APPENDIX A: Use and Toxicology of Chemicals at Rocket-Engine Test Stands

The principal chemicals used at the rocket test stands were hydrazines (i.e., hydrazine, 1- methylhydrazine [MMH], 1,1-dimethylhydrazine [UDMH]), kerosene fuels (RP-1), asbestos, beryllium, chlorine, fluorine, hydrogen peroxide, isopropyl alcohol, nitric acid, nitrogen tetroxide (NTO), trichloroethylene (TCE), and 1,1,1-trichloroethane (TCA). There were other chemicals used at the test stands in considerably smaller quantities, and they are discussed in the UCLA industrial hygiene survey. Information on chemical use derives from industrial hygiene surveys conducted by UCLA students in July and August 1994, materials supplied by Boeing, and information collected from other sites where rocket engines were tested.

The purpose of the UCLA industrial hygiene survey was to identify current and past operations and processes conducted at the site. The secondary goal was to identify chemical usage at the test stands that may have constituted a hazard to employees. The industrial hygiene survey is available to interested persons.

More recently, in 1998, Boeing/Rocketdyne officials provided investigators additional information on chemical use patterns at the sites. They have also given investigators quantitative data on chemical use, although there is no quantitative data on personal exposure to hazardous chemicals.

This discussion will focus on those chemicals with evidence for chemical carcinogenicity. We do not have quantitative exposure data on exposure to these agents, but given the epidemiologic results it is important to review those chemicals which may have been present even if there is not direct evidence of exposure. The chemicals of principal concern are hydrazine, asbestos, beryllium and TCE. UDMH may contain impurities of dimethylnitrosamine (NDMA) and oxidation of UDMH and MMH may also result in production of NDMA and monomethylnitrosamine (NMA), both of which are considered chemical carcinogens.

Hydrazines

There is evidence that three hydrazines were used at the test stands: hydrazine, MMH, and UDMH. Records obtained by UCLA indicate that hydrazine was used from 1955 through 1977, UDMH from 1955 to 1970, and MMH from 1955 to the present. These represent primary use periods, and, while there may be exceptions, the data are likely to be reasonably accurate. Quantitative estimates of pressurants and propellants use is contained within a log supplied by Boeing. Rocketdyne/Boeing provided UCLA with some incident reports; there were reports of accidental releases of hydrazines, but there were limited estimates of worker exposure.

Hydrazine

Hydrazine is classified in Group 2B, a possible human carcinogen, based on

inadequate evidence in humans and sufficient evidence in animals by the International Agency for Research on Cancer (IARC, 1974). The U.S. EPA classifies hydrazine as a probable human carcinogen (Group B2), and the National Toxicology Program (NTP) Annual Report on Carcinogenesis considers hydrazine a substance that may reasonably be anticipated to be a carcinogen (U.S. EPA, 1984a; U.S. DHHS, 1994). The World Health Organization has concluded: "On the basis of the evidence of carcinogenicity in animals and positive results in short-term tests, it would be prudent to consider hydrazine to be a possible human carcinogen" (WHO, 1987a; 1987b).

Hydrazine induces unscheduled DNA synthesis in human cells in vitro, sister chromatid exchange, DNA strand breaks in rat hepatocytes, somatic mutations in Drosophila and chromosomal aberrations and mutations in plants. It was mutagenic to yeast and bacteria. Hydrazine was positive in five strains of bacteria in the Ames bioassay (IARC, 1974; WHO, 1987a; 1987b). Thus, there is a mechanistic basis and biological plausibility to assume hydrazine is carcinogenic to humans. Overall, this conclusion is reasonable for all the hydrazines described here. Information on the human carcinogenicity of hydrazine is inadequate according to IARC and U.S. EPA. This conclusion is based on a lack of human investigations rather than negative evidence.

There is extensive literature on the carcinogenicity of hydrazine in animals. Animal studies have demonstrated cancer of the liver, lung, and mammary tumors in mice. The routes of administration have included gavage, drinking water, inhalation, and intraperitoneal injection. Inhalation studies in mice, rats, hamsters, and dogs were conducted. Significantly increased incidences of tumors were reported for mice (lung), rats (nasal cavity), and hamsters (nasal cavity). The study duration was considered to be inadequate for dogs. Leukemias were also identified in mice exposed to hydrazine via intraperitoneal administration. Hydrazine exposure in drinking water also produced hepatocellular carcinomas in hamsters (IARC, 1974; WHO, 1987a; 1987b).

Monomethylhydrazine (MMH)

MMH is classified by the U.S. EPA (1993) as a probable carcinogen (Group B2) on the basis of animal studies. There is no evidence to date of carcinogenicity in humans. MMH is positive in the Ames assay for mutagenicity (Toth, 1972).

In a study conducted by Toth (1972), the administration of 0.01% methylhydrazine in the drinking water of 6-week-old randomly bred Swiss mice enhanced the development of lung tumors by shortening their latency period. In the MMH-treated mice, 12 females developed 17 lung tumors, all of which were identified as adenomas, with an incidence of 24%. The lung tumors had an average latency period of 51 weeks. In the male group, 11 mice developed 12 lung tumors, all of which were classified as adenomas, with an incidence of 22%. The average latency period for the tumors was also 51 weeks.

In 1973, Toth and Shimizu observed that the administration of 0.01% methylhydrazine in the drinking water to 6-week-old randomly bred Syrian Golden

hamsters, for the remainder of their lifetime, gave rise to malignant histiocytomas of the liver and tumors of the cecum. While no malignant histiocytomas developed in the controls, 32% (16/49) of the females and 54% (27/50) of the males were observed to have developed such lesions. In females, the incidence of tumors of the cecum was 18% (9/49) and 14% (7/50) in males, compared with 1% (1/99 F; 1/97 M) in the controls (Toth & Shimizu, 1973).

UDMH

UDMH is classified in Group 2B--possibly carcinogenic to humans--by IARC (1974), based on there being sufficient evidence for carcinogenicity in experimental animals. UDMH is classified as a substance that may reasonably be anticipated to be carcinogenic by the NTP (U.S. DHHS, 1994). The U.S. EPA (1984b) has classified UDMH as a probable carcinogen (group B2). Information on the carcinogenic effects of UDMH on humans is not available.

Gavage

Roe et al. (1967) report an increased incidence of pulmonary tumors as compared with the control group in 25 virgin Swiss mice given UDMH by gavage at 0.5 mg a day, five days each week for 40 weeks (test for difference: $0.5 > P > 0.2$).

Drinking Water

Administration of 0.01% UDMH in the drinking water of 6-week-old Syrian golden hamsters for the remainder of their lifetime gave rise to tumors of the cecum and blood vessels. While no vascular tumors developed in the controls, 4% (2/50) of the females and 28% (14/50) of the males were observed to have developed such lesions. In females, the incidence of tumors of the cecum was 20% (10/49) and 30% (15/50) in males, compared with 1% (1/100 F; 0/100 M) in the controls (Toth, 1977).

UDMH, when administered in the drinking water, induced a high incidence of angiosarcomas in various organs; and it induced tumors of the kidneys, lungs, and liver in mice of both sexes. In rats, the same route of administration induced liver carcinomas (U.S. EPA, 1984b).

This brief review indicates there is substantial evidence for the carcinogenicity in animals of the hydrazines used at Rocketdyne. Based on the chemical structures of the hydrazines and data from genotoxicity and animal studies, there is a clear biological basis to hypothesize that hydrazines are human carcinogens.

Nitrosodimethylamine (NDMA)

NDMA and nitrosomethylamine (NMA) are produced by the oxidation of UDMH and MMH (Urry et al, 1965; Stone, 1980; Loper, 1977; Carter et al., 1981; Lunn &

Sansone, 1994). NDMA can result from vapor phase oxidation of UDMH; UDMH can be oxidized to NDMA in open air, but oxidation by NTO or other oxygenates may be another pathway for NDMA formation (ARB, personal communication). Formation and contamination from NDMA occurred at Aerojet in Sacramento, California, and the NDMA contamination represents one of the ongoing issues at that Superfund site. Groundwater and surface water NDMA contamination occurred at Aerojet. From 1955 through 1961, at least 1,023,420 pounds of UDMH were used at Rocketdyne. After 1961, there was reduced use of UDMH, but there was some ongoing use until 1970. Environmental pollution by NDMA represents an area requiring further study although Rocketdyne/Boeing samples have apparently not detected NDMA recently.

An industrial hygiene professional, Mr. Cheng, at Edwards Air Force base in California informed us that when conducting air monitoring studies around rocket-engine test sites, he observed a high occurrence of NDMA in the air during the transport and transfer of UDMH fuels. This observation is supported by the summary of the industrial hygiene special study: "Evaluation of potential exposures, conducted at the hydrazine blending facility at the Rocky Mountain arsenal in Commerce City, Colorado." In that study, air samples for UDMH and hydrazine were below levels of analytical sensitivity, yet air samples for NDMA were above the regulatory limit. Two studies were obtained from the U.S. Air Force that reported results from industrial hygiene surveys of Titan II rockets for hydrazine, UDMH, and NDMA; and they demonstrated that measurable levels of NDMA were widespread. Fine and Rounbehler (1981) reported NDMA formation in the rocket-fuel industry. Given the above mentioned reports on the presence of NDMA at other rocket-testing sites, we believe there was a potential for exposure to NDMA at Rocketdyne during the transfer of rocket fuel propellants and testing of rockets.

NDMA is considered a probable carcinogen. It is classified as a probable carcinogen by the U.S. EPA. It is classified as a substance reasonably anticipated to be carcinogenic by the NTP. It is classified as a probable carcinogen by IARC (Group 2A) on the basis of sufficient evidence in animals.

Human Studies

In 1996, DeStefani et al. conducted a hospital-based case-control study in Uruguay in which the dietary intake of NDMA and its food sources was measured in 320 cases of lung cancer and in a control group of 320 patients afflicted with diseases not related to tobacco use or diet. NDMA displayed a marked dose-response pattern, after adjusting for total energy intake and tobacco smoking, with a 3-fold increase in risk for the higher category of intake. The risks were slightly more elevated for adenocarcinomas of the lung.

Animal Studies

Oral administration results in tumors of the liver (hepatocellular carcinomas and hemaniosarcomas), kidney, and lung. Administration via inhalation produces cancers of the lung, liver, and kidney in mice. Inhalation exposure to rats results in tumors of the

lung, liver, kidney and nasal cavity. Tumors have resulted from exposure to NDMA in rabbits, guinea pigs, ducks, fish, hamsters, and newts. Tumors have resulted from different routes of administration in animal studies. Other tumor sites are the nervous system, bile duct and forestomach. NDMA is carcinogenic in all animal species tested. NDMA is highly genotoxic as well as being an animal carcinogen. Ethanol administration enhances the carcinogenicity of NDMA in mice (Anderson et al., 1992). NDMA must be considered a compound of major significance where human exposure is likely to occur, based on the large number of publications (more than 2,000) demonstrating carcinogenic risk. The evidence for NDMA as a carcinogen has now been known for approximately 40 years.

UDMH was known to contain approximately 0.1% NDMA as an impurity, but exposure to workers may also occur if NDMA is formed from oxidation of the parent compound. Unfortunately, there was no quantitative estimation of NDMA at Rocketdyne and we have no evidence regarding worker exposure to the compound. However, NDMA is considered a potent carcinogen; for example, the TD_{50} (the dose producing tumors in 50% of test animals) is 0.124 mg/kg/day. The EPA unit risk estimate for inhalation is 1.4×10^{-2} $(ug/m^3)^{-1}$.

Nitrosomethylamine (NMA)

The nitrosation of secondary amines, e.g., UDMH, is widely recognized as producing relatively stable nitrosamines, whereas nitrosation of primary amines, e.g., MMH, leads to unstable nitrosamines that break down to alcohols. There is no published evidence for the formation of NMA from rocket testing that we were able to identify, but oxidation of MMH is a pathway to NMA. Huber and Lutz (1984a; 1984b) have investigated NMA formation and reaction with DNA and concluded NMA has a sufficiently long lifetime to react with DNA *in vitro*. Results of *in vivo* studies also indicated that nitrosamine has a sufficent lifetime to methylate DNA in the stomach and small intestine of rats following oral administration of methyl amine and nitrite.

A study by Hussain and Ehrenberg (1974) indicated the product of methyl amine and nitrite produces NMA and this nitrosamine is mutagenic. There are no carcinogenicity studies of NMA because of its short life. The data on mutagenicity and DNA adduct formation would lead to a prediction that this compound would be similar to other nitrosamines and would likely be carcinogenic.

Further research on nitrosamine formation from MMH oxidation at test sights is required In our judgement exposure to NMA is likely to have been small because of the instability of the compound, but we mention it to identify areas of uncertainty.

Hydrazine Nitrosation

Lambert and Shank (1988) have suggested that reaction of endogenous formaldehyde with hydrazine may result in the formation of a nitrosamine, which further

reacts to form a formaldehyde hydrazone that is capable of methylating DNA. Methylation of DNA is considered a potentially carcinogenic pathway. The pathway proposed by Lambert and Shank requires formation of NMA; therefore, there are possible similarities between the carcinogenicity of hydrazine and MMH.

Summary

Workers at the test stands were likely exposed to 5 suspected hydrazine-related carcinogens: the three hydrazines and two products of oxidation, NDMA and NMA. Rocketdyne/Boeing has reported to UCLA that 355 soil samples from various chemical-handling locations at SSFL have been collected and analyzed for NDMA. They reported there have been no detects of NDMA in any sample. The date of this sampling was not given UCLA and we have seen no protocol to indicate the location and other details of the sampling. A RCRA Facility Investigation Work Plan Addendum for the Santa Susana Field Laboratory prepared by Ogden Environmental and Energy Services Co., Inc. dated September 1996 and obtained by a member of the Advisory Committee appears to indicate soil samples found concentrations of 40 and 20 ug/kg NDMA. We can not explain the variance between the data reported by Rocketdyne and this sampling report.

Rocketdyne/Boeing did report that NDMA has been detected at least once in 5 groundwater monitoring wells at SSFL. UCLA has not seen these data, nor do we have information about the date and locations of the sampling. Rocketdyne/Boeing reported that no NDMA has been detected since 1988 in four of the wells, and NDMA has not been detected in surface water at SSFL since a surface water monitoring program was begun in 1993. Thus, there is evidence for NDMA formation, but we have no quantitative information on employee exposure during the periods of high UDMH use prior to 1970. Additional sampling may be appropriate to rule out any environmental NDMA contamination.

Trichloroethylene

TCE Release Assessment at the Rocketdyne Facility

The probable major source of TCE exposure and contamination at the Rocketdyne facility resulted from the use of TCE as a rocket-engine cleaning agent. TCE was used in the cleaning operations of large and vernier (small) engines fueled by a propellant mixture of liquid oxygen (LOX) and kerosene (RP-1), and to clean engine components including thrust chamber components, turbo pumps, heat exchangers and gas generators. TCE was used as a utility solvent to clean up testing areas and tools as needed. For example, test personnel would use a 1/4-inch hose with spray nozzle to wash the turbo pump cell for approximately one minute. Kerosene burning engines required TCE flushing to remove residual hydrocarbon deposits and vapors left on the fuel jacket and in the LOX dome from previous tests or engine checkouts prior to ignition. These deposits and vapors are combustible and potentially explosive when exposed to LOX. Other fuels do not leave hydrocarbon residues and consequently did not require TCE flushing.

Prior to a planned test series, between each test and after the test series, large engines and thrust chamber components were flushed on the test stand with TCE. During the 1950s, testing personnel did not necessarily flush the domes after every engine test; on occasion, the surfaces of suspected areas would be wiped off with TCE impregnated rags, resulting in possible exposure by inhalation and dermal absorption. The vernier engines and other engine components were not flushed or cleaned with TCE while mounted on the test stand. Instead, they were often taken to another facility, separate from the test stand, to be cleaned by hand with TCE-impregnated rags or small pressurized TCE spray systems, also resulting in possible exposure by inhalation or dermal absorption.

Prior to 1961, catch pans and recovery systems were not used to recover TCE. The TCE that did not evaporate after flushing operations was discharged from each test stand onto a concrete spillway that drained into unlined channels and then into unlined ponds. TCE recovery systems were introduced in 1961; however, it was not until the early 1970s that they uniformly achieved the minimum percent recovery allowed by environmental permits of 85%. The initial designs of the catch pans were left up to the individual test-stand engineers. The designs varied considerably in such aspects as depth of pan and size of base, resulting in considerable variation of TCE recovery due to evaporative losses and splashing. In addition, the initial materials used for the flexible drain hose were not very compatible with TCE; consequently, leaks and breaks in the hose would occur. Additional leaks occurred due to problems in TCE transfer pipes. During the 1970s, recovery systems improved; however, a 1977 picture of an Alfa test stand shows a TCE recovery catch pan leaning against a railing on the bottom platform.

Based on the uses described above, there appears to have been significant potential for employee exposure to TCE via inhalation and dermal exposure.

Carcinogenicity

The U.S. EPA has previously classified TCE as a probable human carcinogen (Group B2/C). EPA is re-evaluating the carcinogenicity of TCE at present. IARC considers TCE to be a probable human carcinogen (Group 2A). IARC last reviewed TCE in 1995. TCE is not classified by NTP.

Animal studies

Animal studies have reported increases in lung, liver, and testicular tumors via inhalation, one of the likely sources of exposure to employees at Santa Susana. TCE has been shown to cause cancer in mice and rats. The site of tumor induction varies with the route of administration of TCE and with species. An increase in lung, liver, and testicular tumors has been observed as a result of inhalation exposure to TCE, and an increase in liver tumors was observed as a result of exposure to TCE via gavage. The principle tumor site in mice are the liver and lung, while the principle tumor site in the male rat is the kidney. When administered orally, TCE induced liver tumors in mice. TCE elicits acute

pulmonary cytotoxicity in mice, which involves Clara cells of the bronchioles.

Metabolites and Their Modes of Action

Absorption of TCE by inhalation, dermal, and oral exposure is very rapid. Once absorbed, TCE is readily metabolized by humans and animals to chloral hydrate (CHL), trichloroacetic acid (TCA), dichloroacetic acid (DCA), and trichloroethanol (TCOH)--all known toxic substances. These metabolites are thought to be responsible for the cytotoxicity and/or carcinogenicity of TCE in the liver, kidney, and lung. TCA and DCA may play a major role in liver tumor formation in mice exposed to TCE.

Human Studies

The occurrence of specific cancers was increased, at least to a small extent, in each of the following 5 cohort studies; and liver cancer, in particular, was increased in four: German study (Henschler et al., 1995), NCI study (Spirtas et al., 1991), Swedish study (Axelson, et al., 1994), Finnish study (Anttila et al., 1995), and the Hughes Aircraft study (Morgan et al., 1998). Weiss (1996) has reviewed the issue of cancer in relation to occupational exposure to TCE.

Based on these studies, there is some evidence for cancers of the kidney, liver, biliary tract, and non-Hodgkins lymphoma in humans. Axelson et al. (1994) concluded there is no evidence that TCE is a human carcinogen. Weiss (1996) concluded that evidence for the cancers described is quite limited. Further research is necessary to study the effects of high exposures to TCE and to investigate cancers of the liver, kidney, and non-Hodgkins lymphoma. The finding of cancers of the liver in both human and animal studies suggests there is a biological basis for this cancer endpoint. There is no evidence to date for cancer of the lung from inhalation of TCE in humans.

Kerosene-based Fuels

As described above, TCE was used to remove residual hydrocarbon deposits and vapors left on the fuel jacket and in the LOX dome from previous tests or engine checkouts prior to ignition. The hydrocarbon deposits result from incomplete combustion of kersone-based fuels. This incomplete combustion of fuels may result in the formation of soots containing polycyclic aromatic hydrocarbons (PAHs). Dermal absorption of PAHs is now recognized as an important source of exposure to these chemical agents in other industries (Van Rooij et al., 1993a; 1993b).

PAH formation has been demonstrated from kerosene combustion, but these examples were under relatively low temperature conditions, e.g., kerosene heaters. We have not identified any literature demonstrating PAH formation from rocket testing of kerosene fuels. UCLA was able to obtain a report from an Advisory Committee member submitted to the Ventura County Air Pollution Control District for 1992. This report indicates a small amount of PAHs were released from the facility. Further investigation to

rule out the hypothesis that worker exposure may have resulted from PAH contamination may be useful. We were not able to obtain information on this issue from other sites to determine if there is potential for inhalation or dermal uptake of PAHs.

Other PAH Exposures

In the mid-1970s, the bowl area was deactivated, but it was later reactivated in the late 1970s. During the interim period, the area was used for coal gasificiation and liquification. In 1976, coal hydroliquification was carried out. This process included placing of pulverized coal into a high pressure environment to produce aromatic and 2-ring PAH compounds. The liquids produced were then transferred to 55-gallon drums. Employees were exposed to the compounds principally during transfer of the aromatic hydrocarbons. Exposure would have occurred via both dermal contact and inhalation.

Rock coal was ground through a fine mesh in a pressurized system to obtain pulverized coal. Employees would have been exposed to particulate matter during this operation. These operations occurred between 1976 and 1981.

The control center building (B-900) was used to carry out a coal hydrogasification process from the mid-1970s to 1981. In this process, pulverized coal was fed into a pressurized system to produce methane gas. The coal was cooked for a longer period than in the coal hydroliquification process. In addition to methane, other hydrocarbons, including benzene, were likely produced, but we do not have information on the nature and amounts of their various side products. The heavy residues from these processes were further refined. Exposure to aromatic hydrocarbons and some PAHs may have occurred during these processes.

Asbestos

Asbestos is a known human carcinogen. Exposure to asbestos results in cancer of the lung, mesothelioma, and perhaps other sites. Asbestos was used at the Santa Susana Field for insulation and floor tiles. High-temperature asbestos wires and asbestos-wrapped lines were found at the Bravo, Atlas, Delta, Coca, and Bowl test stands. The same asbestos lines were located at APTF, Building 206, and STL-4. In these areas, workers (mostly with the job title of mechanic or technician) would repair torn lines and thermocouple cables with asbestos insulation around the test areas. UCLA industrial hygienists noted there were likely more buildings associated with asbestos use, principally as insulation. Exposure to asbestos would have occurred from cutting, removing, and installing asbestos materials.

A employee interviewed by a UCLA industrial hygienist is illustrative: She stated that she used to file asbestos containing material. She stated there were asbestos wires at the test stands and that they would break apart due to the high heat conditions. She was personally involved in cutting, removing, and installing of asbestos insulation.

UCLA was not able to obtain quantitative information on asbestos monitoring, which was required by the OSHA standard on asbestos, but it appears highly likely there was exposure to asbestos at the test stands by personnel involved in installation and repair of asbestos insulation.

Beryllium

UCLA industrial hygienists were informed by management that beryllium was used at the Happy Valley area from 1962 to 1967. Powdered beryllium was reported to have been mixed with oxidizers for use in rocket propellants (B-917). Beryllium was burned as a solid propellant in a tank ventilated with HEPA filters and a water scrubber system. This work was conducted in a closed system to minimize employee exposure. The residue collected on the HEPA filters was gathered and disposed of by employees. Respirators were used to minimize exposure, and workers' clothes were removed on site and sealed in bags for cleaning. Showers were required every day during operating periods.

UCLA was not able to obtain monitoring data from this period and cannot estimate the degree of personal exposure that may have occurred to beryllium, but it is likely that some exposure may have occurred. We have obtained one incident report on beryllium which occurred in 1967 when a malfunction resulted in 0.87 pounds of beryllium being released. The report indicates there were no personal exposures and wipe tests indicated resultant contamination was within acceptable levels.

Beryllium and beryllium compounds are carcinogenic to humans according to IARC. The U.S. EPA considers beryllium a probable carcinogen (group B1). There is considerable evidence that the current occupational standard for beryllium is inadequate to prevent occupationally related beryllium lung disease and the U.S. Department of Energy (DOE) is currently developing a new occupational beryllium standard. Dr. Froines served on the Federal Advisory Committee on beryllium established by the Secretary of DOE to address health issues associated with workplace exposure to beryllium.

Control of beryllium exposure is very difficult, and the most advanced facilities in the world have difficulty keeping exposures below the current standard, even today when the hazards of beryllium are more widely recognized. UCLA is currently working closely with Los Alamos National Laboratory in the design and maintenance of a beryllium facility used in the production of nuclear weapons, and the control of beryllium exposure is a matter of concern. It is very unlikely that the same level of control would have occurred in the 1960s when the general level of sophistication and concern were more limited. We conclude there may have been exposure to beryllium during this period, but it is unclear of the magnitude and duration.

Conclusion

Over the period of this study, there may have been exposure to hydrazines, nitroso compounds, TCE, beryllium, and asbestos among test-stand personnel. There is

toxicologic evidence for all of these compounds being capable of producing lung and/or other cancers. With the exception of TCE, all the compounds might be considered probable human lung carcinogens.

The lack of quantitative industrial hygiene information prevents the UCLA investigators from making estimates of actual employee exposures, but there is sufficient anecdotal evidence from interviews and records that exposures did occur to the chemicals described above.