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Radon: A Natural Pollutant

Recent evidence suggests that exposure to radon, especially indoors, represents a greater public health threat than previously recognized (Table 1). Efforts to improve the energy efficiency of homes have exacerbated the problem. Indeed, the result is a paradoxical situation where energy conservation may cause more radiation exposure to a population than generation of an equivalent amount of energy.

Radon is a radioactive gas produced by the natural decay of uranium to radium and then to radon. It has a half-life of only 3.8 days and decays into its so called "daughters." It is the short-lived daughters (polonium 218, polonium 214, bismuth 214, and lead 214) which represent a potential health hazard. They are solids and attach to dust particles, which, if inhaled, result in exposure of the bronchial epithelium to the emitted alpha radiation.

Radon is measured in picocuries per cubic meter (pCi/m^3) and its daughters are measured in working levels (WL)*, a unit developed for calculating the exposures of miners. Exposure is expressed as working level months (WLM), which is the product of the WL concentration times the number of months of exposure (months are defined as the number of hours a worker spends on the job during a month, i.e. 170 hours).

Radon is given off by the top few meters of soil at a rate of approximately 0.5 pCi/m^2/sec. Soil radon also dissolves in groundwater and is released if the water is exposed to air. Outdoors, radon is dispersed by wind and diffusion before it decays to its daughters; the average outdoor concentration of daughters is therefore low (<0.001 WL). Indoors, radon may get trapped, preventing dispersion. If this happens, the radon daughter concentration will be higher than outdoors (usually 0.002-0.04 WL).

Table 1: Average U.S. Background Radiation (in mrem/y)

	Gonads	*Bones*	*Bronchi*
Cosmic radiation	28	28	28
External terrestrial	26	26	26
Radionuclides in the body	35	105	40
Inhaled radionuclides	—	—	3000*

**Includes indoor exposure to radon daughters. Previous estimates (based on outdoor levels of radon daughters) had placed this number at 450 mrem/y.*

Miners exposed to radon and its daughters have an excess risk of bronchogenic carcinoma. Using mathematical models, it is possible to estimate the risk associated with any particular radon daughter exposure. Continuous exposure to 2 WLM/y would result in an increased lifetime risk of bronchogenic carcinoma of two percent. Based on current estimates of exposure in the U.S., 10,000 cases per year, including one quarter of all cases in nonsmokers, are attributable to radon daughters.

Outdoor concentrations of radon vary widely depending on season, time of day, and geographic location. Indoor concentrations are unpredictable and apparently depend on type of underlying soil, construction characteristics, ventilation and geographic location. A limited number of sampling surveys have been conducted in the U.S. Best estimates are that one to two percent of U.S. households (approximately one million) have exposures to radon daughters which are in excess of the National Council on Radiation Protection (NCRP) guidelines of 2 WLM/y.

More studies are needed to identify risk factors for excessive exposure. The Department has plans underway to conduct a survey for radon in Virginia homes. If an excessive level of radon is detected in a home, it can usually be controlled through improved ventilation, use of concrete/masonry sealants, or improved air filtration (electrostatic precipitators).

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A Clinician's Guide to Screening and Evaluation

Childhood Lead Poisoning

Editor's note: these recommendations were abridged by Sathyavathi Lingaraju, M.D., M.P.H., Bureau of Maternal and Child Health, VDH, from revised Centers for Disease Control recommendations.[1]

INTRODUCTION

New epidemiologic, clinical and experimental evidence has indicated that lead is toxic at levels previously thought to be non-toxic. In the 1960's, a level below 60 micrograms of lead per deciliter (mcg/dl) of whole blood was not considered dangerous enough to require intervention. By 1975, the intervention level had declined to 30 mcg/dl. Lead toxicity is now defined as an elevated blood lead level of 25 mcg/dl or greater with an erythrocyte protoporphyrin (EP) level in whole blood of 35 mcg/dl or greater.

PREVALENCE RATES

Current information on prevalence of childhood lead toxicity is based on the Second National Health and Nutrition Examination Survey (NHANES II) conducted by the National Center for Health Statistics between 1976 and 1980. Results indicated that 3.9% of all United States children under the age of 5 years had lead levels of 30 mcg/dl or more, an estimated 675,000 children. Two percent of all white children and 12.2% of all black children had elevated lead levels. For black children living in cores of large cities and in families with income of less than $6,000, the rate was 18.6%.

In Virginia, door-to-door screening in 1984 of children living in high risk neighborhoods in the cities of Petersburg and Suffolk revealed that the prevalence of lead levels ≥30 mcg/dl were 3.0% and 3.2%, respectively. In the cities of Norfolk, Lynchburg, Portsmouth and Richmond, where active case finding and environmental hazard reduction have existed for 10 years, the prevalence rates in high risk census tracts have dropped from 6% or more to 2% or less.

SOURCES OF LEAD

Lead-based paint continues to be the major source of high level symptomatic lead poisoning. Since 1977, regulations require that household paint contain no more than 0.06% lead by dry weight. However, an estimated 27 million households in this country still have lead-based interior paint. Increasingly, urban home renovations have been a source of poisoning in both adults and children.

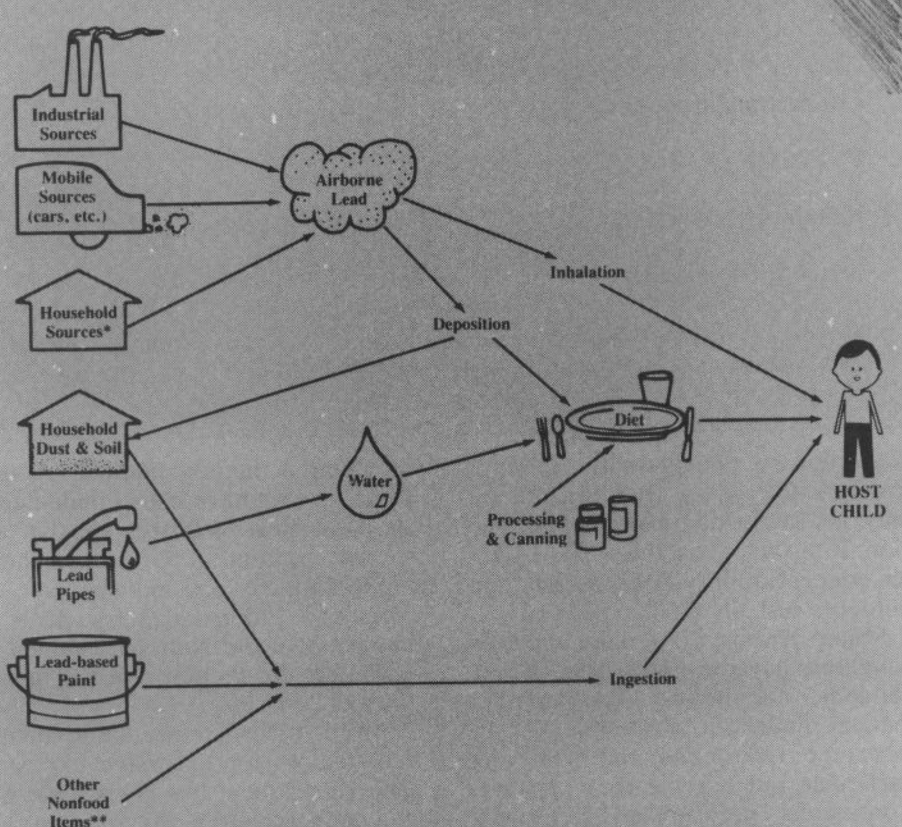
Lead in soil and housedust can be a primary source of lead for toddlers and preschool children whose normal mouthing behavior places them at increased risk. Flaking lead paint from a house exterior, deposition of scraped interior paint in a yard, and deposition of airborne lead in the soil have each been implicated as significant sources of lead exposure.

Lower levels of lead toxicity have been demonstrated in children living near major roadways with high traffic densities. The reduction of lead in gasoline has been associated with a decline in mean blood lead values in the United States population from 14.6 mcg/dl in 1976 to 9.2 mcg/dl in 1980. Stationary sources such as lead smelters are another source of airborne lead.

Lead is carried from the workplace to the home on the body, clothing and vehicles of workers. Lead crosses the placenta in pregnant women to the extent that the lead concentration in umbilical cord blood may equal that in maternal blood.

Acidic soft water leaches lead into drinking water from pipes and soldered joints. Canned foods, crops and

Figure 1: Sources of Lead in a Child's Environment



**Production of bullets or fishing sinkers*
Soldering and stained-glass work
Gasoline sniffing
Pottery glazing
Burning of batteries, colored newsprint, lead-painted objects, and waste oil

***Toys and figures containing lead*
Folk remedies
Cosmetics (especially Oriental cosmetics, e.g., Surma, a black eyeliner)
Jewelry (painted with lead to simulate pearl)
Lead-containing dust transmitted on clothing from workplace

vegetables grown in contaminated soil are potential sources. Eating or drinking from lead-glazed pottery; use of some folk remedies; burning of colored newsprint, battery casings, or lead-painted wood; and sniffing gasoline are all important sources of occasional lead poisoning (Figure 1).

HEALTH EFFECTS

Effects of high level lead poisoning, such as lead encephalopathy and mental retardation, are well known. It is now known that lower levels of lead may cause serious behavioral and biochemical changes with altered neurophysiological performance. The heme biosynthetic pathway is altered early and thus an elevated erythrocyte protoporphyrin (EP) level is one of the most reliable means of screening. Once absorbed, lead is distributed throughout soft tissue and bone. Blood levels reflect only the circulating amounts and not the body burden of lead deposited in bones. Deficiencies in iron, calcium and increased dietary fat enhance the absorption of lead.

Young children absorb and retain more lead on a unit-mass basis than adults and they are also more vulnerable to the neurophysiological effects than adults. Effects of lead toxicity are non-specific and present as altered behaviors such as attention disorders, emotional disturbances and learning disorders. Early signs and symptoms are fatigue, pallor, malaise, loss of appetite, irritability, sleep disturbance, sudden behavior changes and regression in development. More serious symptoms are clumsiness, ataxia, weakness, abdominal pain, vomiting, constipation and changes in consciousness due to encephalopathy.

SCREENING FOR LEAD TOXICITY

Screening is to be distinguished from diagnosis. The child with symptoms or signs suggestive of lead poisoning should have immediate diagnostic evaluation. Screening refers to the detection of asymptomatic lead toxicity in masses of children at risk.

Target Population:

Screening, at least once a year, is recommended for all children between 9 months and 6 years of age (Table 1). EP testing has been used to screen for both iron deficiency and lead toxicity. High risk children (low income, living in old dwellings) are generally eligible for Women and Infant Children (WIC) services. Participation in WIC requires a hematologic determination for iron deficiency state or anemia every six months. Determination of EP instead of hematocrit or hemoglobin may be a cost-effective means of screening for both iron deficiency and lead toxicity.

Table 1: Suggested Priority Groups for Lead Screening

Priority	
1.	HIGHEST—Children, age 12 to 36 months, who live in or are frequent visitors in older, dilapidated housing
2.	Children, age 9 months to 6 years, who are siblings, housemates, visitors, and playmates of children with known lead toxicity
3.	Children, age 9 months to 6 years, living in older, dilapidated housing
4.	Children, age 9 months to 6 years, who live near lead smelters and processing plants or whose parents or other household members participate in a lead-related occupation or hobby
5.	Children, age 9 months to 6 years, who live near highways with heavy traffic or near hazardous waste sites where lead is a major pollutant
6.	All children 12 to 36 months of age
7.	All children 9 months to 6 years of age

Erythrocyte Protoporphyrin:

When capillary blood samples are taken, external contamination of the sample with lead is possible. This would result in a falsely elevated blood lead level but would not influence the EP level. Confirmation of lead values is therefore required by a venous blood sample. An EP value of 35 mcg/dl indicates impaired heme synthesis due to iron deficiency and/or toxic effects of lead.

The advantages of screening with an EP test are that it identifies children with elevated lead levels, contamination of the sample with lead does not affect the EP value, and it is an accepted screening test for iron deficiency. EP is also elevated in sickle cell anemia, other hemolytic anemias, and (slightly) after recent colds, ear infections and other minor illnesses. Hyperbilirubinemia may cause false elevations of EP measured by hematofluorometer, but not by extraction.

Use of Hematofluorometers:

EP can be measured by fluorometry after extraction from red cells or by direct fluorescence in intact cells. The ethylacetate-acetic acid extraction procedure converts zinc protoporphyrin (ZnPP), the metabolite present in red cells, to EP.Hematofluorometers measure ZnPP and report values in EP equivalents. At high levels, values obtained with hemotofluorometers may be lower than those obtained by extraction. Because hematofluorometers give immediate results, are easy to operate and economical, they are most suitable for mass screening.

Table 2 provides a risk classification for priority medical evaluations based on EP determinations. This classification is a general guideline only; blood test interpretation requires clinical judgment, taking into account the age of the child and the presence or absence of symptoms.

Children in class IV are at urgent risk of lead toxicity and should be medically evaluated within 24-48 hours. Those in class III are at high risk, those in class II are at moderate risk, and those in class I, at low risk.

Class I can be subdivided into two additional categories. Class Ia (blood lead, 25 mcg/dl or less, and EP, 35 mcg/dl or more) includes children with iron deficiency. These children should be retested, with additional assessment of iron status. Class IB (blood lead, 25-40 mcg/dl, and EP, less than 35 mcg/dl) covers children who appear to have transient, stable, declining, or increasing blood lead levels. Results should be confirmed by retesting, and the children should be carefully followed.

DIAGNOSTIC EVALUATION:

Blood lead levels may vary as much as ± 5 mcg/dl in a 24 hour period. Symptoms, if present, constitute an urgent risk. Lead encephalopathy is generally associated with lead levels exceeding 100 mcg/dl but may occur at levels as low as 70 mcg/dl. Symptomatic lead poisoning without encephalopathy is usually associated with values of 70 mcg/dl but may occur at levels as low as 50 mcg/dl. A careful, complete pediatric and environmental history should be obtained. The physical examination should include a careful neurological examination and assessment of the nutritional status. Children with confirmed lead toxicity should receive comprehensive neuropsychological evaluations; developmental screening tests are not sufficiently sensitive for assessment of subtle impairments.

The following tests may be useful:

1. *Tests for iron deficiency:* A normal hemoglobin (11 g/dl or more) or a normal hematocrit (33% or more) does not rule out iron deficiency. Serum iron, iron binding capacity or serum ferritin are indicated for an accurate assessment of iron deficiency.
2. *A flat plate of the abdomen* may be positive for radiopaque material only if a large amount of lead was ingested within the preceding 24 to 36 hours.
3. *X-rays of long bones,* usually of the knees, may reveal growth arrest lines of increased density in the metaphyses of the distal femur and proximal tibia and fibula. They are usually not seen in children under 2 years of age, and negative X-rays to not rule out lead exposure.
4. *The Calcium Disodium EDTA Mobilization Test* is used to identify children who will respond to chelation therapy. Children with lead levels above 55 mcg/dl should receive chelation therapy without a chelation test.
5. A search for basophilic stippling in red cells and tests for lead in hair and nails are not considered sufficiently sensitive to be useful in diagnosis.

CLINICAL MANAGEMENT

Space limitations preclude an adequate discussion here of the treatment of lead poisoning; the reader is referred to published literature on the subject.[1,4] In general, increased reliance is now placed on the calcium disodium EDTA mobilization test for children with moderate blood lead levels.

Most children with lead toxicity do not require chelation therapy. Such therapy should not be given without either a confirmed blood lead level of $\geq$56 mcg/dl or a positive mobilization test in children with blood lead levels of 25-55 m cg/dl.

A cornerstone of clinical management is the reduction in further lead exposure through environmental management.

ENVIRONMENTAL MANAGEMENT

Environmental investigation and intervention begin as soon as lead toxicity is confirmed. Priorities for action are based on the child's risk classification. Children may be exposed to lead paint in their own home or in homes they frequently visit or in day care settings, etc.

It is essential to remove children from the home while temporary or permanent abatement is being performed. Families should be instructed in housekeeping techniques such as frequent wet mopping and damp dusting to maintain a reduced level of environmental lead. It may be necessary to work with the local Department of Social Services in order to arrange for alternate housing for families with limited income.

SCREENING IN VIRGINIA

In Virginia, screening for iron deficiency and lead toxicity is being expanded in 1986. Hematofluorometers will be used in an additional fifty health departments for screening children in well child and WIC clinics. Some of the children participating in WIC services obtain primary health care from private physicians. The health department will notify private physicians involved in the care of those children of the EP and lead test results. Physicians interested in obtaining further information may contact the Bureau of Maternal and Child Health, Virginia Department of Health or their local health departments.

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(Continued to page 5)

Table 2. Risk Classification of Asymptomatic Children for Priority Medical Evaluation[a]

Blood Lead[b]	*Erythrocyte Protoporphyrin (EP)[b]*			
	<35	35-74[c]	75-174[d]	>175[e]
Not done	I	*	*	*
<24	I	Ia	Ia	EPP+
25-49	IB	II	III	III
50-69	**	III	III	IV
>70	**	**	IV	IV

a — see text for explanation of classification levels.
b — Units are in mcg/dl of whole blood.
c — Measured by hematofluorometer; use 35-109 if measured by extraction.
d — Use 110-249 if measured by extraction.
e — Use >250 if measured by extraction.
* — Blood lead test needed to estimate risk.
EPP+ — Erythropoietic protoporphyria. Iron deficiency may cause elevated EP levels up to 300 mcg/dl, but this is rare.
** — In practice, this combination of results is not generally observed; if it is observed, immediately retest with whole blood.

NOTE: Diagnostic evaluation is more urgent than the classification indicates for children with any symptoms compatible with lead toxicity, children under 36 months of age, children whose blood lead and EP lead levels place them in the upper part of a particular class, and children whose siblings are in a higher class.

These guidelines refer to the interpretation of screening results, but the final diagnosis and disposition rest on a more complete medical and laboratory examination of the child.

Recommendations for School and Day-Care Center Attendance

Acquired Imunodeficiency Syndrome (AIDS)

***tor's note:** The following recommendations were adopted by the Virginia State Board of Health on November 13, 1985. They were adapted from recommendations issued by the Centers for Disease Control of the U.S. Public Health Service (printed in last month's issue of the* Epidemiology Bulletin*), and the American Academy of Pediatrics. Please note that separate recommendations are given for schools and day-care centers.*

School Attendance

1. Decisions regarding the type of educational and care setting for HTLV-III/LAV-infected children should be based on the behavior, neurologic development, and physical condition of the child and the expected type of interaction with others in that setting. These decisions should be made using the team approach; such a team should include the child's physician, public health personnel and the child's parent or guardian. In each case, risks and benefits to both the infected child and to others in the setting should be weighed.
2. For most infected school-aged children, the benefits of an unrestricted setting would outweigh the risks of their acquiring potentially harmful infections in the setting and the apparent nonexistent risk of transmission of HTLV-III/LAV. These children should be allowed to attend school and after-school day-care and to be placed in a foster home in an unrestricted setting.
3. For the infected preschool-aged child and for some neurologically handicapped children who lack control of their body secretions or who display behavior such as biting, and those children who have uncoverable, oozing lesions, a more restricted environment is advisable until more is known about transmission in these settings.
4. Because other infections in addition to HTLV-III/LAV can be present in blood or body fluids, all schools regardless of whether children with HTLV-III/LAV infection are attending, should adopt routine procedures for handling blood or body fluids. Soiled surfaces should be promptly cleaned with disinfectants such as household bleach (diluted 1 part bleach to 10 parts water). Disposable towels or tissues should be used whenever possible, and mops should be rinsed in disinfectant. Those who are cleaning should wear disposable gloves and avoid exposure of open skin lesions or mucous membranes to the blood or body fluids.
5. Care, which involves exposure to the infected child's body fluids and excrement, should be provided by persons who are aware of the child's HTLV-III/LAV infection and the modes for HTLV-III/LAV transmission. In any setting, especially involving an HTLV-III/LAV-infected person, good handwashing after exposure to blood and body fluids and before caring for another child should be observed, and disposable gloves should be worn when handling such blood and body fluids. Any open lesions on the infected child should also be covered.
6. A plan for periodic review by the medical team described in #1 will be established at the time the initial decision is made regarding school attendance. This periodic review is important because the hygienic practices of a child with HTLV-III/LAV infection may improve sufficiently as he/she matures to allow for school attendance in the future. Alternatively, the hygienic practices may deteriorate if the child's condition worsens and the reevaluation will be necessary to determine if the deterioration of hygienic practices warrants exclusion.
7. Mandatory screening as a condition for school attendance is not warranted based on available data.
8. Persons involved in the care and education of HTLV-III/LAV-infected children should respect the child's right to privacy, including maintaining confidential records. The number of personnel who are aware of the child's condition should be kept at a minimum needed to assure proper care of the child and to detect situations

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Lead Poisoning

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*a working level is that concentration of radon daughters which has a potential alpha energy release of 1.3×10^5 MeV per liter of air.

AIDS Recommendations

(Continued from page 5)

where the potential for transmission may occur (e.g., bleeding injury).

Day-Care Center Attendance

1. A child infected with HTLV-III/LAV should not be allowed to attend day-care centers unless a medical decision can be made that the child does not pose a meaningful risk to others because he/she has poor control of his/her body secretions, displays behavior such as biting, or has any uncoverable, oozing, skin lesions etc. Such decisions are best made using a team approach that should include the child's physician, public health personnel and the child's parent or guardian.
2. Because other infections in addition to HTLV-III/LAV can be present in blood or body fluids, all day-care facilities should adopt routine procedures for handling blood or body fluids. Soiled surfaces should be promptly cleaned with disinfectants such as household bleach (diluted 1 part bleach to 10 parts water). Disposable towels or tissues should be used whenever possible, and mops should be rinsed in disinfectant. Those who are cleaning should wear disposable gloves and avoid exposure of open skin lesions or mucous membranes to the blood or body fluids.
3. A plan for periodic review by the medical team described in #1 will be established at the time the initial decision is made regarding day-care center attendance. This periodic reevaluation is necessary because the hygienic practices of a child with HTLV-III/LAV infection may improve as the child matures and he/she may be able to attend day-care sometime in the future.
4. Mandatory screening as a condition for day-care attendance is not warranted based on available data.
5. Persons involved in the care and education of HTLV-III/LAV-infected children should respect the child's right to privacy, including maintaining confidential records. The number of personnel who are aware of the child's condition should be kept at a minimum needed to assure proper care of the child and to detect situations where the potential for transmission may occur (e.g., bleeding injury).

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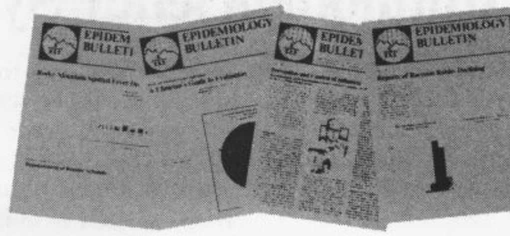


Alternate Sites for HTLV-III Antibody Testing

Alternate Testing Sites

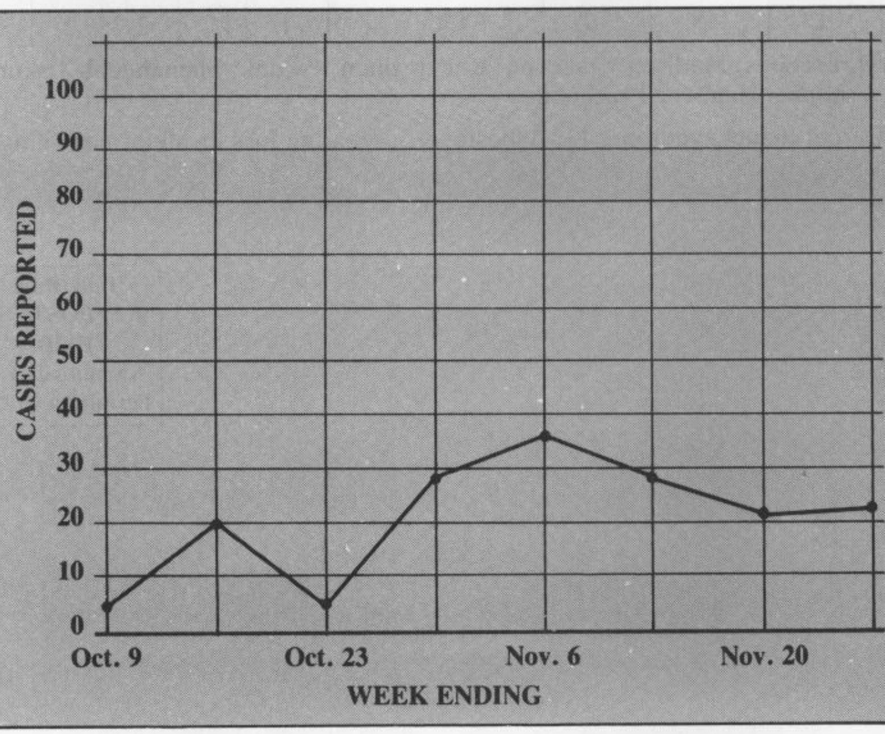
Location	*Testing Facility*
Richmond	Virginia Commonwealth University-MCV Campus Infectious Disease Office Nelson Clinic, Room 212 Richmond, Virginia 23219 Tel: (804) 786-9711 Call for Appointment
Norfolk	Ghent Family Practice 130 Colley Avenue Norfolk, Virginia 23510 Tel: (804) 446-5955 Call for Appointment
Portsmouth	Portsmouth Family Practice 2700 London Boulevard Portsmouth, Virginia 23707 Tel: (804) 397-6344 Call for Appointment
Fairfax	Joseph Willard Health Center 3750 Old Lee Highway Fairfax, Virginia 22030 Tel: (703) 569-6704 Call for Appointment
Roanoke	Roanoke City Health Department 515 Eighth Street, S.W. Roanoke, Virginia 24016 Tel: (703) 981-2636 Call for Appointment

Alternate testing sites were established several months ago using U.S. Public Health Service funds. The purpose for doing this was to make testing for antibody to human T-cell lymphotrophic virus Type III (HTLV-III) available to persons in AIDS risk groups who desire to know their antibody status, since they would threaten the safety of the blood supply if they were to donate blood in order to learn of their antibody status.

The five sites, listed below, have tested 261 individuals, and 50 (19%) of them have been found to be HLTV-III antibody positive. A positive test is defined as two repeatedly positive tests by enzyme immunosorbent assay (ELISA) and a positive Western Blot test. This percentage of positives is consistent with that of the national average for individuals seeking to be tested in alternate testing sites.

Each of the sites provides the following services, free of charge:

- drawing the necessary blood samples;
- performing the screening tests for antibodies to HTLV-III;
- providing pretest counseling about the test;
- informing individuals of their test results;
- ensuring that individuals who test positive receive appropriate counseling;
- ensuring that individuals who test positive are referred to an appropriate source of care for further medical evaluation.



Influenza Surveillance Virginia 1985-86

Surveillance is based on reports from 33 sentinel physicians located throughout the Commonwealth. Influenza activity is judged to be at baseline level. To date, no influenza virus isolates or seroconversions have been reported. Nationally, one virus type A (H3N2) isolation has been reported.

Cases of selected notifiable diseases, Virginia, for the period November 1 through November 30, 1985

Disease	State					Regions				
	This Month	Last Month	Total to Date 1985	Total to Date 1984	Mean 5 Year To Date	This Month N.W.	N.	S.W.	C.	E.
Measles	0	0	27	5	80	0	0	0	0	0
Mumps	4	1	47	18	64	0	0	1	1	2
Pertussis	4	3	21	19	24	1	1	0	0	2
Rubella	0	0	2	0	13	0	0	0	0	0
Meningitis—Aseptic	67	75	379	242	265	6	13	8	17	23
*Bacterial	26	17	234	200	218	6	4	3	4	9
Hepatitis A (Infectious)	24	13	161	102	190	12	2	8	2	0
B (Serum)	60	27	518	460	523	3	8	12	12	25
Non-A, Non-B	15	9	89	83	69	2	3	4	2	4
Salmonellosis	147	128	1489	1197	1413	20	25	29	36	37
Shigellosis	14	7	86	187	411	2	2	1	3	6
Campylobacter Infections	77	77	714	581	393	10	17	15	10	25
Tuberculosis	48	70	416	400	497	3	7	2	9	27
Syphilis (Primary & Secondary)	32	26	283	384	582	1	4	4	9	14
Gonorrhea	1756	1734	17,866	18,204	21,472	—	—	—	—	—
Rocky Mountain Spotted Fever	0	4	25	47	77	0	0	0	0	0
Rabies in Animals	10	20	169	196	356	5	2	2	1	0
Meningococcal Infections	4	4	51	59	78	0	0	2	2	0
Influenza	22	20	998	1123	1721	4	0	0	4	14
Toxic Shock Syndrome	0	0	1	7	8	0	0	0	0	0
Reyes Syndrome	0	0	2	6	10	0	0	0	0	0
Legionellosis	2	4	18	27	25	0	0	0	0	2
Kawasaki's Disease	0	1	26	13	22	0	0	0	0	0
Other: Acquired Immunodeficiency Syndrome	17	8	97	34	—	1	11	2	2	1

Counties Reporting Animal Rabies: Fauquier 2 raccoons; Madison 1 raccoon; Rockingham 1 skunk; Shenandoah 1 skunk; Fairfax 1 raccoon; Loudoun 1 raccoon; Lee 2 skunks; Hanover 1 raccoon

Occupational Illnesses: Pneumoconiosis 20; Carpal tunnel syndrome 13; Asbestosis 5; Hearing loss 2; Metal poisoning 2; Dermatosis 1; Silicosis 1.

*other than meningococcal

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