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Radon In Virginia

The following assessment and recommendations are excerpted from a report issued by the Radon Task Force, members of which were appointed by Virginia Secretary of Human Resources, Eva S. Teig.*

Executive Summary

Radon is a radioactive gas generated by the natural decay of uranium. It is odorless, tasteless, colorless, chemically inert, and highly mobile in the environment. As radon undergoes radioactive decay, it produces new radioactive elements called radon progeny. Radon progeny are solids, chemically active, and electrically charged which allows them to adhere to dust particles in the air or the inner lining of the lungs. Prolonged exposure of miners to high levels of radon progeny in underground mines has caused an increased risk of lung cancer. Since uranium is found in trace amounts throughout the earth's crust, radon can be found in soil or rock and in groundwater which has passed through soil or rock containing radioactive isotopes.

The information available on the concentration of radon or radon progeny in American homes is meager. Collection of data is continuing. The database of measurements of radon or radon progeny in homes in Virginia is also small; nevertheless, indoor radon appears to be a significant problem for some of the citizens of the Commonwealth.

In order to appropriately address this issue, the Radon Study Task Force recommends 1) that reliable information based on current research continue to be made available to the public; 2) that persons living in or purchasing homes in the piedmont and mountain (Blue Ridge and Valley/Ridge) regions of the Commonwealth be encouraged to test their homes for radon and to become knowledgeable on the topic; 3) that other residents of Virginia who are

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**Members included: Maston T. Jacks, Esquire, Deputy Secretary of Human Resources (Chairman); Carl W. Armstrong, M.D., John Einarson, M.D., and Donald W. MacCorquodale, M.D. with the Virginia Department of Health; Stanley S. Johnson, Chief Geologist, Virginia Division of Mineral Resources; Jack A. Proctor with the Department of Housing and Community Development; James E. Sydnor with the Air Pollution Control Board; and Mary Ann Turner, M.D., member of the Virginia Radiation Advisory Board.*

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concerned about the presence of indoor radon have their homes tested; 4) that the results of ongoing radon research be monitored so that risks associated with indoor radon may be better quantified; 5) that home construction techniques being developed to minimize radon exposure be monitored and the results be disseminated to builders, building inspectors, realtors, and other interested parties, statewide; and 6) that the Commonwealth refrain, at this time, from mandating either radon testing prior to real estate transactions or licensing of companies engaged in radon testing or mitigation.

Health Risks Associated with Radon Exposure

Although much has been written about the risks from prolonged exposure to radon, it is important to distinguish between risks which have been estimated and risks which have been observed epidemiologically. Current risk estimates for persons living in homes with radon levels near the EPA action level invoke at least two assumptions. The first is that risk estimates obtained from studies of underground miners can be used for radon exposures in the home. The second is that the observed risks associated with high cumulative doses of radon can be extrapolated (in a linear fashion) to estimate the risk associated with lower cumulative doses to which many home occupants are expected to be exposed. Such extrapolation cannot be done without making a number of scientifically controversial assumptions which need to be validated by current and future epidemiologic research. Assumptions must be made about the duration of exposure; the dose-response relationship (including the presence or absence of a dose threshold below which health effects would not occur); and the influence of individual variations in age, gender, and genetic predispositions.

Much of the fear connected with radiation exposure is due to uncertainty in the estimation of outcome and the period of latency (Adelstein, 1987).

Much of what is known about the health risks associated with exposure to radon and radon progeny comes from analysis of the effects on underground miners of high exposures over long time periods (NAS, 1981). There is considerable controversy over the appropriate application of such data to the general public exposed to radon in their homes alone. Some refer to lung cancer from indoor radon as a "statistical illness" created by multiplying a very small risk by very large populations to give rise to frightening figures (Goldsmith, 1987). Discrepancies between anticipated and actual cases of lung cancer have caused some researchers to propose the possibility of another cancer-causing agent in the mines to account for the discrepancies (Cohen, 1985). All of the miners in these studies were men and most of them were cigarette smokers. Their environment was subject to a variety of airborne particles (NCRP, 1984b).

Radon guidelines recommended by the EPA and other organizations are based on extrapolations of data gathered under circumstances quite different from those found in homes (IDNS, 1986). The data that do exist have been interpreted in different ways. The U.S. EPA has recommended that radon concentrations at or above 4 pCi/l should be lowered. This EPA "action level" applies to annual average exposure under normal living conditions. According to the EPA, projected health effects resulting from 70 years of exposure for 18 hours per day at this level of exposure to radon increases the risk of developing lung cancer by approximately 3 times the normal risk (EPAORD, 1986b).

Currently, the National Cancer Institute and other researchers have begun epidemiologic studies in an effort to more clearly document the link between indoor radon and lung cancer. These studies are being conducted in Maine (600 female cases, 600 controls); in New Jersey (500 female cases, 500 controls); in Pennsylvania (2,000 female cases, 4,000 controls); in Sweden (200 female cases, 400 controls); and in Canada (1,000 male and female cases, 2,000

controls). In most of these studies, only women are being observed since it is likely that women spend more time at home than men and they are less likely to smoke or to be exposed to occupational carcinogens (Goldsmith, 1987). Research of this nature requires extensive amounts of manpower, money, and time. These studies mentioned above will be completed in the next five years (by 1992). The applicability of the findings of such studies will not be restricted to the localities in which they were conducted *i.e.* the findings will be of immense value in assessing the radon problem in Virginia.

In conclusion, a number of statements can be made about the health risk from radon. First, a number of epidemiologic studies have documented an increased risk of lung cancer in miners exposed to radon (NAS, 1981). Few authorities question radon's role in causing cancer in those mining populations. Second, the exact magnitude of the risk to miners is still uncertain. The reasons for this include the fact that the miner studies did not quantitate the radon levels in the mines, or parts of a single mine, over the time period of exposure (doses were estimated retrospectively); miners were exposed to other pollutants in the mines; many miners smoked tobacco products; and miner cohorts studied have not yet been followed for a lifetime, necessitating an estimation of total lifetime lung cancer deaths (those observed to date plus an estimate of future deaths among those still living). Third, based on the studies of miners, it is reasonable to assume that there is a risk associated with prolonged exposure to high levels of radon in the home. The exact magnitude of that risk is, however, unknown at this time. In addition, it is not clear whether or not there is a risk associated with prolonged exposure to low levels of radon in the home; most authorities, however, believe it is prudent practice, in the absence of evidence to the contrary, to assume the risk at low dose exposures is a linear or curvilinear function of the risk at higher dose exposures.

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Flu Reports Increase

Reports of flu-like illness from 30 sentinel physicians increased sharply at the beginning of February, with the greatest activity in the northern, northwestern, and central regions of the State. Most influenza virus isolates in Virginia have been type A (subtype pending), consistent with the predominant subtype being isolated nationally, i.e., A(H3N2). Due to antigenic drift, the A/Leningrad/360/86 component of the current vaccine may not provide optimal protection against the circulating strain, underscoring the importance of the judicious use of the antiviral drug amantadine for the prophylaxis and treatment of high risk patients.

Recommendations of the Immunization Practices Advisory Committee (ACIP) of the U.S. Public Health Service

Enhanced-Potency Inactivated Poliomyelitis Vaccine

The supplementary statement provides information on and recommendations for the use of inactivated poliovirus vaccine (IPV) of enhanced potency.* The Immunization Practices Advisory Committee (ACIP) believes that, in the United States, polio immunization should rely primarily on oral poliovirus vaccine (OPV), with selected use of enhanced-potency IPV as specified in this document. However, this subject should be reviewed on a continuing basis, and an extensive review of polio vaccines and potential vaccine policies will take place during 1988. General recommendations on poliomyelitis prevention, including the use of and schedules for OPV, are found in the current ACIP recommendations (*1*).

Introduction

Conventional IPV. IPV was introduced in the United States in 1955 and was used widely until OPV became available during the period 1961-1964. Thereafter, the use of IPV rapidly declined to a level of less than 1% of all polio vaccine distributed annually in the United States.

In recent U.S. studies, three doses of IPV administered in the first year of life produced antibodies to poliovirus serotypes 1, 2, and 3 in 87%, 97%, and 95% of recipients, respectively. More than 99% of children completing the four-dose primary series by 18 months of age produced antibodies to all three serotypes (*2*).

Enhanced-Potency IPV. A method of producing a more potent IPV with greater antigenic content was developed in 1978 and led to the newly licensed IPV, which is produced in human diploid cells (*3*). Results of studies from several countries have indicated that a reduced number of doses of IPV produced with this technique can immunize children satisfactorily (*4-6*). A clinical trial of two preparations of enhanced-potency IPV was completed in the United States in 1984 (*7*). Children received three doses of one of the enhanced-potency IPVs at 2, 4, and 18 months of age. In spite of the presence of maternal antibodies in the majority of the infants at the time of the first dose, 99%-100% of the children were seropositive for all three poliovirus types at 6 months of age (2 months after their second dose). The percentage of seropositive children did not rise or fall significantly during the 14-month period following the second dose, a result that confirms that seroconversion had occurred in almost all children. Furthermore, geometric mean titers increased 5- to 10-fold following both the second and third doses. Conclusive studies are not yet available concerning antibody persistence following three doses of the enhanced-potency IPV to be made available in the United States. However, unpublished studies of an IPV with lower antigen content have shown 100% seropositivity 5 years after the third dose (*2*).

The effect of enhanced-potency IPV on the circulation of poliovirus in a community has not yet been determined, but it is likely to be at least as good as that seen with conventional IPV. In a recent study of poliovirus excretion following type 1 vaccine-virus challenge after the third dose of enhanced-potency IPV, the decrease in excretion was at least as great as that after conventional IPV, but still significantly less than that found after three doses of OPV (*8*).

Vaccine Usage

Indications. Persons with a congenital immune deficiency disease, such as agammmaglobulinemia; an acquired immune deficiency disease, such as acquired immunodeficiency syndrome (AIDS); or an altered immune status as a result of other diseases or immunosuppressive therapy are at increased risk for paralysis associated with OPV. Therefore, if polio immunization is indicated, these persons and their household members and other close contacts should receive IPV rather than OPV. Although a protective immune response following receipt of enhanced-potency IPV cannot be assured, some protection may be provided to the immunocompromised patient. Available data on children previously diagnosed with asymptomatic human immunodeficiency virus (HIV) infection do not suggest that they are at increased risk of adverse consequences from OPV. However, for such persons, use of IPV rather than OPV is prudent since family members may be immunocompromised because of AIDS or HIV infection and may be at increased risk for paralysis from contact with an OPV virus.

Routine primary poliovirus vaccination of adults (generally those 18 years of age or older) residing in the United States is not recommended. Adults at increased risk of exposure to either vaccine or wild poliovirus (*1*) should receive polio vaccination in accordance with the schedule prescribed below.

In households where polio vaccine is to be administered to immunologically normal children, ACIP recommends giving OPV regardless of the poliovirus-vaccine status of adult household contacts (*1*). The overall risk of vaccine-associated paralytic disease in immunologically normal contacts of OPV recipients is one case per 5.5 million doses of OPV distributed (*9*). As an alternative, adult contacts can first complete their primary series of polio vaccine as detailed in the schedule below, if there is strong assurance that subsequent immunization of the child will not be jeopardized or unduly delayed.

Schedules. The primary series for enhanced-potency IPV consists of three 0.5-mL doses administered subcutaneously. The interval between the first two doses should be at least 4 weeks, but preferably 8 weeks. The third dose should follow in at least 6 months, but preferably nearer to 12 months. A primary series can be started as early as 6 weeks of age, but preferably at 2 months of age. Although studies have not been conducted, young children should receive the third dose along with diphtheria, tetanus, pertussis vaccine (DTP) and measles, mumps, rubella vaccine (MMR) at 15 months of age, if possible.

**Poliovirus Vaccine Inactivated, which is manufactured by Connaught Laboratories Ltd., will be distributed by Connaught Laboratories Inc. beginning in March 1988.*

A primary series of polio vaccine usually consists of enhanced-potency IPV alone or OPV alone. However, a combination of both vaccines totalling three doses and separated by appropriate intervals constitutes a primary series. If enhanced-potency IPV is administered to persons with a previously incomplete series of conventional IPV, a final total of four doses of polio vaccine is necessary for a primary series.

All children who received a primary series of enhanced-potency IPV or of a combination of polio vaccines should be given a booster dose before entering school, unless the final dose of the primary series was administered on or after the fourth birthday. The need for routinely administering additional doses is unknown at this time.

For unvaccinated adults at increased risk of exposure to poliovirus, a primary series of enhanced-potency IPV is recommended. While the responses of adults to a primary series have not been studied, the recommended schedule for adults is two doses given at a 1- to 2-month interval and a third dose given 6 to 12 months later. If less than 3 months but more than 2 months are available before protection is needed, three doses of enhanced-potency IPV should be given at least 1 month apart. Likewise, if only 1 to 2 months are available, two doses of enhanced-potency IPV should be given at least 1 month apart. If less than 1 month is available, a single dose of either OPV or enhanced-potency IPV is recommended.

Adults who are at increased risk of exposure and have had 1) at least one dose of OPV, 2) fewer than three doses of conventional IPV, or 3) a combination of conventional IPV and OPV totalling fewer than three doses should receive at least one dose of OPV or enhanced-potency IPV. Additional doses needed to complete a primary series should be given if time permits.

Adults who are at increased risk of exposure and who have previously completed a primary series with any one or combination of polio vaccines can be given a dose of OPV or enhanced-potency IPV.

Side Effects and Adverse Reactions. Available data indicate that the rate of adverse reactions in the kidney cells of monkeys receiving enhanced-potency IPV are low and that the reactions are not different from those following administration of a placebo. The recently licensed human diploid cell-derived vaccine was not compared to a placebo. Rates of local adverse events following its use are similar to rates found in controlled studies using vaccine derived from the kidney cells of monkeys. There is no evidence that conventional IPV causes any serious side effects. Consequently, serious side effects are not expected to occur with enhanced-potency IPV. This conclusion can be confirmed only with postmarketing surveillance. Parents of children receiving the vaccine, older vaccine recipients, and health-care providers are encouraged to report all adverse events occurring within 4 weeks of receipt of enhanced-potency IPV to the manufacturer and to local or state health departments. The information will be forwarded to the appropriate federal agency.†

Precautions and Contraindications. Vaccine administration should not be postponed because of minor illnesses, such as mild upper-respiratory infections. Generally, however, persons with severe febrile illnesses should not be vaccinated until they have recovered.

The enhanced-potency IPV may contain trace amounts of streptomycin and neomycin. Persons who have had anaphylactic reactions to topically or systemically administered streptomycin and neomycin should not receive enhanced-potency IPV.

There is no convincing evidence documenting adverse effects of conventional IPV on the pregnant woman or developing fetus. Data on adverse events following use of enhanced-potency IPV are not available. On theoretical grounds, it is prudent to avoid vaccinating pregnant women. However, if a pregnant woman needs immediate protection against poliomyelitis, OPV is recommended.

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†*Center for Biologics Evaluation and Research, Food and Drug Administration, or the Centers for Disease Control.*

Have an Idea for the *Bulletin?*

The editor welcomes any reports of cases, outbreaks, or public health problems of interest to the Bulletin*'s readers. Such accounts and any other comments or suggestions regarding the* Bulletin *should be addressed to: Editor,* Epidemiology Bulletin, *Office of Epidemiology, Room 700, 109 Governor Street, Richmond, Virginia 23219.*

Cases of selected notifiable diseases, Virginia, for the period January 1, through January 31, 1988.

Disease	State					Regions				
			Total to Date		Mean 5 Year To Date	This Month				
	This Month	Last Month	1987	1988		N.W.	N.	S.W.	C.	E.
Measles	0	0	0	0	0	0	0	0	0	0
Mumps	3	8	0	3	3	0	3	0	0	0
Pertussis	1	6	13	1	4	1	0	0	0	0
Rubella	0	0	0	0	0	0	0	0	0	0
Meningitis—Aseptic	7	19	11	7	16	1	3	0	1	2
*Bacterial	6	27	12	6	22	3	0	1	0	2
Hepatitis A (Infectious)	10	23	25	10	14	0	6	0	2	2
B (SERUM)	15	43	42	15	40	4	5	3	1	2
NON-A, NON-B	2	7	5	2	7	0	0	0	1	1
Salmonellosis	72	113	73	72	72	8	15	11	17	21
Shigellosis	35	34	14	35	16	5	18	4	1	7
Campylobacter Infections	28	50	29	28	29	2	6	3	9	8
Tuberculosis	23	61	23	23	12	1	6	6	4	6
Syphilis (Primary & Secondary)	26	21	23	26	39	2	6	1	12	5
Gonorrhea	1230	1021	1562	1230	1572	0	0	0	0	0
Rocky Mountain Spotted Fever	0	1	0	0	0	0	0	0	0	0
Rabies in Animals	13	23	18	13	19	3	3	2	4	1
Meningococcal Infections	4	5	10	4	6	3	1	0	0	0
Influenza	16	14	826	16	178	5	2	2	2	5
Toxic Shock Syndrome	0	0	0	0	0	0	0	0	0	0
Reye Syndrome	0	0	0	0	0	0	0	0	0	0
Legionellosis	0	1	1	0	1	0	0	0	0	0
Kawasaki's Disease	0	6	0	0	2	0	0	0	0	0
Acquired Immunodeficiency Syndrome	18	45	19	18	—	0	7	0	3	8

Counties Reporting Animal Rabies: Augusta 1 raccoon; Botetourt 1 skunk; Fairfax 1 fox; Hanover 1 raccoon; Henrico 2 raccoons; New Kent 1 raccoon; Prince William 2 raccoons; Rockingham 1 raccoon; Shenandoah 1 skunk; Washington 1 skunk; Westmoreland 1 fox.

Occupational Illnesses: Asbestosis 12; Carpal tunnel syndrome 4; Dermatitis 3; Loss of Hearing 6; Pneumoconioses 25.

*other than meningococcal

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