APPENDIX G
HUMAN HEALTH

The material in this appendix was taken directly from appendix D of the LANL SWEIS. Only the section and table numbering was change.

G.1 PUBLIC HEALTH CONSEQUENCES: PRIMER AND RECENT STUDIES NEAR LANL

In this appendix, supplemental information is presented on the effects on human health of radioactive and chemical exposures. The information is presented in two sections: that addressing our general knowledge and understanding (section G.1.1) and that presenting in more detail the findings of the recent studies of public health in the community of Los Alamos, and New Mexico and U.S. studies (including Native Americans in New Mexico, Hispanic white and nonhispanic white populations throughout the U.S. (section G.1.2). The presentation in section G.1.1 is useful to the reader as a primer on human health effects of exposures to radioactivity or to chemicals. The summaries presented in section G.1.2 are the results of descriptive epidemiology studies. That is, they are analyses of disease incidence rates and causes of death using statistical analytical methodologies.

Exposure to toxic chemicals is regulated by other agencies, and DOE subscribes to and applies those regulations without change to its own activities. The Occupational Safety and Health Administration (OSHA) promulgates and enforces regulations for the protection of workers, and EPA regulates exposures to the public. Chapter 7 provides a detailed review of the regulatory requirements for the operation of LANL.

G.1.1 Primer on Human Health Consequences of Radiological and Chemical Exposures

Table G.1.1–1 summarizes the differences in consequences between exposures to radioactive materials and exposures to chemicals. More detailed information on the modes of exposure and potential effects of these exposures are given in the sections below.

G.1.1.1 *About Radiation and Radioactivity*

In the simplest sense, radiation is defined as energy propagated through space (NBS 1952). This definition covers a broad range, including visible light, radio and television transmissions, microwaves, and emissions from atomic and nuclear reactions and interactions. The method by which radiation interacts with matter is by transferring its energy to the atoms of the matter. The amount of energy transferred determines the effect that it will have on matter. The broad spectrum of radiation can be subdivided into two groups, ionizing and nonionizing. Ionization occurs when the radiation transfers enough energy to strip one or more electrons from the interacting atom. When ionization takes place in the body, it can cause chemical and physical changes that are of concern to human health. Radiation that does not have enough energy to strip electrons is called “nonionizing.”

Ionizing radiation is used in a variety of ways, many of which are familiar to us in our everyday lives. The machines used by doctors to diagnose and treat medical patients typically use x-rays, which is one form of ionizing radiation. The process by which a television displays a picture is by ionizing coatings on the inside of the screen with electrons. Most home smoke detectors use a small source of ionizing radiation to detect smoke particles in the room’s air.

Ionizing radiation is generated through many mechanisms. The two most common mechanisms are the electrical acceleration of atomic particles such as electrons, as in x-ray machines, and the emission of energy from nuclear reactions in atoms. This second process is termed “radioactive decay.” Atoms are made up of various combinations of particles called protons, neutrons, and electrons. In most cases, the numbers of neutrons and protons are balanced such that the atom will stay together

TABLE G.1.1–1.—*Comparison of Consequences of Radioactivity and Toxic Chemicals*

	RADIOACTIVE MATERIALS	TOXIC CHEMICALS
Threshold for effects?	Assume no threshold (stochastic effects).	Yes, and different thresholds for different effects.
Accumulative effects?	Assumed exposures accumulate over a lifetime, with no repair.	Typically, the body repairs itself between exposures; may build sensitive allergic reaction or interact with cells.
Sensory perception?	We do not feel, smell, or otherwise sense ionizing radiation.	Very low concentrations not sensed. Often an annoying odor and irritating effects at low concentrations. Some gases are visible when in high concentrations.
Carcinogenic?	All ionizing radiation is regulated as carcinogenic.	Only some chemicals are confirmed human carcinogens. Some others are suspected, and some are animal (mammal, or closer to human, primate) carcinogens.
Effects-exposure relationship?	Usually treated as linear at low doses, although this is a conservative simplification (BEIR V 1990).	Typically nonlinear and nonadditive. Thresholds exist. For some chemicals, effects can be treated as linear with exposures, but only over small ranges. Synergisms among chemicals are not understood.
Acute effects?	Acute deterministic effects are soon observed, but occur only above a threshold of about 50 rem (less for the eye).	Effects may be immediately observed for levels of exposures above the thresholds.
Entry paths of particulates into the body?	Radionuclides enter through inhalation, ingestion, and wounds. A few are absorbed through the skin.	Same routes, except a greater percentage of chemicals than of radionuclides are absorbed through the skin.
Target organs?	The chemistry of the radionuclide determines its residence time and location in the body.	Same as for radionuclides. Except, the body also metabolizes chemicals, sometimes into more toxic chemicals.
Penetrating?	Alpha and beta radiation do not penetrate skin. In contrast, dense materials are needed to shield against gamma and x-ray radiation.	About 20% of OSHA-regulated chemicals have skin as an import route of entry. Only corrosive chemicals penetrate protective gear rapidly.

forever. An atom formed with too many of either the neutrons or protons will attempt to change itself into a more stable form. To do this, the atom will emit an atomic particle, such as an electron, normally called a beta particle, or a "packet" of energy called a photon. This is the process of radioactive decay. The time that it takes for the atom to decay is characterized by a value called the half-life. This is the time it takes for a quantity of radioactive material to decay to one-half its original amount. In general, radioactive materials are identified by their half-lives and the type and energy of their emissions. In some cases, atoms may emit a highly energetic, ionized, helium atom, called an alpha particle. The energy carried away by these emissions is normally capable of creating a large number of ionizations in matter.

Besides ionization, other particles can often be emitted during interactions between radiation and matter, depending upon the type and energy of the interaction. Neutrons, protons, and some other more exotic particles are often emitted during various processes. Nuclear reactors use neutrons to break apart, or fission, particular isotopes of uranium and plutonium in order to release heat and more neutrons to continue the reaction. Large machines, often called "atom smashers," cause atoms at high energies to collide and break apart, releasing particles in order to study their nuclear structure. However, due to the design and operation of these types of facilities, it would be highly unlikely for these types of radiations to reach the public outside the boundaries of the facility.

When an individual is in the presence of an unshielded radiation source, this is referred to as being exposed. The amount of ionizing radiation that the individual receives during the exposure is referred to as dose. The measurement of radiation dose is called radiation dosimetry, and is done by a variety of methods depending upon the characteristics of the incident radiation. The units of measure for radiation doses are normally rads and rem. (Note that the term millirem [mrem] is also used often. A millirem is one one-thousandth of a rem.) The rad is a measure of the energy deposited in the body by the radiation, regardless of the type of emission. The rem is a measure of the biological effect, by including the effectiveness of the particular type and energy of the incident radiation for causing biological effects. This is due to the fact that some heavier or higher energy radiations, such as alpha particles or neutrons, can deposit their energy into much smaller volumes, and consequently, cause more intense damage through localized, chemical changes.

When an individual is exposed to an unshielded radiation source, this is called external radiation. If radioactive material is incorporated into the body and consequently decays, it is called internal radiation. The external radiation is measured as a value called the deep dose equivalent (DDE). Internal radiation is measured in terms of the committed effective dose equivalent (CEDE). More information about the CEDE is presented in the discussion about the processes by which radioactive material enters the body. The sum of the two contributions (DDE and CEDE) provides the total dose to the individual, called the total effective dose equivalent (TEDE). Often the radiation dose to a selected group or population is of interest, and is referred to as the collective dose equivalent, with the measurement units of person-rem.

G.1.1.2 *About Radiation and the Human Body*

Ionizing radiation affects the body through two basic mechanisms. The ionization of atoms can generate chemical changes in body fluids and cellular material. Also, in some cases the amount of energy transferred can be sufficient to actually knock an atom out of its chemical bonds, again resulting in chemical changes. These chemical changes can lead to alteration or disruption of the normal function of the affected area. At low levels of exposure, such as the

levels experienced in occupational or environmental settings, these chemical changes are very small and ineffective. The body has a wide variety of mechanisms that repair the damage induced. However, occasionally, these changes can cause irreparable damage that could ultimately lead to initiation of a cancer, or changes to genetic material that could be passed to the next generation. The probability for the occurrence of health effects of this nature depends upon the type and amount of radiation received, and the sensitivity of the part of the body receiving the dose.

At much higher levels of exposure, at least 10 to 20 times higher than the legal limits for occupational exposures, the body is unable to recover from the large amount of chemical changes occurring during the exposure. At these levels, damage is much more immediate, direct, and observable. Health effects range from reversible changes in the blood to vomiting, loss of hair, temporary or permanent sterility, and other changes leading ultimately to death at exposures above about 100 times the regulatory limits. In these cases, the severity of the health effect is dependent upon the amount and type of radiation received. Exposures to radiation at these levels are quite rare, and, outside of intentional medical procedures for cancer therapy, are always due to accidental circumstances.

For low levels of radiation exposure, the probabilities for induction of various cancers or genetic effects have been extensively studied by both national and international expert groups. The problem is that the potential for health effects at low levels is extremely difficult to determine without extremely large, well-characterized exposed populations. Therefore, only particular groups with fairly high exposures, such as atomic bomb survivors, radiation accident victims, and some groups receiving large medical exposures, can be studied to evaluate the probabilities. Unfortunately, the levels and rates of exposures, and the conditions under which they occurred, are very different from those in which the normal population is exposed to background radiation or to normal operational releases from nuclear operations. Therefore, expert groups must make significant approximations and assumptions in order to apply the study results to the lower levels of exposure. This is done in a manner that attempts to ensure that the resulting risk factors are conservative estimates of the actual probabilities. In other words, it is unlikely that the actual risks are greater than the estimates, while it is fairly likely that the actual risk is smaller than the estimate.

There is another type of study, referred to as an epidemiology study, that attempts to estimate the risk factors in populations with much lower doses than mentioned above. These studies are even more difficult to perform. There are two types of epidemiology studies: descriptive (based on statistical analyses of death and disease incidences) and analytical (case studies and observational analysis within a community or work force). The studies summarized in section G.1.2, are descriptive. The risk factors for radiation-induced cancer at low levels of exposure are very small, and it is extremely important to account for the many nonradiation related mechanisms for cancer induction, such as smoking, diet, lifestyle, and chemical exposures. These multiple factors also make it difficult to establish cause-and-effect relationships that could attribute high or low cancer rates to specific initiators. As a consequence, the results of such studies have not been generally accepted within the scientific community and are not currently used as the primary basis for establishing the risk factors.

Risk factors are estimated for a large number of fatal and nonfatal cancers, for hereditary effects, and a few other identified radiation-induced health effects. Table G.1.1.2–1 lists the fatal cancer risk factors used in this SWEIS, which are based upon the recommendations of a recognized authoritative international expert group, the International Commission on Radiological Protection (ICRP). The other,

smaller risk factor in the table for nonfatal cancer and hereditary effects may be similarly applied by interested readers.

In keeping with the previous discussion of the difficulties in determining the risk factors used in this document, it is worthwhile to discuss the level of confidence that is associated with those factors. The ICRP, in the recommendation that established the risk factors used here, stated that, "The nominal values of fatal cancer risk, which form the basis of the detriment following radiation exposure, are not to be regarded as precise and immutable. They are, unfortunately, at this time still subject to many uncertainties and to many assumptions involving factors which may be subject to change. ...It is hoped, and indeed expected, that these uncertainties will diminish in the future as the accumulated experience in exposed populations such as the Japanese survivors increases and as more information develops from a broader variety of human experiences" (ICRP 1991). The Committee on the Biological Effects of Ionizing Radiations (BEIR), which developed the risk factors that the ICRP recommends, also discussed the uncertainty of the factors: "Finally, it must be recognized that derivation of risk estimates for low doses and dose rates through the use of any type of model involves assumptions that remain to be validated. ...Moreover, epidemiologic data cannot rigorously exclude the existence of a threshold in the millisievert (1 millisievert = 100 millirem) dose range. Thus the background radiation cannot be ruled out. At such low doses and dose rates, it must be acknowledged that the lower limit of the range of uncertainty in the risk estimates extends to zero" (BEIR V 1990).

Given these concerns, the reader should recognize that these risk factors are intended to provide a conservative estimate of the potential impacts to be used in the decision-making process, and are not necessarily an accurate representation of actual anticipated fatalities. In other words, one could expect that the stated impacts from an activity or accident form an envelope around the situation, and that actual consequences could be less, but probably would not be worse.

When considering the risks from exposure to ionizing radiation, it is important to remember that we are always being exposed to the radiation in the environment around us. Natural background radiation is the collective term for all of the sources that occur naturally, such as cosmic radiation and naturally occurring radioactive materials, such as potassium, uranium, thorium, radium, and others. These sources contribute an average of 0.3 rem per year to each individual. Manufactured radiation sources contribute another 0.06 rem per year on

TABLE G.1.1.2–1.—*Risk Factors for Cancer Induction and Heritable Genetic Effects from Exposure to Ionizing Radiation*

EXPOSED POPULATION[a]	FATAL CANCER[b]	NONFATAL CANCER	HEREDITARY EFFECTS (SEVERE)[d]	TOTAL DETRIMENT
Adult Workers	0.0004[c]	0.00008	0.00008	0.00056
Whole Population	0.0005[c]	0.0001	0.00013	0.00073

[a] The distinction between the worker risk and the general public risk is attributable to the fact that sensitivities vary with age, general health, and other factors that contribute more to the general population than to the worker population.
[b] When applied to an individual, units are lifetime probability of excess cancer fatalities per rem of radiation dose. When applied to a population of individuals, units are excess numbers of fatal cancers per person-rem of radiation dose.
c This is the source of the 4×10^{-4} worker and 5×10^{-4} public risk factors used in this SWEIS.
[d] Heritable genetic effects as used here apply to populations, not individuals. For the other columns, the units would change accordingly, in terms of number of effects per unit dose.
Source: ICRP 1991

the average, with the majority coming from medical procedures. Fallout from the atmospheric testing of nuclear weapons currently contributes less than 0.001 rem per year to our doses (NCRP 1987).

G.1.1.3 *About Radioactive Material Within the Body*

Typically, radioactive material that is released into the environment is in the form of very fine particulates, gases, or liquids. That is usually because these forms are the hardest to contain in a facility. This material is easily carried into and spread around the air, soil, and water. As these materials move through the environment, it is possible for them to be taken into the body, through breathing, eating, or drinking. During normal operations of a facility, every effort is made to minimize these releases to levels well below natural background. During accidents, it is possible that higher levels may be released; but, the facilities are designed and operated to control these releases as much as possible.

Radioactive material normally enters the body through one of three mechanisms. When the material is in the air, it is inhaled into the lungs, where a fraction will be trapped, depending upon the size of the particles. When it is ingested by eating or drinking, or by clearing of the respiratory tract, it passes through the stomach and into the gastrointestinal tract. Under the right conditions, it can also be absorbed through the skin or enter through open wounds.

Once in the body, the fate of the material is determined by its chemical behavior. Some material will be dissolved into bodily fluids and transferred into various organs of the body. Remaining material may either be retained at its point of entry, such as in the lungs, or pass through the body rapidly, as in the gastrointestinal tract. The effect of material in the body is characterized by the type of radiation it delivers and the organs in which it tends to collect. The rate at which the material is removed from the body is represented by a value called effective biological half-life (the time it takes for the activity in the body to be reduced to one-half as a consequence of radioactive decay and biological turnover of the radionuclide).

When radioactive material is in the body, it irradiates the living tissue around it. Some radiation types, like beta and alpha particles, are much more effective at causing changes when inside the body than when outside. This is because these types of radiation cannot effectively penetrate the dead layer of the skin from an external source. As mentioned above, the radiation dose from material inside the body is called the CEDE. Remember that the dose from an external source stops when you walk away or are shielded from it. But you cannot walk away from an internal source. Therefore, the CEDE is designed to determine the risk commitment from the intake. It is the dose that will be received over the next 50 years from the material in the body. Because of the assumptions that doses are cumulative and their effects are not repaired, this means that the lifetime risk from an internal source in rem CEDE can be directly compared to the risk from an external source in rem DDE.

G.1.1.4 *About the Material of Interest at LANL*

LANL has a large involvement in nuclear science and applications. Therefore, there are many types of radioactive material and radiation sources in use. However, many of the uses require only very small amounts of material. Note that all radioactive materials are considered in this SWEIS; but, there are three types that tend to dominate the human health effects and DOE accident scenarios. This is due to either their particular radioactive and biological characteristics, the quantities of material being used, or the potential for

dispersion in an accident. These materials are plutonium, uranium, and tritium.

Plutonium is a man-made element that has several applications in weapons, nuclear reactors, and space exploration. There are several types of plutonium atoms, called isotopes, which are distinguished by the different numbers of neutrons in their nucleus. (Note that isotopes of a particular atom all behave the same chemically.) In most cases, the isotopes of plutonium of interest here decay by alpha particle emission with radioactive half-lives ranging from tens to thousands of years. There is nothing unique about plutonium as a health risk compared to other radioactive materials. It is only that once incorporated into the body, it tends to stay for a very long time and deposits a lot of localized energy due to its alpha particles.

Uranium is a naturally occurring radioactive element. The discovery that an atom of uranium could be fissioned with neutrons was the starting point of the Nuclear Age. Uranium-235 is one of several fissile materials that fission with the release of energy.

Various applications require the use of different isotopes of uranium. Because isotopes cannot be chemically separated, processes have been developed to enrich uranium to various isotopic ratios. Enriched uranium is uranium that is enhanced in the isotope uranium-235 above its natural ratio of 0.72 percent. Highly enriched uranium (HEU) is where the uranium-235 content is 20 percent or greater. Depleted uranium (DU) is where the content of uranium-235 is below its natural value. Obviously, natural uranium is where the material is in its natural isotopic ratios.

Most uranium isotopes of interest here have very long half-lives and are alpha emitters. Their half-lives are much longer than the plutonium isotopes, and as a result uranium is generally of lower radiological concern than plutonium. However, its actual radiological concern varies with its enrichment. As a heavy metal, uranium also can be chemically toxic to the kidneys. Depending upon the enrichment and chemical form, either chemical or radiological considerations will dominate.

Tritium is a radioactive isotope of hydrogen. It is generated at low levels in the environment by interactions of cosmic radiation with the upper atmosphere, but for practical applications it is normally produced in a nuclear reactor. Tritium has a half-life of around 12 years and decays by emitting a low energy beta particle. Because tritium is an isotope of hydrogen, it can be incorporated into the water molecule, forming tritiated water. In the environment, tritium is most often found either in its elementary form as a gas, or as water. Tritiated water is a significant concern to the human body because the body is composed mostly of water. This actually is a mixed blessing. Tritiated water will easily and rapidly enter the body and irradiate it rather uniformly; however, it also is removed from the body rather quickly, being easily displaced with regular water and with a biological half-life of about 12 days under normal conditions.

G.1.1.5 *How DOE Regulates Radiation and Radioactive Material*

Radiation doses to workers and the public and the release of radioactive materials are regulated by DOE for its contractor facilities. Under the conditions of the *Atomic Energy Act* (as amended by the *Price-Anderson Amendments Act of 1988)*, DOE is authorized to establish federal rules controlling radiological activities at DOE sites. The act also authorizes DOE to impose civil and criminal penalties for violations of these requirements. Some activities are also regulated through a DOE Directives System that uses contractual means to regulate the contractor activities.

Occupational radiation protection is regulated by the *Occupational Radiation Protection Rule*,

Title 10 of the Code of Federal Regulations, Part 835 (10 CFR 835). Environmental radiation protection is currently regulated contractually with DOE Order 5400.5, which is in the process of being converted to a rule. There is a process by which these regulations are developed. The EPA, working with other agencies such as DOE and the NRC, develops a federal guidance document that is signed by the President (52 *Federal Register* [FR] 2822–2834). This document is based upon the recommendations of the National Council on Radiation Protection and Measurements (NCRP), and considers recommendations of international expert groups such as the ICRP. This federal guidance then becomes the basis for all federal regulations for radiation protection, including DOE's and also U.S. Nuclear Regulatory Commission (NRC) rules. This process ensures a common, scientifically based approach to all radiation protection in the U.S.

G.1.1.6 *About Chemicals and Human Health*

The characteristics and consequences of exposures to chemicals are quite different from those of exposure to ionizing radiation. Table G.1.1–1 summarizes the differences.

For noncarcinogens, there are threshold concentrations that must be exceeded for observable adverse effects to happen; whereas, for ionizing radiation it is assumed that the integrated (accumulated) exposure determines the likelihood of observable effects.

The threshold values for effects from toxic chemicals vary somewhat among individuals, but values can be determined that represent most of the more vulnerable people among the general population. The several different effects from a chemical each have different thresholds. For instance, there may be different concentrations that produce odor, irritation, effects that last only a short time, permanent effects, and death. Older and ill people, and those with a particular sensitivity such as respiratory problems, are more vulnerable and will have lower thresholds for effects.

Using human inhalation of chlorine in illustration, 0.2 to 0.4 parts per million (parts of chlorine per million parts of air) is the odor threshold; 1 to 3 parts per million for periods less than an hour produce burning eyes, scratchy or irritated throat, and headache; 15 parts per million is the lowest concentration observed to cause respiratory distress; no deaths were observed in any animals exposed to 50 parts per million for 30 minutes; and 210 parts per million has been estimated to be the 30-minute LC50 for humans, although 50 parts per million might cause death in some vulnerable individuals. (The 30-minute LC50 is defined as the concentration that produces 50 percent fatalities among individuals exposed for 30 minutes.)

The ability to resist a potential effect and to recover from that effect clearly depends upon a person's health and age. For the population of workers, presumed to have few individuals who are especially vulnerable, regulatory agencies set permissible exposure limits and average concentrations for the 8-hour and 10-hour work day. Lower values than these would be appropriate to public exposures; whereas, higher values are deemed acceptable for military personnel under military exigencies.

Again using inhalation of chlorine gas in illustration, the OSHA permissible exposure limit is a time-weighted average (TWA) over the 8-hour work day of 0.5 parts per million[1]. There also is an OSHA short-term exposure limit of a 1-part-per-million 15-minute TWA that should not be exceeded at any time during the work day. The immediately dangerous to life and health (IDLH) value is 30 parts per million; this is the concentration from which a

1. The definition of the TWA is the sum of all the instantaneous air concentrations over the 8 hours, averaged by dividing by the 8 hours.

worker could escape within 30 minutes without a respirator and without escape-impairing or irreversible effects.

This SWEIS analysis uses the TWA as a convenient measure for screening the chemical inventory at LANL, and then uses Emergency Response Planning Guidelines (ERPGs) or their surrogate Temporary Emergency Exposure Limits (TEELs) for bounding the consequences to persons exposed to a release to the atmosphere. ERPGs are provided by the American Industrial Hygiene Association (AIHA) for planning for emergencies, rather than for determining consequences. ERPG–1, ERPG–2, and ERPG–3 are defined and described in detail in appendix G, Accident Analysis. They are intended to provide protection for most members of the public, and so their exposure time (up to one hour) and their concentrations are directly related to effects (no safety factor of ten was applied).

Again using chlorine in illustration, the ERPG–2 is 3 parts per million, the concentration at which nearly all individuals could be exposed without irreversible or other serious health effects or impairment of ability to take protective actions. The ERPG–3 is 20 parts per million, below which nearly all individuals could be exposed without life-threatening effects.

Only for some chemicals and only for a limited extent, effects are directly related to the product of the concentration and length of exposure ("Haber's Law"). Chlorine is not such a chemical. When attempting to apply an existing guideline to a different exposure period than for which the guideline applies, toxicologists must be consulted, and they will consider actual effects data.

G.1.1.7 *How Toxic Chemicals Affect the Body*

Some toxic chemicals can have direct effects upon the eyes and the skin through contact and can enter the body by absorption through the skin. These are considered in the derivation of guides and limits for airborne concentration. Toxic chemicals also can enter the body via ingestion (eating and drinking). All the LANL accidents considered in the SWEIS that pose significant risk to the public produce their exposure through airborne releases, and so airborne concentrations guides and limits are used in the screening and consequence analyses.

After intake, the chemical may follow primarily one or more routes within the body, involving the respiratory system and digestive system, the blood circulatory system, and the urinary tract. The route and residence time before excretion is strongly determined by the chemical's solubility, and if particulate, by its particle size. The chemical may be metabolized, usually in the liver, into other chemicals that are either more or less toxic. For carcinogens, the principal target organs (i.e., where the effects primarily occur) are the respiratory tract, urinary bladder, and to a lesser extent the bone marrow, gastrointestinal tract, and liver.

G.1.1.8 *About Chemical Carcinogens*

Some chemicals are regulated as carcinogens because they or their metabolites may cause cancer. There are limited data on chemical carcinogens for humans, and there are problems with applying the results of animal studies to humans. Therefore, these chemicals are classified as known human carcinogens, potential or suspected carcinogens, and chemicals that cause cancer in animals. Exposure to chemical carcinogens is treated in the same manner as cumulative exposure to ionizing radiation; that is, exposures are assumed to be additive in producing cancer.

Some chemicals are carcinogenic at concentrations that do not produce observable effects from acute (short-term) exposures. For these, the airborne exposure limits and guidelines are based on their carcinogenicity. Some chemicals may produce an irreversible change to cells (tumor initiation), which then may be submitted to chemicals that are promoters of cancer. Such promoters must be given repeatedly to be effective. For this reason, chemical carcinogens are regarded as additive to one another, and individual chemicals are regulated at 1/100 of the exposure level regarded as hazardous, perhaps to account for the conservative possibility of having 100 such chemicals in one's environment.

The carcinogenic effects of certain chemicals are similar to those of ionizing radiation and have been noted in virtually every organ, depending on the chemical, the species, and conditions of exposure. The cancers induced by chemicals and by ionizing radiation cannot be distinguished from cancers induced by other causes. Therefore, the effects of chemicals and ionizing radiation are inferred only on a statistical basis, and must inferred from exposures at higher doses and dose rates. The choice of model has a large influence on the estimated excess cancer risk. The extrapolation is made by assuming an uncertain and controversial no-threshold, linear mathematical relationship between dose and resultant effects. This model is usually thought likely to overestimate the risk at low doses, and so is often said to estimate the "upper limit" of risk (NCRP 1989).

Chemicals vary widely in their capacity to induce cancer. There are even fewer data on the carcinogenic effects for chemicals than for radiation. With most chemicals, assessment of risks for humans must be based on extrapolation from laboratory animals or other experimental systems. Hence, the risk assessment for chemicals has even more uncertainty than risk assessment for ionizing radiation (NCRP 1989). Ultimately, the desired certainty in risk assessment at low-level exposures to chemicals and radiation will require better understanding of their effects at all stages of carcinogenesis.

The EPA, in setting standards for compliance with the *Clean Air Act*, is required by judicial decision and the *Clean Air Act* to determine a "safe" level with an "ample margin of safety to protect public health" without consideration as to cost or technology feasibility (Bork 1987). After that level is determined, costs and feasibility can be considered in setting the standard. Although this decision applied specifically to vinyl chloride and the *Clean Air Act*, it aids in understanding the EPA challenge faced in determining what is "safe," "adequate," or "acceptable" when setting standards for protection of workers, public, and environment. In the attempt to provide an objective context for evaluating the risks posed by LANL operations, the SWEIS authors have searched for authoritative statement on acceptable risk levels. A few such statements and inferences can be found in ICRP, NCRP, EPA, and OSHA documents.

EPA regulations provide goals for environmental remediation (cleanup). The EPA goals "for acceptable exposure levels to known or suspected carcinogens are generally concentration levels that represent an excess upper bound lifetime cancer risk between 10^{-4} and 10^{-6}. The 10^{-6} risk level shall be used as the point of departure for determining remediation goals" when existing and relevant requirements are not available or sufficiently protective because there are multiple contaminants or pathways. When the combined risk from multiple contaminants exceed 10^{-4}, then factors such as detection limits and uncertainties may be considered in determining the cleanup level to be attained (40 CFR 300.430). Note that this is the lifetime risk to an undetermined public population group.

OSHA (OSHA 1997) expressed that its proposed worker permissible exposure limit for methylene chloride of 25 parts per million

(average for 8 hours per day) would entail an employment lifetime risk of 3.62×10^{-3}, and that this was "clearly well above any plausible upper boundary of the significant risk range defined by the Supreme Court and used by OSHA in its prior rulemaking." OSHA noted that typical lifetime occupational risk for all manufacturing industries is 1.98×10^{-3}, and that the risk in occupations of relatively low risk, like retail trade, is 8.2×10^{-4}. Note that worker risk is generally accepted at a higher level than public dose because it is an accepted risk of employment. This is compatible with the EPA upper bound lifetime public cancer risk of between 10^{-4} and 10^{-6}.

G.1.1.9 *Radionuclides and Chemicals of Interest at LANL*

LANL has used, uses, and will use a wide variety of chemicals because of its research mission. LANL has a chemical database that tracks the quantity and location of chemicals on site. About 51 of the chemicals tracked in the database are carcinogenic. A large number of the chemicals tracked in the database are toxic; that is, they are able to produce harm to humans. The analysis of the consequences to the public from chemical emissions under normal operations of LANL is provided in chapter 5, sections 5.2.4 and 5.2.6 of the LANL SWEIS. Methodology is provided in section 5.1.4 and 5.1.6 of the LANL SWEIS. Those of risk to the public, should they be accidentally released to the atmosphere, were determined by screening the entire database. Details on the accidental release screening and its results are presented in appendix G, Accident Analysis of the LANL SWEIS.

G.1.2 Supplemental Information on Public Health: U.S., New Mexico, and the Local LANL Community

The information presented below is supplemental to the information presented in chapter 4, section 4.6. It is presented to provide the context of the human health analysis provided in chapter 5, which estimates potential consequence to public health.

The population of Los Alamos County has grown primarily by immigration. The average annual fertility rate has remained at approximately 48/1,000 women across all races (DOC 1990 and Athas and Key 1993), which would produce annual growth of only 2.4 percent if there were no deaths. However, the growth rate has been approximately 25 percent between 1950 and 1960, more than 16 percent between 1960 and 1970 as well as between 1970 and 1980, and approximately 3 percent between 1980 and 1990.

Several studies have been conducted in the community due to concerns expressed within the community concerning the rates of some cancers. While these are summarized in section 4.6 of the SWEIS, additional information is presented here in order to meet the request of many during the scoping meetings for presentation of these results in the SWEIS.

These studies are largely descriptive; that is, they use statistical analyses to identify patterns of disease or death in a community. The thyroid cancer study (Athas 1996) reported below is a mixture of descriptive and analytical approaches (based on case studies and observational analyses). All epidemiological studies are subject to limitations in attempting to determine cause and effect relationships. Some of these limitations are:

- Small population sizes in the community to be studied

- Relatively few total numbers of cases of the specific disease or cancer to be studied
- High mobility in the population to be studied (if a large portion of the community has been in the community for shorter periods of time than that necessary to detect chronic disease, results are inconclusive)
- Disease etiology—one may have received the causative exposure decades before its diagnosis; households in the U.S. move on average every 3 years; in Los Alamos County in 1980, 45 percent of residents had been in the same home for 5 years; earlier census data showed lesser periods of time in the same residence
- Comparability—for instance, the makeup of Los Alamos County is quite dissimilar from its surrounding counties in ethnic distribution and in socioeconomic and occupational conditions
- Natural variability in disease incidence within the human population from any and all sources
- Increased technology efficiency used in disease detection, therefore, causing apparent increases in rates of incidence of the better-detected disease
- More than one causal agent suspected or known to cause the disease being studied, including lifestyle choices such as smoking and dietary patterns
- Disease cause from multiple sources in the same community
- Methodology limitations such as multiple comparison across differing time periods, across studies made for different purposes, consideration of all combinations across the study time frame, etc.

G.1.2.1 *Public Health: United States*

Heart disease remains the leading cause of death in the U.S. (Table G.1.2.1–1). There has been a significant decrease in mortality in the U.S. attributable to heart disease and cerebrovascular disease over the last 20 years. Cancer remains the second leading cause of death.

Table G.1.2.1–2 identifies the lifetime risk of dying from cancer for men and women by cancer type. Over all cancer types, the lifetime risk of dying from cancer is approximately 24 percent for men and 21 percent for women.

Cancer incidence and mortality trends have changed over the last 20 years (Table G.1.2.1–3). Melanoma of the skin, for example, has increased in both incidence and mortality rate, as has brain and other nervous system

TABLE G.1.2.1–1.—*Leading Causes of Death in U.S.: Percent of All Causes of Death (1973 Versus 1993)*

CAUSE OF DEATH	PERCENT OF ALL CAUSES (1973)	PERCENT OF ALL CAUSES (1993)
Heart Disease	38.4	32.8
Cerebrovascular	10.9	6.6
Cancer	17.1	23.4
Pneumonia and Influenza	3.2	3.7
Chronic Lung Disease	1.5	1.2
Accidents	5.9	4.0
All Other Causes	22.5	28.4

Source: Ries et al. 1996

TABLE G.1.2.1–2.—*Lifetime Risk (Expressed as Percent) of Dying from Cancer: SEER[a] Areas (1973 Through 1993), All Races*

TYPE OF CANCER	MEN	WOMEN
All Types	23.77	20.66
Oral and Pharynx	0.45	0.24
Esophagus	0.65	0.23
Stomach	0.81	0.53
Colon and Rectum	2.54	2.54
Liver and Bile Duct	0.52	0.33
Pancreas	1.11	1.21
Larynx	0.25	0.07
Lung and Bronchus	7.11	4.35
Melanomas of Skin	0.31	0.20
Breast	0.03	3.54
Cervix Uteri	—	0.27
Corpus and Uterus	—	0.53
Ovary	—	1.12
Prostate	3.62	—
Testis	0.02	—
Urinary Bladder	0.69	0.34
Kidney and Renal Pelvis	0.49	0.33
Brain and Other Nervous	0.51	0.41
Thyroid	0.04	0.07
Hodgkin's Disease	0.06	0.05
Non-Hodgkin's Lymphoma	0.90	0.85
Multiple Myeloma	0.47	0.43
Leukemias	0.93	0.74

[a] SEER is the NIH/NCI Surveillance, Epidemiology, and End Results Program.
Source: Ries et al. 1996

cancers. Leukemia incidence and mortality rates have decreased.

G.1.2.2 *Comparison of Cancer Mortalities Between the U.S. and New Mexico*

A comparison of cancer mortality rates between the U.S. as a whole and New Mexico is given in Table G.1.2.2–1. These comparisons were made for 1989 through 1993 based on the National Institute of Health/National Cancer Institute (NIH/NCI) Surveillance, Epidemiology, and End Results (SEER) Program (Ries et al. 1996). For most cancers, differences were insignificant.

However, New Mexico had significantly higher mortality from thyroid cancer. (The reader is referred also to Athas 1996 for the local Los Alamos County study of thyroid cancer presented below.) New Mexico deaths due to thyroid cancers ranked 4th among the states. Thyroid cancers are associated with some types of radiological processes and research applications, principally those that could result in emitted radio-iodine. LANL has historically not used more than research amounts of radio-iodine. Radio-iodine emissions from LANL have been measured and have continually been very low (chapter 4, section 4.4 and the tables of emissions estimated for key LANL facilities, in chapter 3, section 3.6 discuss this further).

New Mexico had statistically lower rates of cancer mortalities for several cancers (Table G.1.2.2–1) relevant to the Los Alamos cancer studies, specifically, brain and other nervous system cancers and breast cancer.

G.1.2.3 *Cancer Incidence and Mortality Among Ethnic Groups Relevant to the LANL Area*

While the Native American population within Los Alamos County remains less than 3 percent (DOC 1990), the populations down gradient (with respect to air emissions and water flow) in the adjacent Santa Fe County Area are

TABLE G.1.2.1–3.—*Trends in Cancer Incidence and Mortality for Selected Cancers (1973 Through 1993), All Races, Both Sexes*

DECREASING INCIDENCE; DECREASING MORTALITY	INCREASING INCIDENCE; DECREASING MORTALITY	INCREASING INCIDENCE; INCREASING MORTALITY
Oral Cavity and Pharynx	Ovary	Total Cancers
Stomach	Testis	Esophagus
Colon and Rectum	Urinary Bladder	Liver and Bile Duct
Pancreas	Thyroid	Lung and Bronchus
Larynx		Melanoma of Skin
Cervix Uteri		Breast
Corpus and Uterus		Prostate
Hodgkin's Disease		Kidney and Renal Pelvis
Leukemia		Brain and Other Nervous
		Non-Hodgkin's Lymphoma
		Multiple Myeloma

Source: Ries et al. 1996

TABLE G.1.2.2–1.—*Comparison of Cancer Mortality Rates for the United States and New Mexico (1989 Through 1993), All Races, Both Sexes (Rate per 100,000 Population, Age Adjusted to 1970 U.S. Standard Population)*

TYPE OF CANCER	U.S. RATE	NEW MEXICO RATE	RANKING (AMONG STATES)	COMPARISON U.S. VS. NEW MEXICO
Breast	26.8	23.4	49th	NM < U.S.
Colon and Rectum	18.4	14.2	50th	NM < U.S.
Esophagus	3.5	2.4	49th	NM < U.S.
Hodgkin's Disease	0.6	0.6	25th	NSD
Larynx	1.4	1.2	34th	NSD
Leukemia	6.4	6.1	40th	NSD
Liver and Bile Duct	3.0	3.2	15th	NSD
Lung and Bronchus	49.9	35.0	49th	NM < U.S.
Melanomas of Skin	2.2	2.1	49th	NSD
Non-Hodgkin's Lymphoma	6.4	5.6	46th	NSD
Brain and Nervous	4.2	3.5	48th	NM < U.S.
Stomach	4.6	5.0	12th	NSD
Testis	0.3	0.2	43rd	NM < U.S.
Urinary Bladder	3.3	2.7	47th	NM < U.S.
Oral/Pharynx	2.9	2.6	32nd	NSD
Pancreas	8.4	8.1	40th	NSD
Thyroid	0.3	0.4	4th	NM > U.S.
Prostate	26.4	23.2	49th	NM < U.S.
Ovary	7.8	6.7	47th	NSD
Kidney and Renal Pelvis	3.5	3.4	36th	NSD
Multiple Myeloma	3.0	3.0	30th	NSD
Corpus and Uterus	3.4	3.0	43rd	NSD
Cervix Uteri	2.9	2.7	33rd	NSD

Sources: SEER Database and Ries et al. 1996
NSD = No significant difference

dominantly Native American (San Ildefonso Pueblo).

Table G.1.2.3–1 summarizes the findings regarding the top five cancers (both incidence and mortality) among nonhispanic whites (U.S.), Hispanic whites (U.S.), and Native Americans (New Mexico). The Native American cancer incidence and cancer mortality rates are lower than either of the other examined populations for both men and women. This is the case for all cancer types, not just the top five cancers with respect to incidence and mortality rate.

Among men, lung and prostate cancer dominate incidence and mortality. Among women, breast and lung cancer dominate cancer incidence and mortality. A fairly rare cancer, gall bladder, is the leading cause of cancer mortality among New Mexican Native American women. However, because there were so few cases, and the uncertainty level thus associated with the observation is so high, it is inappropriate to draw conclusions even regarding gall bladder cancer incidence in this population of women.

G.1.2.4 *Supplemental Information on Recent Studies of Los Alamos County Cancer*

Objectives

The primary objective of the study was to review Los Alamos County incidence rates for brain and nervous system cancer and other major cancers during the 21-year time period 1970 to 1990 (Athas and Key 1993). Secondary objectives were to review mortality rate data for select cancers of concern and to review Los Alamos County mortality data relating to benign brain and nervous system tumors.

Specific aims developed for incidence study were as follows:

- To calculate age-adjusted cancer incidence rates for Los Alamos County and a New Mexico state reference population using data of the New Mexico Tumor Registry (NMTR)
- To compare Los Alamos County cancer incidence rates to (1) incidence rates calculated for a New Mexico state reference population, and (2) national rates obtained from the SEER Program of the National Cancer Institute
- To determine if any of the Los Alamos County cancer incidence rates were elevated in comparison to rates observed in the reference population

The study protocol specified that statistical tests would be used to determine whether any of the Los Alamos County rates were elevated in comparison to the reference populations. Early in the course of the study, however, it became apparent that the small number of cases for virtually all of the Los Alamos County cancers reviewed would make the finding of statistical significance unlikely for small to modest elevations in a rate. Consequently, the analysis of the Los Alamos County incidence data was expanded to include not only statistical considerations but other types of information such as temporal patterns of cancer occurrence, prevalence of established risk factors, case characteristics, and tumor cell types. Cancers of concern were: oral cavity and pharynx, digestive system, respiratory system, melanoma of the skin, female breast, female genital system, urinary system, male genital system, lymphoreticular system, childhood cancers (ages 0 to 19 years) thyroid, and brain and nervous system cancers.

Following a review of tabulated incidence rate data for 23 major cancers, nine were selected for additional review and evaluation: liver and intrahepatic bile duct cancer, non-Hodgkin's lymphoma, leukemia, melanoma of skin, ovarian cancer, breast cancer, childhood cancers, thyroid cancer, and brain and nervous

TABLE G.1.2.3–1.—*The Five Most Frequently Diagnosed Cancer and the Five Most Common Types of Cancer Death (1988 Through 1992) Among White Non-Hispanics (all U.S.), White Hispanics (all U.S.), Native Americans (New Mexico)*

POPULATION GROUP	CANCER INCIDENCE[a]		CANCER MORTALITY[a]	
	CANCER TYPE (RATES/100,000 POPULATION, AGE ADJUSTED TO 1970 U.S. STANDARD)			
	MEN	WOMEN	MEN	WOMEN
White, Non-Hispanic	Prostate (137.9)	Breast (115.7)	Lung (74.2)	Lung (32.9)
	Lung (79.0)	Lung (43.7)	Prostate (24.4)	Breast (27.7)
	Colon/Rectum (57.6)	Colon/Rectum (39.2)	Colon/Rectum (23.4)	Colon/Rectum (15.6)
	Bladder (33.1)	Corpus Uteri (23.0)	Pancreas (9.8)	Ovary (8.2)
	Non-Hodgkin's Lymphoma (19.1)	Ovary (16.2)	Leukemia (8.6)	Pancreas (7.0)
White, Hispanic	Prostate (92.8)	Breast (73.5)	Lung (33.6)	Breast (15.7)
	Lung (44.0)	Colon/Rectum (25.9)	Prostate (15.9)	Lung (11.2)
	Colon/Rectum (40.2)	Lung (20.4)	Colon/Rectum (13.4)	Colon/Rectum (8.6)
	Bladder (16.7)	Cervix (17.1)	Stomach (8.8)	Pancreas (5.4)
	Stomach (16.2)	Corpus Uteri (14.5)	Pancreas (7.4)	Ovary (5.1)
Native American, NM	Prostate (52.5)	Breast (31.6)	Prostate (16.2)	Gallbladder (8.9)[b]
	Colon/Rectum (18.6)	Ovary (17.5)	Stomach (11.2)[b]	Breast (8.7)[b]
	Kidney (15.6)	Colon/Rectum (15.3)	Liver (11.2)[b]	Cervix (8.0)[b]
	Lung (14.4)	Gallbladder (13.2)	Lung (10.4)[b]	Pancreas (7.4)[b]
	Liver (13.1)[b]	Corpus Uteri (10.7)	Colon/rectum (8.5)[b]	Ovary (7.3)[b]

[a] NIH/NCI SEER Program statistics from several regions around the U.S.
[b] Statistics calculated with extremely high uncertainty because they are based on fewer than 25 cases. Other rates (not footnoted) were calculated from larger total numbers of cases and, therefore, have less uncertainty associated with them.
Source: Miller et al. 1996

system cancer. The majority of these cancers were chosen on the basis of incidence rates, which were higher in Los Alamos County in comparison to the reference populations. Childhood cancer was chose for further review based on mortality rate data showing an apparent excess of childhood cancer deaths in Los Alamos County. Leukemia and liver cancer where chosen as cancers of concern specifically to examine tumor cell types. Cancers not chosen for further review included major sites in the respiratory, digestive, and urinary systems.

Incidence Data: Data Sources

Information regarding newly diagnosed cancers among Los Alamos County residents and New Mexico non-Hispanic Whites was compiled from records collected since 1969 by the NMTR at the University of New Mexico Cancer Center. Cancer is a reportable disease in New Mexico by regulation of the New Mexico Department of Health (NMDOH). Since the late 1960's, NMTR has been the repository of the confidential medical record abstracts and computerized masterfile for cancer in New Mexico. NMTR has been a part of the SEER Program since that program began in 1973.

Cancer Incidence Findings (1970 to 1990)

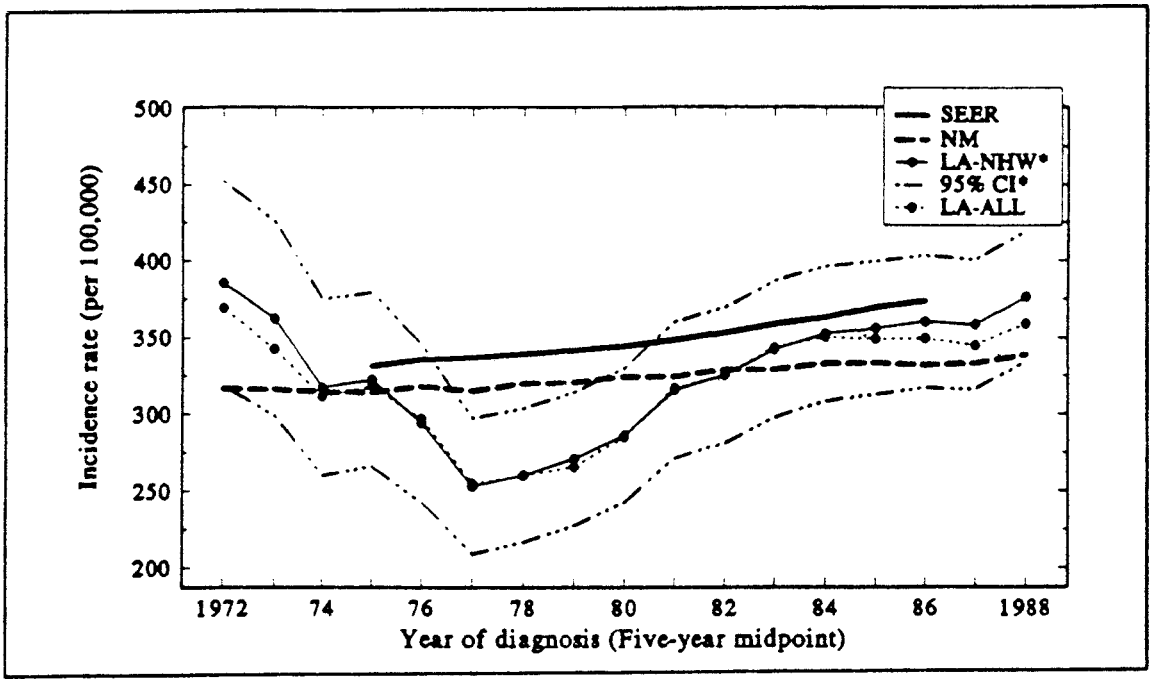
All Cancers. Figure G.1.2.4–1 shows that the Los Alamos County incidence rates for "all cancers" fluctuated considerably; but the rates generally were comparable to or lower than rates observed in the state and national reference populations.

Liver and Intra-Hepatic Duct Cancer. Seven cases of primary liver and intra-hepatic bile duct cancer occurred in Los Alamos County. Four of the seven cases (57 percent) were diagnosed between 1981 and 1982. Los Alamos County incidence rates were highly variable as a result of the small number of cases and the clustered temporal distribution of cases. No cases were reported up until the early 1980's, at which time the four cases diagnosed in 1981 to 1982 caused a marked elevation in the Los Alamos County rates in comparison to the state and national reference rates (Figure G.1.2.4–2). Los Alamos County rates subsequently diminished to a level consistent with the reference rates.

Non-Hodgkin's Lymphoma. Los Alamos County consistently experienced a small to modest elevation in incidence compared to the reference populations (Figure G.1.2.4–3). The magnitude of the elevated Los Alamos County incidence varied widely up to a two-fold higher than expected level. None of the Los Alamos County lower confidence limits excluded the reference rates. Incidence in the Los Alamos County non-Hispanic White population was consistently higher than that observed in the total county population. All Los Alamos County rates were based on 14 or fewer cases. For the most recent five-year time period (1986 to 1990), the rate for non-Hispanic Whites in Los Alamos County was 57 percent greater than the state reference rate.

Leukemia. The incidence of leukemia in Los Alamos County generally was the same or lower than that observed in the reference populations (Figure G.1.2.4.–4). Wide fluctuations in the Los Alamos County rates occurred as a result of low case numbers. All Los Alamos County rates were based on nine or fewer cases. For the most recent 5-year time period (1986 to 1990), the Los Alamos County rate equalled the state reference rate.

Melanoma. The incidence of melanoma consistently was around 50 percent higher in New Mexico non-Hispanic Whites compared with SEER Whites. Melanoma incidence steadily increased in both reference populations. Incidence rates in Los Alamos County were higher than the state reference rates over most of the 21-year study time period (Figure G.1.2.4–5). Early time periods were characterized by a small elevation in the Los Alamos County incidence; whereas, a more pronounced excess of melanoma in Los Alamos County began to appear in the mid 1980's.



SOURCE: Athas and Key 1993

FIGURE G.1.2.4–1.—*5-Year Average Annual Incidence of All Cancer Sites, Los Alamos County, New Mexico NHW, SEER Whites, 1970 to 1990.*

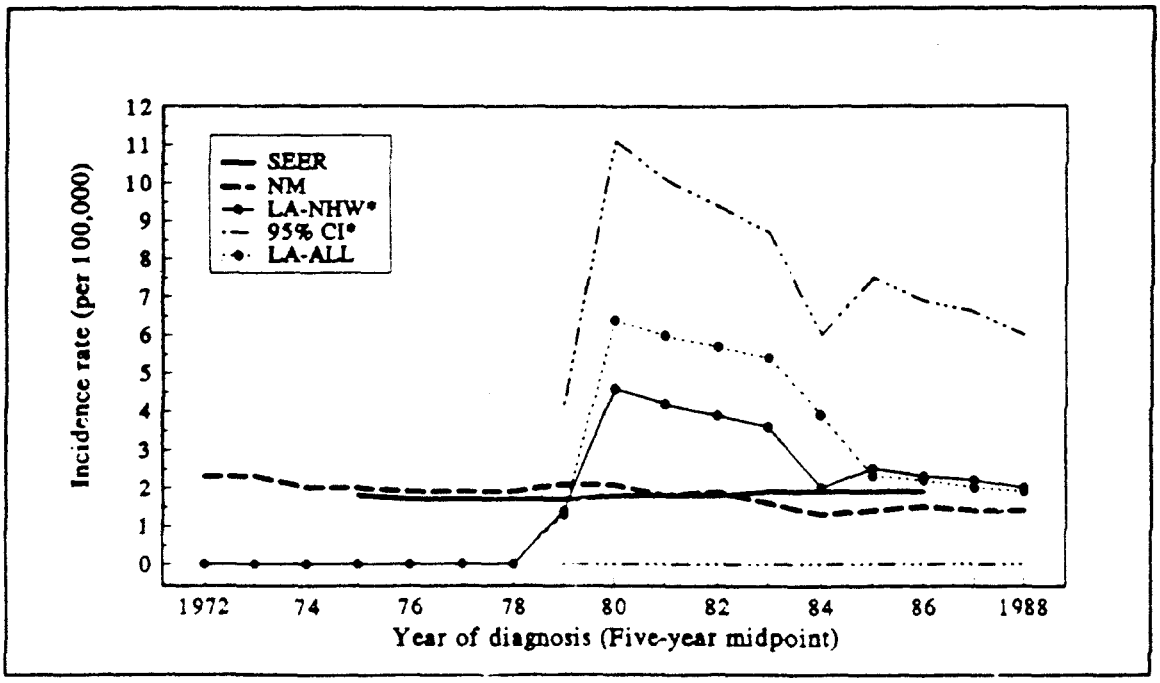


FIGURE G.1.2.4–2.—*5-Year Average Annual Incidence of Liver and Intra-Hepatic Bile Duct Cancer, Los Alamos County, New Mexico NHW, SEER Whites, 1970 to 1990.*

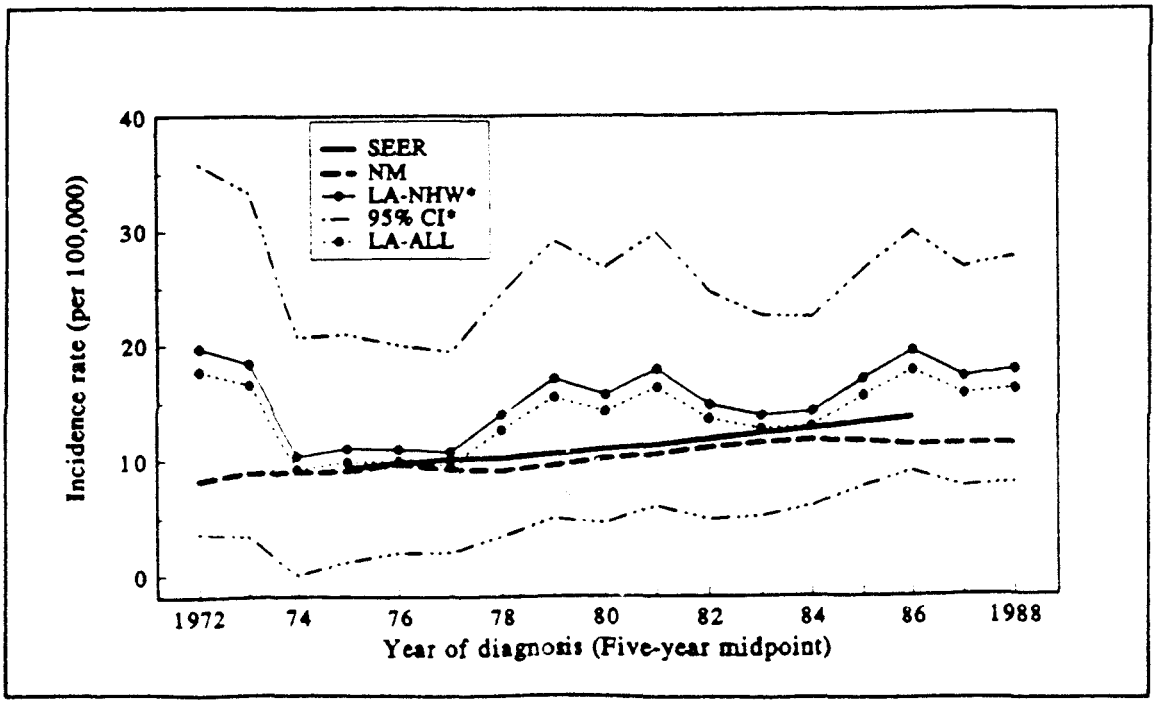


FIGURE G.1.2.4–3.—*5-Year Average Annual Incidence of Non-Hodgkin's Lymphoma, Los Alamos County, New Mexico NHW, SEER Whites, 1970 to 1990.*

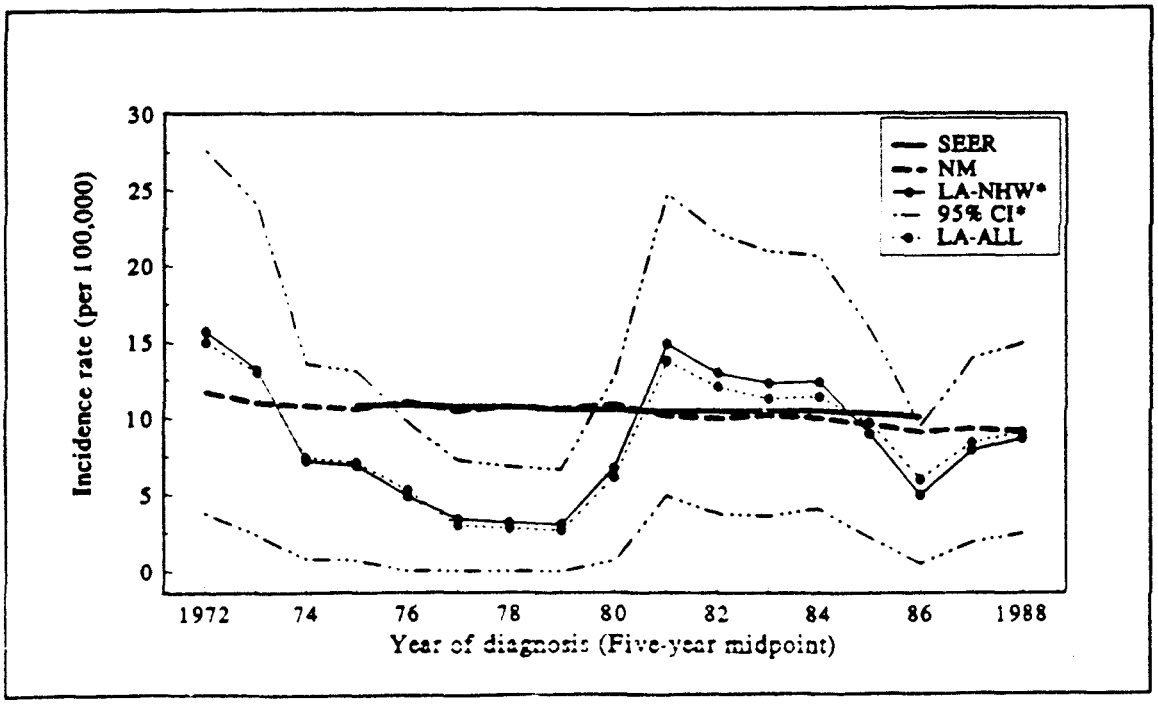


FIGURE G.1.2.4–4.—*5-Year Average Annual Incidence of Leukemia, Los Alamos County, New Mexico NHW, SEER Whites, 1970 to 1990.*

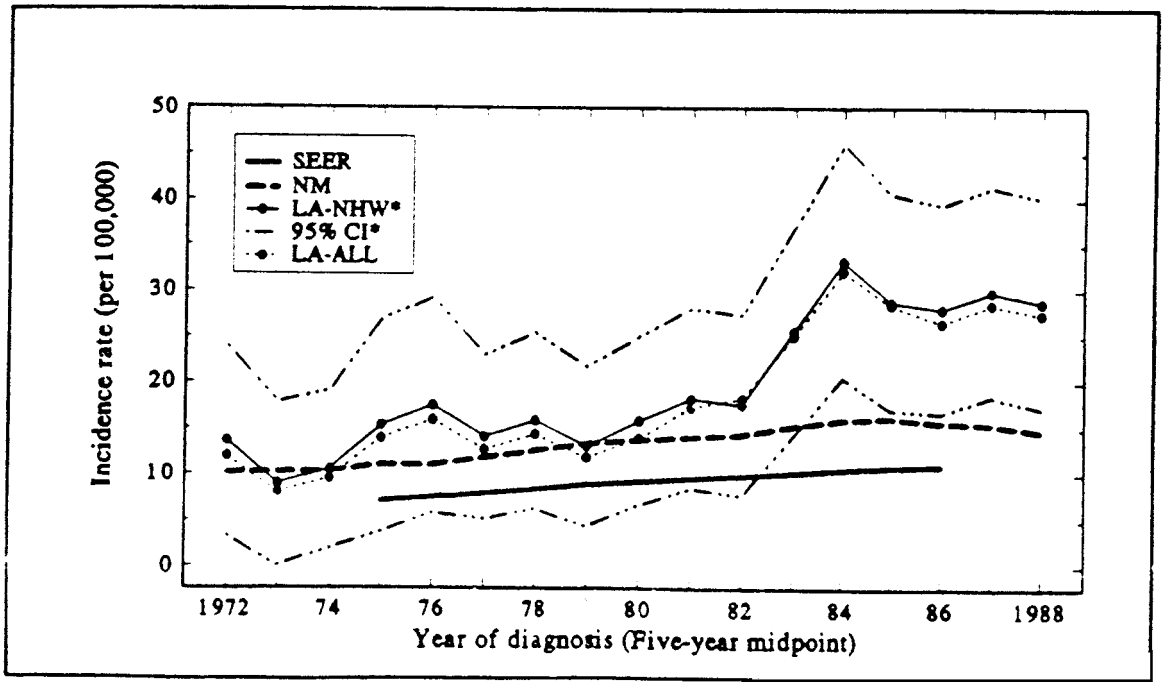


FIGURE G.1.2.4–5.—*5-Year Average Annual Incidence of Melanoma of Skin, Los Alamos County, New Mexico NHW, SEER Whites, 1970 to 1990.*

Beginning with the 1982 to 1986 period, and for all subsequent periods, the lower confidence limit of the Los Alamos County rate excluded the state reference rates. During these later periods, the incidence of melanoma in Los Alamos County increased roughly two-fold over that observed statewide.

Ovarian. Los Alamos County rates steadily rose by three-fold during 1970 to 1990, while both the sate and national reference rates remained essentially constant (Figure G.1.2.4–6). Initially lower than the reference rates, Los Alamos County incidence climbed to a statistically significant three-fold excess level during the 1982 to 1986 period. Half of all the Los Alamos County cases (15 out of 30) were diagnosed during these 5 years. Los Alamos County ovarian cancer incidence was two-fold higher than that observed in the state during the most recent 5-year period (1986 to 1990).

Breast. Breast cancer incidence in Los Alamos County women varied little over time; whereas, both reference populations displayed increasing incidence over time (Figure G.1.2.4–7). Los Alamos County incidence rates were 10 percent to 50 percent higher than the state and national reference rates over the entire study period. The lower confidence limits for the Los Alamos County rates consistently were near the reference rates, but excluded the reference rates in only several instances.

Childhood Cancers. Los Alamos County childhood cancer rates fluctuated around the more stable state and national reference population rates (Figure G.1.2.4–8). Following an initial two-fold elevation during the earliest period (1970 to 1972), subsequent periods were characterized by incidence rates that were slightly higher than or lower than the reference incidence rates. Two childhood brain cancer cases not in the original childhood cancer data set were discovered through a supplemental review of childhood cancer mortality statistics. The two additional cases, diagnosed in 1978 and 1980, would raise the original 1978 to 1982 Los Alamos County rate (13.7 per 100,000) by about

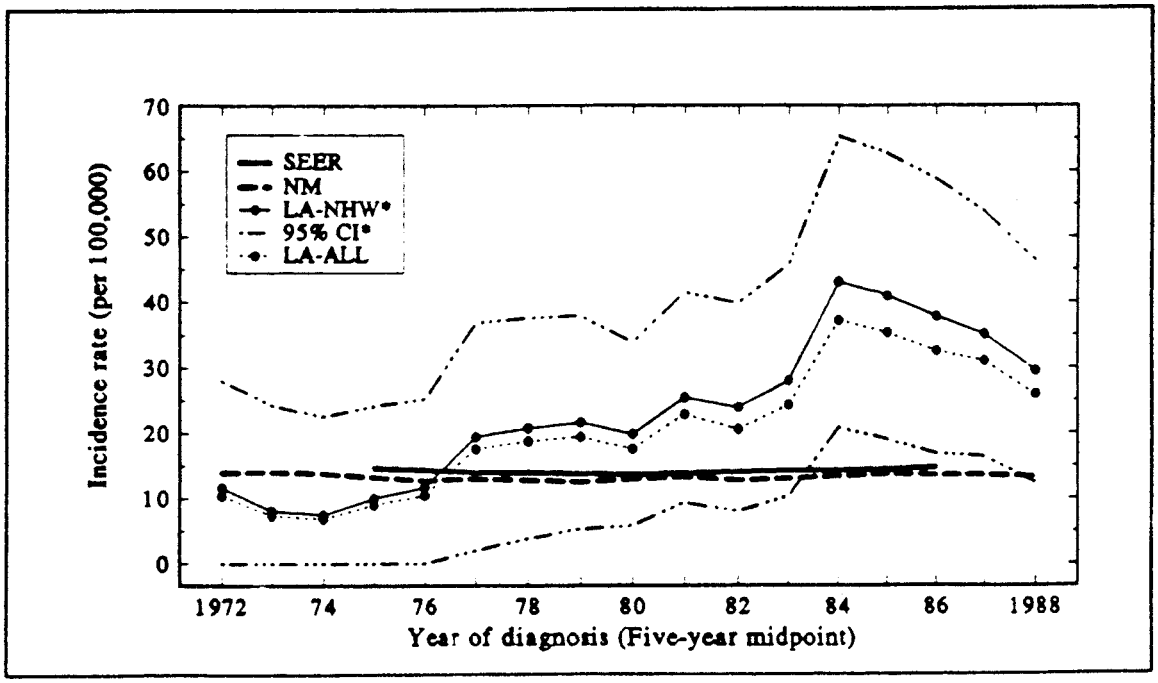


FIGURE G.1.2.4–6.—*5-Year Average Annual Incidence of Ovarian Cancer, Los Alamos County, New Mexico NHW, SEER Whites, 1970 to 1990.*

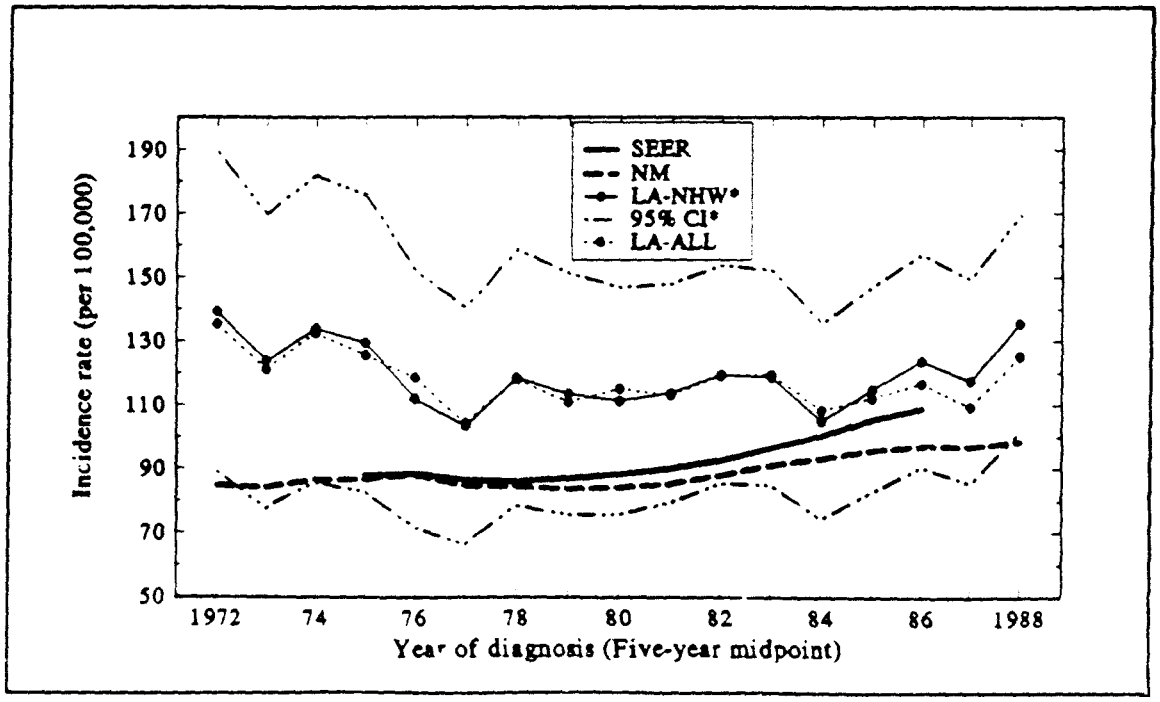


FIGURE G.1.2.4–7.—*5-Year Average Annual Incidence of Female Breast Cancer, Los Alamos County, New Mexico NHW, SEER Whites, 1970 to 1990.*

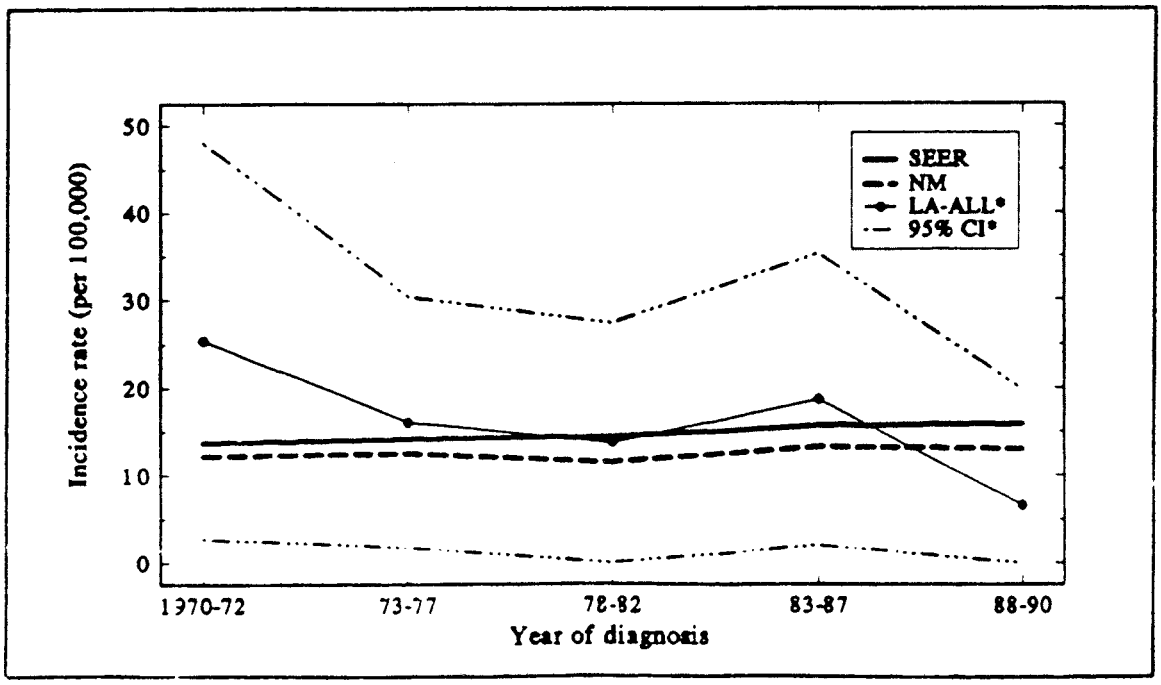


FIGURE G.1.2.4–8.—*Average Annual Incidence of Childhood Cancer (0 to 19 Years), Los Alamos County, New Mexico NHW, SEER Whites, 1970 to 1990.*[a]

[a] Incidence rate data based on independent time periods and not 5-year moving averages.

50 percent to 20.3 cases per 100,000. For the latest period (1988 to 1990), the incidence of childhood cancers in Los Alamos County was roughly 50 percent lower than that seen in the state reference population; however, the Los Alamos County rate was based on only one case.

Thyroid. The incidence of thyroid cancer in Los Alamos County prior to the mid 1980's was roughly stationary and less than two-fold higher than that seen in the reference populations (Figure G.1.2.4–9). Los Alamos County incidence rates began to rise during the mid 1980's and continued to climb up until the latest time interval (1986 to 1990). The incidence of thyroid cancer in Los Alamos County during 1986 to 1990 was nearly four- fold higher than that observed in the state reference population. The near four-fold elevation for Los Alamos County was statically significant. Roughly half (17 out of 37) of all thyroid cancer cases that occurred in Los Alamos County between 1970 and 1990 were diagnosed during the 1986 to 1990 interval.

Brain and Nervous System. The incidence of brain cancer in Los Alamos County increased over time (Figure G.1.2.4–10). Los Alamos County incidence rates were lower than or comparable to the reference rates up until the mid 1980's. Increases in Los Alamos County brain cancer incidence became apparent during the mid to late 1980's. Los Alamos County incidence rates (all races) during this period were 60 to 80 percent higher than rates for the state and national reference populations. Diagnosed in 1978 and 1980, two additional cases raised the central portion of the incidence rate curve to a range more comparable with the reference rates, but had no effect on the rates observed during the period of elevated incidence.

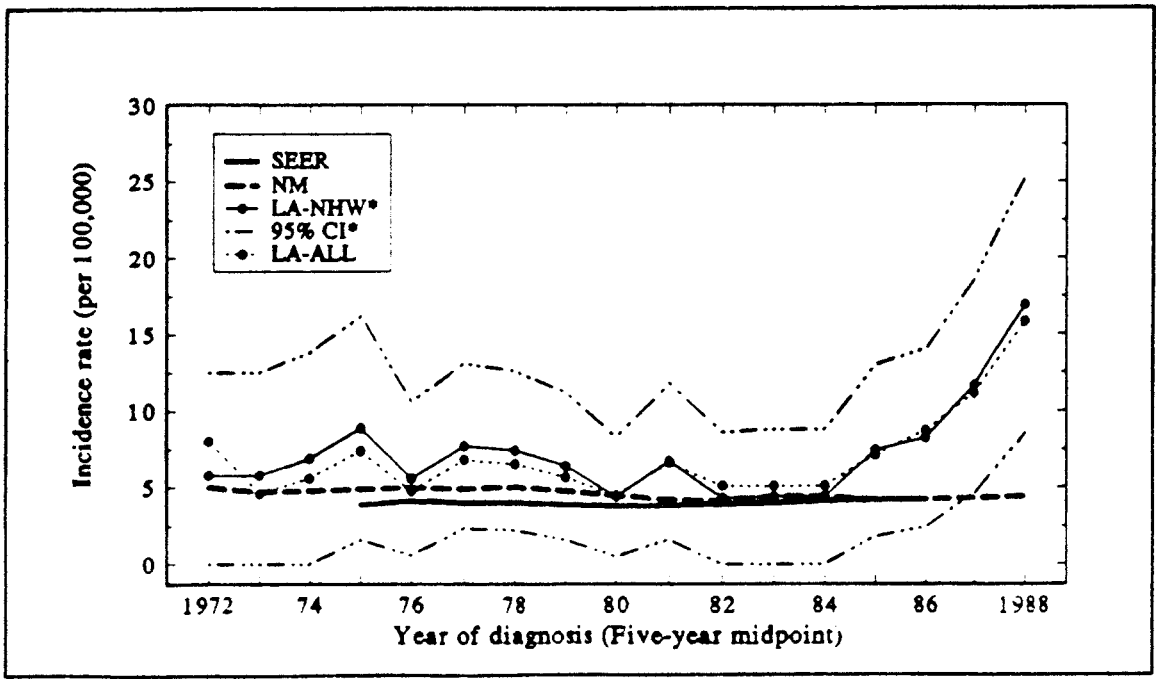


FIGURE G.1.2.4–9.—*5-Year Average Annual Incidence of Thyroid Cancer, Los Alamos County, New Mexico NHW, SEER Whites, 1970 to 1990.*

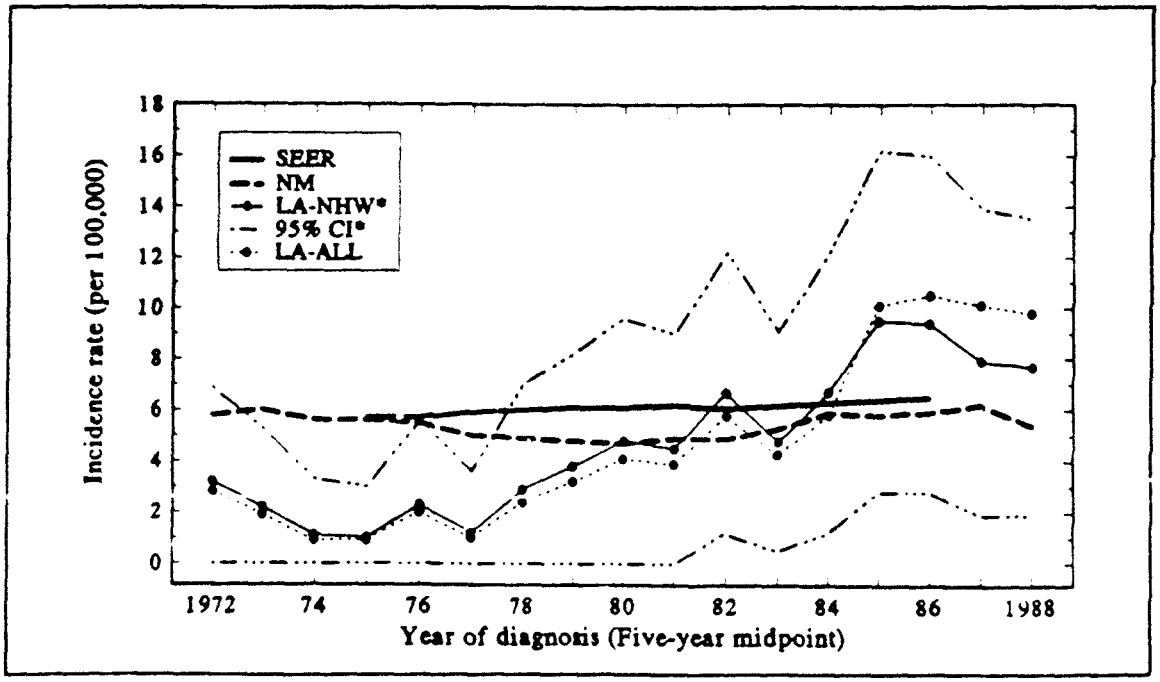


FIGURE G.1.2.4–10.—*5-Year Average Annual Incidence of Brain and Nervous System Cancer, Los Alamos County, New Mexico NHW, SEER Whites, 1970 to 1990.*

Mortality

Mortality rates for Los Alamos County and the U.S. were obtained as age-adjusted average annual mortality rates from the National Center for Health Statistics (NCHS) and the National Cancer Institute. All rates were standardized to the 1970 U.S. standard population and were race-specific for Whites. Site-specific Los Alamos County mortality rates were available for the periods 1969 to 1972, 1973 to 1977, 1978 to 1982, and 1983 to 1987. U.S. rates were available for the time period 1968 to 1972. For some cancers, both Los Alamos County and U.S. rates were available for the period 1968 to 1972. The confidence intervals that accompany the mortality rates were calculated as described for the incidence rates. Table G.1.2.4–1 summarizes the mortality rates by cancer type for Los Alamos County. Nationwide rates are also reported for comparison.

Subcounty Cancer Incidence

Table G.1.2.4–2 describes the cancer incidence for the five census tracts within Los Alamos County for all races, 1980 to 1990. The New Mexico non-Hispanic White population rates are provided also.

TABLE G.1.2.4–1.—*Average Annual Age-Adjusted Mortality Rates by Cancer Type for Los Alamos County and U.S. Whites (1969 to 1987)*

CANCER TYPE	LOCATION	MORTALITY RATE[a]			
		1969 TO 1972	1973 TO 1977	1978 TO 1982	1983 TO1987
Liver and Bile	Los Alamos	14.6 (2)[b]	0 (0)	5.4 (3)	7.1 (4)
	U.S.	—	2.1	2.1	2.3
Non-Hodgkin's Lymphoma	Los Alamos	13.5 (2)	5.8 (2)	12.0 (6)	2.3 (2)
	U.S.	NA[c]	4.9	5.2	5.9
Leukemia	Los Alamos	1.2 (1)	11.2 (6)	1.3 (1)	4.5 (4)
	U.S.	NA	6.8	6.7	6.5
Melanoma	Los Alamos	0 (0)	6.5 (3)	2.9 (2)	1.0 (1)
	U.S.	1.7	1.9	2.2	2.3
Ovarian	Los Alamos	19.7 (3)	5.7 (1)	8.9 (3)	3.8 (2)
	U.S.	NA	8.6	8.1	7.9
Breast	Los Alamos	39.6 (8)	17.4 (7)	60.7 (20)	29.7 (12)
	U.S.	26.9	26.9	26.6	27.2
Childhood Cancer	Los Alamos	3.6 (1)	12.3 (4)	16.1 (5)	10.6 (3)
	U.S.	6.6	5.4	4.6	4.0
Brain and Nervous System	Los Alamos	0 (0)	6.3 (4)	5.8 (5)	5.8 (5)
	U.S.	NA	4.0	4.1	4.3
Thyroid	Los Alamos	0 (0)	0 (0)	0 (0)	0 (0)
	U.S.	NR[d]	NR	NR	NR

[a] Rates per 100,000 and are age-adjusted to the 1970 U.S. standard population.
[b] Number of deaths given in parentheses.
[c] NA = Not available
[d] NR = Not reported

TABLE G.1.2.4–2.—*Average Annual Age-Adjusted Cancer Incidence Rates for Sub-County Regions of Los Alamos County, All Races (1980 to 1990)*[a]

SITE	CENSUS TRACT[b]					CDP[c]		LOS ALAMOS COUNTY	NEW MEXICO NHW[d]
	1	2	3	4	5	LOS ALAMOS	WHITE ROCK		
Non-Hodgkin's Lymphoma	18.9 (2)	4.5 (2)	20.4 (5)	11.1 (5)	16.7 (10)	12.6 (14)	16.7 (10)	14.3 (24)	11.0
	{0.0 to 45.6}	{0.0 to 11.0}	{2.2 to 38.7}	{1.2 to 21.0}	{6.1 to 27.2}	{5.8 to 19.3}	{6.1 to 27.2}	{8.5 to 20.1}	
Leukemia	1.9 (1)	10.3 (4)	17.5 (2)	5.5 (3)	11.8 (7)	7.1 (10)	11.8 (7)	8.5 (17)	9.5
	{0.0 to 5.7}	{0.0 to 20.6}	{0.0 to 42.2}	{0.0 to 11.8}	{2.9 to 20.7}	{2.6 to 11.6}	{2.9 to 20.7}	{4.4 to 12.6}	
Melanoma[e]	33.8 (10)	22.0 (10)	35.8 (7)	13.5 (6)	21.7 (11)	23.2 (32)	21.7 (11)	22.0 (43)	14.5
	{12.4 to 55.2}	{8.1 to 35.9}	{8.7 to 62.9}	{1.5 to 24.5}	{8.6 to 34.8}	{15.0 to 31.4}	{8.6 to 34.8}	{15.3 to 28.7}	
Ovary (Female)	76.7 (9)	19.4 (4)	19.5 (2)	14.0 (3)	12.7 (4)	27.4 (18)	12.7 (4)	23.0 (22)	12.8
	{25.6 to 127.8}	{0.0 to 38.8}	{0.0 to 47.0}	{0.0 to 30.2}	{0.0 to 25.4}	{14.5 to 40.3}	{0.0 to 25.4}	{13.2 to 32.8}	
Breast (Female)	145.3 (28)	120.5 (21)	159.2 (16)	85.3 (21)	116.0 (41)	119.8 (86)	116.0 (41)	119.0 (127)	92.2
	{90.4 to 200.2}	{67.9 to 173.1}	{79.6 to 238.9}	{48.1 to 122.5}	{79.8 to 152.3}	{93.9 to 145.6}	{79.8 to 152.3}	{97.9 to 140.1}	
Childhood (< 20 years)	21.9 (2)	6.7 (1)	0.0 (0)	24.5 (2)	16.9 (4)	14.2 (5)	16.9 (4)	15.2 (9)	14.8
	{0.0 to 52.8}	{0.0 to 20.2}	{ - }	{0.0 to 59.2}	{0.0 to 33.9}	{1.5 to 26.9}	{0.0 to 33.9}	{5.1 to 25.3}	
Thyroid	16.0 (6)	3.8 (2)	5.8 (1)	8.7 (4)	9.3 (9)	9.0 (13)	9.3 (9)	9.8 (22)	4.3
	{2.9 to 29.1}	{0.0 to 9.1}	{0.0 to 17.5}	{0.0 to 17.4}	{3.1 to 15.4}	{4.0 to 14.0}	{3.1 to 15.4}	{5.6 to 14.0}	
Brain	7.3 (2)	5.7 (3)	14.2 (3)	7.4 (2)	8.2 (7)	7.4 (10)	8.2 (7)	7.9 (17)	5.1
	{0.0 to 17.5}	{0.0 to 12.4}	{0.0 to 30.6}	{0.0 to 18.0}	{2.0 to 14.3}	{2.7 to 12.1}	{2.0 to 14.3}	{4.1 to 11.7}	

[a] Rates are for residence at diagnosis for all races per 100,000, age-adjusted to U.S. 1970 standard population; number of cases in parentheses ( ); 95% confidence limits in brackets { }, truncated at zero.
[b] Census Tract Designations: (1) North/Barranca Mesa; (2) North Community; (3) Western Area; (4) Eastern Area; (5) White Rock.
[c] Los Alamos Census Designated Place (CDP) comprises census tracts 1 through 4, White Rock CDP comprises census tract 5.
[d] Non-Hispanic Whites
[e] Excludes two cases with unknown residence at diagnosis.
Source: New Mexico Tumor Registry