antibody (DFA) and/or culture. Twenty-nine cases were from the central region, eleven were from the northwest region, nine were from the eastern region, five were from the southwest region and two were from the northern region.

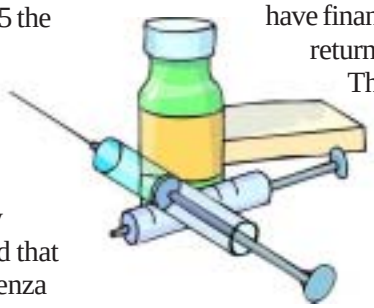
For up-to-date information on national influenza patterns go to: www.cdc.gov/flu/weekly/fluactivity.htm. Information on influenza surveillance in Virginia is available on the VDH website at www.vdh.virginia.gov/epi/flu.htm.

Influenza Vaccine Update

This season's influenza vaccine strains have been well matched antigenically to the influenza viruses isolated to date. Late-season vaccination can still offer protection against influenza this season. Therefore, the CDC continues to recommend aggressive efforts to vaccinate people in priority groups.

On January 26, the State Health Commissioner authorized health directors in Virginia to relax restrictions on the use of influenza vaccine in their respective districts if they feel the demand for vaccination within priority groups has been met. This will allow remaining doses of vaccine to be used to provide protection against influenza for as many people as possible.

In addition, in January 2005 the demand for Vaccine for Children (VFC) influenza vaccine had decreased. To increase the availability of the influenza vaccine to those who need it, on January 27, 2005, the CDC announced that limited amounts of VFC influenza vaccine available to health departments could be used for any non-VFC individuals (child or adult) in localities where the demand for influenza vaccine among VFC eligible children has already been met. Preservative-free pediatric 0.25ml doses may be administered to adults if two doses are administered at separate injection sites. This use of VFC vaccine is limited to health departments and non-profit organizations, such as federally qualified health centers and rural health clinics, in accordance with policies established by the district director, and only for the 2004-2005 influenza season.



Private physicians may not administer VFC vaccine to non-VFC patients. **Unused VFC influenza vaccine in private physicians' offices may be transferred to health departments.** Only unopened vials may be accepted in the transfer. However, private physicians have the opportunity to purchase additional vaccine directly from sanofi pasteur (formerly Aventis). A vaccine "return policy" has been developed (for the current influenza season only) where providers will be allowed to return unused vaccine for a credit and will have financial responsibility for return shipping costs only. These strategies should help ensure that as many people as possible are protected from influenza.

Virginia Joins the National Electronic Disease Surveillance System (NEDSS)

The Virginia Department of Health (VDH) has begun to submit its weekly disease report data to the U.S. Centers for Disease Control and Prevention (CDC) using the National Electronic Disease Surveillance System (NEDSS). This makes Virginia the tenth state in the country to utilize this new system. While this is the first step in the process of implementing the system statewide, it represents the culmination of years of effort by VDH staff to prepare to implement NEDSS in Virginia. For now, use of the system is limited to certain central office staff of the Office of Epidemiology. Use of the system will expand to district offices and other reportable disease program offices within VDH during 2005.

Cases of Selected Notifiable Diseases Reported in Virginia*

Disease	Total Cases Reported, December 2004: State	Regions: NW	N	SW	C	E	Total Cases Reported Statewide, January through December: This Year	Last Year	5 Yr Avg
AIDS	164	14	24	11	84	31	774	772	829
Campylobacteriosis	48	10	7	15	6	10	643	882	664
E. coli O157:H7	6	1	0	3	2	0	39	50	66
Giardiasis	48	9	8	7	19	5	523	426	427
Gonorrhea	603	49	40	83	157	274	8,506	9,063	10,018
Hepatitis, Viral									
A	15	0	6	3	3	3	135	141	164
B, acute	38	3	4	11	13	7	277	227	189
C, acute	1	1	0	0	0	0	17	15	9
HIV Infection	104	5	24	14	31	30	880	797	882
Lead in Children†	42	5	3	9	14	11	800	769	707
Legionellosis	4	0	2	0	2	0	53	110	52
Lyme Disease	8	2	5	0	0	1	174	195	176
Measles	0	0	0	0	0	0	0	0	4
Meningococcal Infection	0	0	0	0	0	0	20	28	44
Mumps	0	0	0	0	0	0	3	1	7
Pertussis	37	19	7	4	3	4	233	219	172
Rabies in Animals	28	4	6	10	2	6	474	542	558
Rocky Mountain Spotted Fever	7	1	2	0	2	2	38	34	29
Rubella	0	0	0	0	0	0	0	0	0
Salmonellosis	73	10	17	21	9	16	1,146	1,183	1,227
Shigellosis	16	1	6	2	6	1	165	453	579
Syphilis, Early§	31	0	11	3	3	14	216	155	237
Tuberculosis	84	8	44	7	12	13	329	332	316

Localities Reporting Animal Rabies This Month: Accomack 1 raccoon; Appomattox 1 raccoon; Augusta 1 raccoon; Bedford 1 cow; Chesterfield 1 raccoon; Fairfax 2 foxes, 2 raccoons, 1 skunk; Floyd 1 raccoon; Goochland 1 raccoon; Hampton 1 raccoon; Highland 1 fox; King William 1 skunk; Loudoun 1 raccoon; Montgomery 1 cow; Northampton 1 cat; Patrick 1 raccoon, 1 skunk; Pittsylvania 1 skunk; Pulaski 1 raccoon; Rockbridge 1 skunk; Rockingham 1 skunk; Russell 1 skunk; Smyth 1 raccoon; York 2 raccoons.

Toxic Substance-related Illnesses: Asbestosis 4; Adult Lead Exposure 15; Lung Cancer/Metal Exposure 1; Pneumoconiosis 6; Silicosis 1.

*Data for 2004 are provisional. †Elevated blood lead levels ≥10µg/dL. §Includes primary, secondary, and early latent.

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