A CASE STUDY IN AVOIDING A DEADLY LEGACY IN DEVELOPING COUNTRIES

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"All scientific work is incomplete — whether it be observational or experimental. All scientific work is liable to be upset or modified by advancing knowledge. That does not confer upon us a freedom to ignore the knowledge we already have, or to postpone action that it appears to demand at a given time" (Hill, 1965).

INTRODUCTION

Industrial expansion has always brought with it massive change, not only to the economy of a nation, but to the lives of people in the affected country. The transfer of technology from the industrialized world to the developing world involves many risks. Damage to the health and safety of workers and their families may ultimately be the most severe. Countries experiencing industrialization are changed from agrarian disperse populations to urban convergences, often with resultant poverty, malnutrition, inadequate housing, and related diseases. People who are expected to benefit most by industrialization are often the ones most adversely affected.

While the Industrial Revolution of the current industrialized world took several hundred years to complete, the revolution now taking place in the developing world is occurring much more rapidly and is escalating with a momentum that is not likely to decrease any time soon. The developing world, adjusting to its new found "benefaction," may not be able to cope with such rapid change. While industrialization may indeed be related to economic expansion and monetary gain, it is often realized without consideration for the long-term consequences. Placing blame for the problems associated with rapid expansion will become the province of the politicians. Future foreign policy for the industrialized world as it relates to the developing world will undoubtedly become enmeshed in debates over who is to blame. Some concern is now surfacing about such issues and plans of action have been suggested (ILO, 1988).

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Are the lessons of the first industrial revolution relevant to industrialization of the developing world? Historians tell us that "the present is the past rolled up for action, and the past is the present unrolled for understanding" or "Is it possible that, after all, 'history has no sense,' that it teaches us nothing and that the immense past was only the weary rehearsal of the mistakes that the future is destined to make on a larger stage and scale?" (Durant and Durant, 1968). Translating this to public health, what can we learn from the history of asbestos that will prevent a tragic legacy from continuing in developing countries which now import asbestos for domestic use?

PROBLEM

The exportation of asbestos to developing countries is taking place partly because of stricter regulation of its use in the industrialized world, and also because there is little regulation of its use in the developing world. The vast market for its use, along with the seemingly unlimited low cost labor pool of the developing world, play an important role in creating products made from asbestos. In most of the developing countries, principle uses of asbestos are not primarily for its insulation properties (as was the case in the industrialized world), but its abundance on the world market as a building material for dwellings and potable water pipe. The asbestos industry touts such uses by saying that "While I doubt that exposure to asbestos from drinking water will lead to any significant increase in cancer risk, I can say with confidence that the reduction in the availability of potable water to the Third World's children will ensure that thousands will never survive long enough to have that worry" (Helmer, 1989). As the asbestos industry campaigns for the use of asbestos in cement pipe for the transport of potable water, they neglect to mention the adverse effects of inhaling asbestos fibers by the workers who will manufacture or install such products. Their advertising campaigns also neglect to note the possibility of carrying home the deadly material to household members, or that its indiscriminate disposal may lead to the exposure of many non-workers.

It appears that Canada, a major asbestos producer, has chosen to target Thailand for the exportation of asbestos. According to Sentes, Thailand wages are generally less than $3 per day, and less than 6% of the workers are unionized (Sentes, 1989). Public officials, including Thailand's energy minister, encourage using asbestos, and soft peddle its health risk when he noted "It's an emotional issue for some people . . . why so much focus on asbestos? Because it was badly used a couple of years ago, which is not the way it's used now. So it's totally different." Not so, according to the Canadian Broadcast Company's (CBC's) fifth estate television crew who found "Thailand's official exposure levels clearly aren't being enforced . . ." (Sentes, 1989). Certain Thai government officials contradicted the energy minister. Dr. Virah Mavichak, the official responsible for asbestos control, said, "We don't have the proper equipment, we don't have the trained people . . . [workers] don't know exactly what are the effects caused by asbestos and employers may not know how to control the fibers" (Sentes, 1989). Personal experience by one of us (Richard A. Lemen), surveying a jointly-run Thai-United Nations refugee camp at Phanat Nikhom, Chonburi Province, Thailand, where workers

and refugees were building housing made of asbestos wall board and roofing shingle, confirms the findings related in the Sentes article. Workers were unaware of the dangers related to asbestos, there were no controls to reduce exposures, and they were not provided with protective clothing or gear. Further, they were allowed to wear their clothing home, unwashed and contaminated with asbestos (Lemen et al., 1984). Until such indiscriminate use of this highly hazardous material is curtailed, countless unsuspecting workers and their families will pay a high personal cost for potable water and cheap housing in the temporarily advantaged population. The wholesale abandonment of asbestos for safer substitutes will not eliminate the hazards from the uncontrolled use of the existing asbestos which will still be present for years to come. The substitutes themselves may have properties similar to asbestos, and could pose similar health risks (IPCS, 1988).

What, then, are the solutions to prevent the occurrence of a second international tragedy which would result from the indiscriminate and uncontrolled usage of asbestos in the developing world? To answer this question, it is necessary to examine the legacy of asbestos and then develop the epidemic scenario of events that led to the present state of affairs concerning asbestos in the industrialized world.

ASBESTOS MINING, PRODUCTION, AND USES

Asbestos is a term derived from Greek, meaning inextinguishable (American Heritage Dictionary, 1985). The use of asbestos dates back to 2500 B.C. (Noro, 1968) when asbestos fibers were used to make pottery. One of the earliest descriptions dates from the fifth century B.C., when asbestos was used in the wick of a gold lamp. Other uses were described by Theophrastus (372–287 B.C.), Strabo (63 B.C.–24 A.D.), Pliny the Elder (23–79 A.D.), and Dioscorides (40–90 A.D.) (Strack, 1941; Hoover and Hoover, 1950). Pliny described the weaving of asbestos fibers into cloth that was used to wrap the corpses of apparently very wealthy persons. He referred to it as the "funeral dress of kings" (Cirkel, 1910).

Asbestos deposits have been found in the European Alps since ancient times. The spinning and weaving of the mineral fibers were practiced by the Romans, Vikings, and the medieval emperor, Charlemagne, born in 742 A.D. Charlemagne was reputed to have thrown a used table cloth into the fire to clean it and then recovered it unharmed, ready to be re-used (Jones, 1897; Durant, 1950; Murray, 1990). Marco Polo described how natives of one of the northern provinces of the Great Khan wove an indestructible cloth from what some believed to be salamander skin, but in actuality " . . . is not a beast or serpent, for it is not true that these cloths are of the hair of an animal which lives in fire, as one says in our country, but is a vein of earth" (Collis, 1949; Latham, 1958). Benjamin Franklin even had a purse made from tremolite asbestos (or, as it was described, "stone asbestos" or "salamander cotton") which he took to London in 1725, prior to any knowledge of asbestos deposits in North America. Most likely, the purse was originally acquired in Europe, taken to America, and then brought back by Franklin (____, 1939). During the reign of Peter the Great in Russia (about 1720), the discovery of deposits of asbestos in the Urals led to the production of textiles, socks, gloves,

and handbags. This continued until the demand lapsed, about fifty years later (Cirkel, 1910). It is reported that the Italians advanced the techniques of producing asbestos paper and cloth, and that Pope Pius IX set up a paper mill to produce paper that would be used to protect vatican documents from fire (Jones 1897; Cirkel, 1905). Other uses of asbestos have been described and, by some accounts, exceed 3,000 (IARC, 1977).

The modern industry dates from about 1880, when asbestos was used to make heat- and acid-resistant fabrics (Hendry, 1965; Hueper, 1965). A professional journal article published in 1883 discusses the use of asbestos in packings for steam engines; it was thought that asbestos was first used in this manner by the British in 1879, and then by the Germans. The article also reported that asbestos was used in soapstone for locomotives, as a base for filter material, in millboard for fire protection and electrical insulation, in cement and putty, in elements for gas fires, and that it was woven into theater curtains. There was no mention of adverse health effects (____, 1883).

In a paper by Fisher (1892), there is a discussion of the mineral deposits of asbestos throughout the World, including Newfoundland, the United States, South and Central America, China, Japan, Australia, Spain, Portugal, Hungary, Germany, Russia, and both the Cape and Central Africa. However, Fisher considered the best deposits to be located in Italy and Canada. The first commercial mine in Italy was opened in 1870, and the last mine closed in 1989 in Balangero (Murray, 1990). The mines were located high in the mountains where the only concern of occupational safety or health for the miners was related to landslides and avalanches (Fisher, 1892). In 1860, asbestos was found in a deposit in the Des Plantes River region of Canada and a sample of this asbestos was displayed at the International Exposition in London in 1862 (Bowles, 1955). These deposits were not mined until 1878, when about fifty tons were produced. This asbestos was of high quality, with good spinning ability. Subsequently, these characteristics became highly marketable in the United Kingdom where the earliest asbestos manufacturing company had opened in Glasgow in 1871 (Fisher, 1892). The Canadian market grew from its original annual yield of fifty tons to 911,226 short tons by 1953, with an average value of $65 in 1879 to approximately $95 per short ton in 1953 (Fisher, 1923; ____, 1953).

Two basic fiber classifications of commercial importance exist for asbestos. They are the fibrous serpentine mineral, chrysotile, and the fibrous amphiboles – actinolite, cummingtonite-grunerite including amosite, anthophyllite, crocidolite, and tremolite. A thorough review of the mineralogy of the asbestos minerals can be found elsewhere (Campbell et al., 1977; IARC, 1977; Selikoff and Lee, 1978; Pooley, 1987).

Crocidolite (meaning, "woolly stone") asbestos was first discovered in the early 1800s, northwest of the Cape Province of South Africa. However, it was not actively mined until World War II, when the demands for asbestos increased (Hall, 1930; Cilliers and Genis, 1961; Sleggs et al., 1961; Sluis-Cremer, 1970). A second deposit of asbestos was found in South Africa around 1904 in northeast Transvall and, in 1918, was named *amosite* for the village

Amosa, the acronym for *Asbestos Mines of South Africa.* Production began in the mid-1920s by Cape Asbestos Company, the same company which was mining and producing crocidolite (____, 1958; Sluis-Cremer, 1970). Most of the labor for the mining of amosite was generated by the native Africans, as reported by Cilliers (1963), who claimed that 35 Europeans and 6,500 native black Africans worked at the largest group of mines. Production of asbestos in Russia also began during the mid-1920s, even though the discovery in the Urals had occurred during the time of Peter the Great (____, 1948; Korepin and Med'nikov, 1967).

Production of asbestos in the United States was never significant when compared to that of Canada, Italy, Russia, or South Africa. The earliest recorded listing of asbestos in the United States was in 1861 in Vermont (____, 1948). The deposits found in the United States were chrysotile. Deposits were later found in Arizona, California, Georgia, Nevada, and North Carolina. In 1970, production in the United States was 3.3% of the world market, just behind Italy (____, 1948; Clifton, 1972).

Highlights in the production history of asbestos include: the first processing in 1890 of Canadian asbestos into textiles in the United States (Berger, 1963); the origins of the asbestos cement pipe industry in Italy at the turn of the twentieth century (____, 1973); the beginning of asbestos cement production in the United States in 1903 (Berger, 1963); production of flat asbestos cement board in 1904 in the United States (____, 1958); the first use of asbestos as a brake lining in 1906 (____, 1953); importation of the first pipe-making machines in 1928 into the United States (Berger, 1963); the use of asbestos protective clothing in World War II, between 1939 and 1946 (____, 1946); and asbestos spraying of deckheads and bulkheads in British Navy Ships in 1944 (Harries, 1968, 1971).

It is interesting to note that the British Navy decided in 1891 to discontinue the use of cotton silicate used for boiler insulation, partly because it was found to cause respiratory irritation, but also because, with increasing temperatures, it no longer provided adequate insulation properties. The recommended substitute was asbestos (Wright, 1980). In 1963, the spraying of British Navy ships with asbestos was discontinued (Harries, 1968, 1971).

Japan is the world's largest user of asbestos (____, 1989). World production and consumption of asbestos peaked in 1976 and declined only slightly during the early 1980s. World production was at 4.5 million tons in 1985 (Stack, 1989), dropped to 4.2 million tons in 1988 (AI, 1989), and was projected to be 4.4 million tons in 1990 (Stack, 1989). The USSR, the world's major producer during this period, mined 60% of the world's asbestos (AI, 1989). Production levels for the main producers declined. Canada's output decreased 30% between 1981 and 1985, or from 1.1 million tons to 0.7 million tons (WHO, 1989). A similar decrease occurred in Zimbabwe and South Africa (WHO, 1989). Over the same time period, asbestos production increased in countries that previously had lower production levels (WHO, 1989).

South Africa is the only country producing the amphiboles, and essentially all of it is being exported (Becklake, 1987). According to the World Health Organization (1989), demand for asbestos is increasing in South America, Asia, and the Middle East, accounting for one-third of the world's demand. World asbestos production for 1988, by country, can be seen in Table 1.

TABLE 1. Asbestos World Production for 1988

Country	Metric Tons
Brazil	230,000
Bulgaria	600
Canada	704,989
China	157,478
Colombia	10,500
Cypress	18,000
Egypt	450
Greece	72,000
India	26,500
Italy	100,000
Japan	4,000
S. Africa	145,690
S. Korea	2,428
Taiwan	1,400
U.S.S.R.	2,560,000
United States	18,000
Yugoslavia	10,500
Zimbabwe	90,560

Source: The Asbestos Institute, 1989.

Asbestos cement products make up 70% of the commercial application for asbestos. Other major uses include: vinyl asbestos flooring (10%); friction products (7%); asbestos paper and felt (5%); gaskets and packings (3%); paints, roof coatings, caulks, etc. (2%); filter media (2%); asbestos textile products (1%); and all other uses less than 1% (IPCS, 1988). The major type of asbestos used throughout the world today is chrysotile; the amphiboles comprise less than 3% of the asbestos in use today (IPCS, 1988).

TYPES AND NUMBERS OF WORKERS

First estimates for the number of U.S. workers exposed to asbestos in their work environment were 3–5 million (NIOSH, 1972). While no actual surveys had been conducted, it was estimated that the most direct exposures were to miners (1,000), workers in manufacturing of asbestos-containing products (50,000), and construction workers (40,000) (NIOSH, 1972). The most recent estimate for potential exposure of persons in the work environment is 395,181; 31,764 of these workers are women. Construction and transportation-related industries account for the greatest numbers of workers (mostly men) exposed to asbestos.

Women are most likely to be exposed while working in the manufacturing sectors of industry, particularly in the manufacture of electric- and electronic-equipment, and instruments and related products. After examining specific job categories, it appears that assemblers, roofers, carpenters, and construction laborers constitute the top four categories of workers who are potentially exposed to asbestos. Broken down by job category and by sex, men are mostly exposed to asbestos while working as roofers, assemblers, carpenters, and construction laborers; women are mostly exposed while working as assemblers. The limited data currently available on fiber type indicate that most potential exposures are to chrysotile (NIOSH, 1990).

Exposures to asbestos in the mining and milling segments have ranged from 1.1–5.1 fibers/ml in the mines, with a mean of 2.5 fibers/ml, while the concentrations increase during the milling of the asbestos ore. Some mill processes have reported fiber concentrations up to 189 fibers/ml, with means as high as 70 fibers/ml (Selikoff and Lee, 1978). It is difficult to historically determine asbestos concentrations found in industry, except for cases in which exposure measurements have been made for compliance purposes. These generally represent cases that do not comply with existing asbestos standards. The only major cross-sectional industry-wide approach of the asbestos manufacturing industry was conducted by the U.S. Public Health Service between 1964 and 1972 (NIOSH, 1972). In that survey, hundreds of personal asbestos sampling devices were worn by workers to determine time weighted average (TWA) exposures to asbestos. Such an industry-wide study has not been conducted since. Six asbestos manufacturing industry sectors were studied, including seven asbestos cement pipe plants; five friction plants; three cement shingle, millboard and gasket manufacturing plants; one paper, packing, and asphalt products plant; five insulation plants; and eight textile plants. The highest mean concentrations were found in the mixing areas of the insulation plants (74.4 fibers longer than 5μm/cc of air). In most cases, the dirtiest jobs occurred at the initial part of the manufacturing process. These processes usually involved handling the asbestos in non-enclosed areas, in bulk quantities and in a dry state. As the manufacturing process continued, the asbestos was usually combined with another material, either binding it to the other material or putting it into a wet slurry. Higher exposures did not usually occur again until the product was in the finishing process, when it was either sawed or trimmed in other ways to complete the product for sale.

Worker exposure has also been found in the shipyard industry, the construction industry, the brake repair and transportation industry, the electronic and electrical industries, the paint industry, the optical goods industry, and other general industry manufacturing sectors (NIOSH, 1990). Exposures have varied with the use of the asbestos-containing materials, and are sometimes elevated, as in the brake repair industry (Lloyd, 1975; Nicholson et al., 1982). Exposures in the construction industry can also be elevated, as shown in the study by Reitze et al. (1972) when they measured fiber counts from spraying asbestos onto buildings. They found 70 fibers/ml at 10 feet and 3 fibers/ml at 25 feet from the nozzle of the spray gun. This indicates that not only were the spray operators at risk, but the auxiliary workers, such as

carpenters, pipe fitters, welders, electricians, plumbers, etc., were also at risk (Reitze et al., 1972).

Familial exposures to asbestos can occur when the worker brings home asbestos-containing material from the worksite, or when the worker does not shower or wears the same clothes home that have been worn during the work process. These practices were common until it was recognized that they could lead to familial exposure, resulting in asbestos-related disease and mortality (Wagner et al., 1960; Newhouse and Thompson, 1965; Nicholson, 1975; Anderson et al., 1976). Further, domestic exposures have been associated with household repairs and do-it-yourself construction jobs that either use products which contain asbestos or that cause the disturbance of asbestos-containing materials (Rohl et al., 1975).

EARLY KNOWLEDGE OF THE HEALTH EFFECTS

With modern asbestos usage, reports of asbestos-related diseases began to be recognized. The first recorded case of asbestosis was reported by Montague Murray in 1906 (Murray, 1907). It is interesting to note that Adilaide Anderson, Lady inspector of Factories, included asbestos among the dusts known to cause injury to man, in a 1902 publication on dangerous industries in England. (Anderson, 1902). In 1918, American and Canadian insurance companies would not insure asbestos workers, due to the unhealthy conditions in the industry (Hoffman, 1918). The first complete description of asbestosis, including naming the disease and "curious bodies" observed in lung tissue, appeared in 1927 (Cooke, 1927; McDonald, 1927). Mills (1930) reported the first case of asbestosis in the United States, and in the same year, Lynch and Smith (1930) reported on "asbestos bodies" found in the sputum of asbestos-exposed workers. Other early studies led many investigators to conclude that people who were exposed to asbestos dust developed the disease "asbestosis" if the dust concentrations were high or if their exposures were lengthy (Merewether, 1930; Merewether and Price, 1930; Fulton et al., 1935; Dreessen et al., 1938).

Lynch and Smith (1935) in the United States, and Gloyne (1935) in the United Kingdom, both associated occupational asbestos exposure with lung cancer. Approximately ten years later, case reports appeared of pleural and peritoneal tumors associated with asbestos exposures (Wedler, 1943a, 1943b; Wyers, 1946). In 1955, Doll reported a ten-fold excess risk of lung cancers in those United Kingdom asbestos textile workers who had been employed before 1930. Similar findings appeared in the United States for excesses of both lung cancer and mesothelioma (Mancuso and Coulter, 1963; Selikoff et al., 1964). Possible variations in the risks associated with different fiber types were rarely considered in these early reports.

HEALTH EFFECTS — ANIMAL DATA

The animal studies of exposure to asbestos have produced fibrotic lesions, lung tumors, and pleural and peritoneal tumors (mesotheliomas). However, none of the animal studies have produced gastrointestinal or other cancers (WHO, 1989). With one exception, all of the animal

bioassays have mimicked human epidemiologic results. That exception pertains to the development of mesothelioma. Most human exposures to chrysotile have actually been to a mixed variety of asbestos, making it impossible to determine which fiber exposures were responsible for the mesotheliomas. However, because of the nature of animal bioassay studies, control for fiber type can be defined accurately, which in turn provides much more definitive results. Wagner et al. (1979) demonstrated that a commercially grade 7 (predominantly short) Canadian chrysotile can induce mesotheliomas when injected intrapleurally into rats, and primary lung neoplasms when the animals were exposed by inhalation. This further supports Wagner et al. (1973, 1974) data which demonstrate that not only is chrysotile as potent as crocidolite asbestos fibers and other amphibole fibers in inducing mesotheliomas after intrapleural injections, but it is also equally potent in inducing pulmonary neoplasms after inhalation exposures.

In terms of degree of response related to the quantity of dust deposited and retained in the lungs of rats, Wagner et al. (1974) found chrysotile was much more fibrogenic and carcinogenic than amphiboles. Finally, the question of which fibers produce the carcinomas appears to best be answered by Stanton and Wrench (1972), who have shown that durable fibers with diameters less than 1.5 μm and lengths greater than 8 μm are carcinogenic in experimental animals, regardless of their physicochemical properties (Murray, 1990).

Chrysotile fibers are much more chemically and biologically reactive than amphibole fibers. Because of this reactivity with the tissues, they lose their structural elements and divide into smaller fibrils, making it difficult to recognize them by the usual analytical methods. In fact, many of the fibers are removed from the lung and exhaled back through the bronchi, or travel via the lymphatic system to other organs of the body (Davis, 1979; Davis et al., 1986a, 1986b; Marten et al., 1989). The concentration of dust in the lungs of rats exposed to Canadian chrysotile was only 1.8%–2.2% that of the dust concentration in the lungs of animals exposed to amphiboles (after 24 months of inhalation exposures), yet the lung tumor incidence and degrees of pulmonary fibrosis were similar in all groups. The reasons for the higher incidence of lung cancer and mesotheliomas in workers exposed to amphiboles is, therefore, probably related to higher concentrations of respirable fibers during their exposures (NIOSH/OSHA, 1980). These findings support the idea that chrysotile fibers cause more cellular injury, fibrosis, and lung cancer, while at the same time are less readily detected in the tissue after the damage is done. Churg et al. (1989a) conclude that the failure of chrysotile to accumulate in the lung is a result of preferential chrysotile clearance during the first few days to weeks after exposure, and that dissolution plays no role in the clearance. Further, the preferential clearance may be a result of fragmentation and rapid removal of the chrysotile fibers. Malorni et al. (1990) suggest that fiber penetration can rearrange the cytoskeletal apparatus of the cell, and that, since giant multinucleated cells are formed, this could indicate an interaction between the chrysotile fibers and the normal mitotic process. Churg et al. (1989b) further believe that the short fibers may be more fibrogenic than previous animal data indicate and that further study is needed.

HEALTH EFFECTS — EPIDEMIOLOGICAL EVIDENCE

All of the commercially important fiber types have been responsible for nonmalignant respiratory disease and lung cancer in man and, with the exception of one fiber type, anthophyllite, all have been reported to cause mesothelioma. Following is a list of fiber types and various studies that support their relationship to mesothelioma:

- Crocidolite - Wagner et al., 1960; McNulty, 1962; Harrington et al., 1971; Webster, 1973; Jones et al., 1976; Armstrong et al., 1988
- Chrysotile - Kogan et al., 1972; Borow et al., 1973; Enterline and Henderson, 1973; McDonald, 1973a, 1973b; Wagoner et al., 1973; McDonald et al., 1974; Liddell et al., 1977; McDonald, 1977; Selikoff, 1977; Nicholson et al., 1979; Robinson et al., 1979; Rubino et al., 1979; Boutin et al., 1980; Dement et al., 1983; McDonald et al., 1983; Huncharek, 1987; Piolatto et al., 1990
- Amosite - Selikoff, 1976; Seidman et al., 1977; Johnson et al., 1983; Seidman et al., 1986; Finkelstein, 1989; Ribak et al., 1989
- Tremolite - McDonald et al., 1986; Amandus et al., 1988
- Anthophyllite - Meurman et al., 1974, 1979).

The report of a cohort studied by Tuomi et al. (1989) has raised some question concerning the conclusion that no association exists between exposure to anthophyllite and the occurrence of mesothelioma. It should also be emphasized that most of the earlier studies suffered from mixed exposures, and that what the authors reported was the predominant fiber type; this was particularly true for the chrysotile cohorts.

Exposures to mixed fiber types have also been associated with bronchial cancer, as well as pleural and peritoneal mesotheliomas reported from several countries, including the United Kingdom (Newhouse, 1969), Federal Republic of Germany (Bohlig et al., 1970), United States (Selikoff et al., 1970), The Netherlands (Stumphius, 1971), and Italy (Rubino et al., 1972). The relative risk for lung cancer has varied from 1.0 (Knox et al., 1968) to 17.6 (Elmes and Simpson, 1971), with an average 9.8 relative risk. The prognosis and treatment of asbestos-induced lung cancer is no different than lung cancer having another etiology. It appears that all cell types of lung cancer occur in asbestos workers, and that the presence or absence of one cell type cannot be used to prove or disprove an association of asbestos exposure with the lung cancer (Churg, 1985).

Recent reports have appeared in the scientific literature that suggest that chrysotile is less pathogenic than the amphiboles (Mossman and Gee, 1989; Mossman et al., 1990). They speculate that the difference in the pathogenicity may be due to differences in patterns of deposition in the lung, the subsequent degree of fiber clearance from the lung, as well as the difference in fiber solubility while in the lung.

A review of available epidemiology studies on chrysotile was conducted by Doll and Peto (1985) in which they concluded that the studies show that exposures to chrysotile alone produced less lung cancer and mesothelioma than did mixed exposures or exposures with amphiboles. The studies did not comment on the ability to produce fibrosis of the lung.

A recent review by the WHO (1989) differs somewhat with the conclusions of Doll and Peto (1985). Based on the human evidence, the WHO suggests that the risk of lung cancer from chrysotile is less than that from exposures to crocidolite or amosite, but also states in a report that "...it is difficult to substantiate this difference firmly after standardization for exposure levels, type of industry, duration of employment, etc." This conclusion agrees with the conclusions of the 1984 *Report of the Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario,* which concludes that "As for chrysotile, it is necessary to distinguish the industrial processes in which asbestos is being used." Some of the highest risks to workers for lung cancer have occurred in the asbestos textile production industry, where the Commission recommended that chrysotile "be banned." In fact, the Commission concluded that "All fiber types can cause all asbestos-related diseases, . . ." (Dupré et al., 1984). This concept of looking at the particular use of chrysotile asbestos and its related potency is supported in the paper by Huncharek (1989). He supports this contention with the reported cases of mesothelioma among brake mechanics (Langer and McCaughey, 1982; Huncharek, 1987). Mancuso (1988, 1990) contends, based on his analysis of railroad machinists, that commercial chrysotile asbestos has caused mesotheliomas and that the risk is greater than previously asserted.

Occurrence of mesothelioma in the pleural area, when compared to the peritoneal area, appears to be associated with the degree of exposure (Newhouse et al., 1972). In the peritoneal area, mesothelioma occurs as a result of heavier exposures (Selikoff et al., 1970). Among the number of occupationally exposed groups studied, approximately 5–7% of the deaths were due to mesothelioma (Gilson, 1973; Hammond and Selikoff, 1973; Selikoff, 1976). Another estimate has projected that as many as 11% of all asbestos workers' deaths in England will be from mesotheliomas (Newhouse and Berry, 1976).

Other malignant disease patterns have also been reported in epidemiological studies of asbestos workers, the most common of which is gastrointestinal tract cancer, with a relative risk of 0.5 (Meurman et al., 1974) to 3.1 (Mancuso and El-Aher, 1967; Selikoff, 1974). Reports of gastrointestinal tract cancers associated with asbestos exposure have been reviewed by the WHO (1989), which concluded that "overall, there seems that there is a correlation between lung cancer and gastrointestinal cancer rates in occupational cohorts [exposed to asbestos] which is not due to chance." The WHO is uncertain whether these two cancers are due to the same factor "asbestos," and implies that the answer is still open for debate (WHO, 1989).

Other diseases reported in asbestos-exposed workers include laryngeal cancer (Newhouse and Berry, 1973; Stell and McGill, 1973; Shettigara and Morgan, 1975; Morgan and Shettigara,

1976; Doll and Peto, 1985); oropharyngeal cancer (Selikoff et al., 1970); multiple primary cancer (Dohner et al., 1975); suicides (Wagoner et al., 1973; Robinson et al., 1979); ovarian cancer (Parkes, 1973; Acheson and Gardner, 1983; Doll and Peto, 1985); renal cancer (Selikoff et al., 1979; MacLure, 1987); penile cancer (Raffn and Korsgaard, 1987); bladder cancer (Bravo et al., 1988); breast cancer (Doniach et al., 1975) and leukemia, multiple myeloma and Waldenstrom's Macroglobulinemia (Gerber, 1970; Parkes, 1973; Kagen et al., 1977; Kishimoto and Okada, 1988). Edge (1976) reported that shipyard workers with mixed asbestos exposures and pleural plaques (without evidence of pulmonary fibrosis) had a 2.5 times greater risk of developing carcinoma of the bronchus when compared to the matched controls without plaques. Further research needs to be conducted to determine if these are chance findings or repeatable findings resulting from exposure to asbestos. Regardless of the final magnitude of these associations, it is clear that asbestos is a fibrogenic and carcinogenic agent and, therefore, poses a hazard to those who are exposed to it.

RISK TO WORKERS

Since the cohort studies generally involved workers who were exposed more than three or four decades ago, when routine measurement of asbestos was not practiced, dose-response relationships have been extremely difficult to establish. In addition, the analytical methodology changed from measuring total dust particles to counting individual fibers. The first study to suggest a "guidance limit" for asbestos used the midget impinger counts to estimate dust exposure (Dreessen et al., 1938). This methodology was gravimetric in nature and included all dust particles, whether they were grains or fibers. This continued as the method of choice until the 1960s when it was replaced by the "membrane filter" method, which utilized the collection of dust on a membrane filter, then dissolved the filter and counted the fibers under a phase contrast microscope (NIOSH, 1972). Of the two, analysis methodologies failed to show a consistent conversion factor for fiber mass to fiber count (NIOSH, 1976). This is still generally the case; however, in a study by Dement (1983), conversion factors were fairly accurate when done according to job and operation within one specific factory. It should be noted that neither method can determine the specific concentration of asbestos since neither is capable of differentiating an asbestos fiber from other fibers of similar dimension and size.

Risks for the development of non-malignant and malignant diseases vary considerably. The higher the exposures, the greater the risks; the lower the exposures, the lower the risks. The earlier "guidance limits" were established, based on little or no prevalence of malignant disease (Dreessen et al., 1938; ACGIH, 1946). Currently, exposure limits are set because of the concern for malignant diseases. An example is the rationale of the Occupational Safety and Health Administration in setting their standard for asbestos (OSHA, 1986). In this standard, the exposure limit was set, in part, after a risk assessment showed a decreased lifetime risk from 64 deaths per 1,000 at 2 million fibers/cc to 6.7 deaths per 1,000 at 0.2 fiber/cc. Even at the current NIOSH recommendation of 0.1 fibers/cc, the risk of death is 3.4 per 1,000 at 0.1 million fibers/cc (OSHA, 1986). With the OSHA limit, it can clearly be seen that the risk for

dying from cancer is not zero, nor does it even approach zero. The WHO (1989) is but the latest to state that "[T]he human evidence has not demonstrated that there is a threshold exposure level for lung cancer or mesothelioma, below which exposure to asbestos dust would not be free of hazard to health." The implication is that no exposure limit will suffice in the complete protection of workers exposed to asbestos.

There is marked enhancement of the risk of lung cancer in workers exposed to asbestos who also smoke cigarettes (Selikoff et al., 1968; Berry et al., 1972; Hammond and Selikoff, 1973; Hammond et al., 1979). Data from Weiss (1971) and Hammond et al. (1979) suggest that cigarette smoking may also contribute to the risk of asbestosis. Smoking, however, has not been found to be associated with an increased risk of pleural or peritoneal mesothelioma, or cancers of the stomach, colon, and rectum, which occur with equal frequency among smoking and non- smoking asbestos workers.

REGULATORY ACTIONS

The first regulations for asbestos were developed jointly by the United Kingdom government and the industry being regulated, but were not applied to most users of asbestos (____, 1931). The first recommended guidance concentration for asbestos was 5 mppcf, and was provided in a 1938 report by the United States Public Health Service (USPHS) (Dreessen et al., 1938). This was followed by the adoption of the 5 mppcf by the American Conference of Governmental Industrial Hygienists (ACGIH) in 1946 (ACGIH, 1946). Regulation did not take place in the United States until 1960 (U.S. DOL, 1960). The chronology of asbestos regulation in the United States can be seen in Table 2.

Currently, the regulated concentrations for asbestos vary widely throughout the world. For example, fourteen countries now regulate all asbestos types in the same manner, while another fourteen countries or communities differ in their regulation of the asbestos fiber types (IPCS, 1988).

The ACGIH suggests a TLV for chrysotile at 2 fibers/cc(ml); amosite at 0.5 fibers/cc(ml); crocidolite at 0.2 fibers/cc(ml); and all other forms of asbestos at 2 fibers/cc(ml) (ACGIH, 1988). The Commission for the Investigation of Health Hazards of Chemical Compounds in the Work Area of the Deutsche Forschungsgemeinschaft recommends as technical guiding concentrations (TRK) that crocidolite be controlled to 0.5 fibers/ml and that chrysotile, amosite, anthophyllite, tremolite, and actinolite be controlled to 1 fiber/ml (DFG, 1989). Both groups designate all types of asbestos as carcinogenic and recognize that the TLV's and the TRK's are only guidance limits expected to reduce, but not eliminate, the carcinogenic hazard. Unfortunately, neither of these two guidance groups go beyond a set of numbers for controlling asbestos. Neither discuss prevention or control strategies.

TABLE 2. The History of the Occupational Regulation of Asbestos in the United States

Date	U.S. Government	Private	Regulation	Comments
1938	USPHS		5 mppcf	Proposed for guidance; no legal effect.
1946		ACGIH	5 mppcf	Guidance only; no legal effect.
1960	DOL		5 mppcf	Standard for employers under Longshoremen's Act.
1960	DOL		5 mppcm *	Standard under Walsh-Healey Act.
1964	DOL		5 mppcf	Standard for employers under Longshoremen's Act.
1967	DOL		5 mppcf	Adopted ACGIH for Standard for Longshoremen's Act.
1968		ACGIH	12 f/ml or 2 mppcf †	Guidance only; No legal effect.
1969	DOL		12 f/ml or 2 mppcf	Standard for employers under Walsh-Healey Act.
April 1971		ACGIH	5 f/ml	Guidance only; No legal effect.
May 1971	DOL/OSHA		12 f/ml or 2 mppcf	Legal Standard.
Nov. 1971	HEW/NIOSH		5 f/cm	Recommendation; No legal effect.
Dec. 1971	DOL/OSHA		5 f/ml	Emergency Legal Standard.
Jan. 1972	DOL/OSHA		5 f/cc	Legal Standard.
Feb. 1972	HEW/NIOSH		2 f/cc	Recommendation; No legal effect.
June 1972	DOL/OSHA		5 f/cc	Legal Standard.
July 1976	DOL/OSHA		2 f/cc	Legal Standard.
Dec. 1976	HEW/NIOSH		0.1 f/cc	Recommendation; No legal effect.

TABLE 2. The History of the Occupational Regulation of Asbestos in the United States (Cont'd)

Date	U.S. Government	Private	Regulation	Comments
April 1980	NIOSH/OSHA		0.1 f/cc	Recommendation; No Legal effect.
Nov. 1983	DOL/OSHA		0.5 f/cc	Emergency Temporary Std. Fifth Circuit-vacates ETS.
June 1986	DOL/OSHA		0.2 f/cc	Legal Standard.
April 1987	DOL/OSHA		2.0 f/cc	Administrative stay on tremolite, actinolite, and anthophyllite.
May 1990	HHS/NIOSH		0.1 f/cc	Testimony for tremolite, actinolite, and anthophyllite.
July 1990	DOL/OSHA		0.1 f/cc	Proposed rulemaking.
Jan. 1991	HHS/NIOSH		0.1 f/cc	Testimony for new OSHA Std.

* mppcm was apparently a typographical error in the final rule and most likely should have been mppcf.

† The British were the first to respond to growing evidence that fibers and not particles were responsible for asbestos-related diseases, particularly asbestosis (Lane, 1968).

Source: Adopted from Weiss, 1983; HEW/NIOSH, 1971, 1972, 1976; NIOSH/OSHA, 1980; HHS/NIOSH, 1990, 1991; U.S. DOL/OSHA, 1983, 1986, 1987, 1990; U.S. Circuit Court, 1984.

Based on very limited evidence which is principally related to epidemiology studies, fourteen countries and communities differ in their approach to controlling worker exposures to asbestos. The amphiboles are controlled more stringently than chrysotile. Considering the extensive evidence described previously for chrysotile's fibrogenicity and carcinogenicity to animals, such an approach may be premature. Existing evidence from human data which pertains to pure chrysotile exposure does not lead to the conclusion that it is less hazardous than the amphiboles. There is additional concern that chrysotile is rarely found in its pure form, and that most chrysotile deposits will be contaminated with the amphibole, tremolite. Most experts agree that tremolite is a toxic form of asbestos (Sebastien et al., 1989). From a public health perspective, the segregation of various fiber types for regulation is a practice that is not well founded and presents major analytical and regulatory problems.

The findings in the animal and the epidemiologic literature, as well as the conclusions of reports by the Ontario Royal Commission (Dupré et al., 1984) and by the WHO (1989), support an argument against less stringent regulations on exposure levels for chrysotile asbestos than for the amphibole forms of asbestos.

In the process of controlling industrial exposures, there is a hierarchy of controls of which the concentration limit or number is but one factor. A number does provide guidance to the design engineer for developing the necessary engineering controls that will reduce the dust and thus the fibers from asbestos. However, unless engineering controls are effective, continually maintained, and used in combination with good work practices, no standard for occupational exposure control can be adequate. Further, when the standard is inadequate, personal protective equipment must be maintained. Training for both management and workers is essential! Control is not complete unless there are methods to assess its effectiveness. Such assessments rely heavily on surveillance, both medical and environmental, and therefore are an important part of any occupational standard. The U.S. occupational standard for asbestos describes this hierarchy and can serve as a model for a complete occupational health standard (OSHA, 1986).

EPIDEMIC SCENARIO

1. As knowledge surfaced in the medical literature, initially in the British literature and then in the U.S. literature, there was little movement on the part of key policy players toward acting upon the dangers posed by asbestos exposure. Indeed, some policy makers even denied these dangers, though many were fully aware of them, almost from the very beginning. Nonetheless, they withheld the information for as long as possible, sometimes deliberately concealing the facts. If the information had been made public, the fate of many asbestos-exposed victims could have been significantly different. (Castleman, 1984; Lilienfeld, 1991). These people are the "criminals" of this chronology. They included not just company officials, but also scientists, government officials, and other economically-interested parties. Why they did nothing or chose non-preventive actions is beyond the scope of this chapter. These individuals may have been, for the benefit of doubt, included in the group described by Lilienfeld (1983): "In the history of science, there are always a few individuals who are never

convinced when a new concept appears." In any case, if these people had revealed the information available to them, methods to prevent asbestos-related diseases could have been identified much earlier.

Although asbestos has been in use for thousands of years, its commercial value was not realized until the turn of the present century. Shortly thereafter, scattered case reports of asbestos-related diseases began to appear, with the first epidemiological evidence reported by the mid 1920s. Perhaps the landmark epidemiologic study concerning the relationship of asbestos to cancer occurred in 1955 with the publication of the Richard Doll mortality study of British asbestos workers (Doll, 1955). Similar findings of lung cancer then began to appear in U.S. medical literature (Mancuso and Coulter, 1963; Selikoff et al., 1964). This literature, while reporting a valid association between asbestos exposure and disease, did not attract much attention beyond a few other researchers who read the medical literature. Selikoff and his colleagues, however, did something different; they studied the records of the major asbestos union that represented workers who were exposed to asbestos in North America. Previous studies had depended upon company-obtained records. The first health based guidelines in the United States appeared in 1938 and again in 1946 (Dreessen et al., 1938; ACGIH, 1946). Regulations, however, did not take effect in the United States until 1960 (U.S. DOL, 1960).

Broader public awareness of the health hazards associated with asbestos began to surface with the passage of the Occupational Safety and Health Act of 1970 (OSHA, 1970). Asbestos was the subject of the first criteria document for a recommended standard written by the newly created National Institute for Occupational Safety and Health (NIOSH) and was then the subject of early regulation by the newly created Occupational Safety and Health Administration (OSHA). While these organizations drew greater attention to worker problems associated with exposure to asbestos, no widespread public concern occurred until people became aware of the potential exposures of children in schools and of occupants of buildings containing asbestos insulation material. Now the problem transcended the factory wall and the issue of exposure to asbestos became a concern to everyone. Public action was demanded, stricter workplace rules for asbestos were sought, and demands for removing asbestos became common. From the time that asbestos was first used commercially, almost seventy-five years had passed before the industrialized world became fully aware of its hazards and attempted to provide remedies. By that time, hundreds of thousands of deaths had occurred, and countless others had suffered from exposure-related diseases (Nicholson, 1983).

Currently, asbestos is being promoted as an inexpensive and useful material for extensive usage in the developing world. However, to ignore seventy-five years of developing scientific knowledge and gaining social awareness of the problems associated with exposure to asbestos would be a dreadful mistake for a new market of users. The long period before suitable controls were proposed for asbestos was the result of a multifaceted phenomena. The first epidemiology studies occurred with the 1920s discovery of non-malignant asbestos lung disease, but it was not until the case reports of the 1930s and 1940s, along with the more directed epidemiology studies of the 1950s and 1960s, that a relationship between asbestos

exposure and cancer was recognized by the general medical community. In the industrialized world, exposure controls for asbestos developed after these studies were conducted and they are still evolving. In many cases, however, they are still inadequate.

2. The basic mechanisms for policy change that would have supported preventive actions were available, but were not utilized in a timely fashion.

3. The infrastructure around basic mechanisms for policy change was not developed to a degree that would have prevented the first epidemic of asbestos-related diseases.

In the case of asbestos-related disease prevention, the beginning of an infrastructure was available. That beginning was the form of government. The governments of most major industrialized countries were democracies and, as such, had legislative and executive authorities, given by the people, to enact appropriate measures for the protection of public health. The form of government is important since it is in the government where effective change can be affected and endorsed. Democracies are probably the most effective in responding to grassroots appeals of the people, because it is through local initiatives that people control their government. This in turn allows and forces social action. One example of this is the passage of the Occupational Safety and Health Act of 1970 in the United States. However, meaningful regulations were not established until after the fateful chain of events was well into motion. Although some non-binding guidelines had been in existence since the 1930s (Dreessen et al., 1938; Selikoff and Lee, 1978), it was not until 1960 that regulations were established in the United States (Weiss, 1983). In other countries of the industrialized world, regulations continued to be developed over the next two decades (Selikoff and Lee, 1978). Further, as early as 1918, asbestos workers were denied insurance coverage by certain parties who found an economic advantage in doing so (Hoffman, 1918).

The history of governmental intervention to provide worker protection in the United States has been fragmented and was not at all inclusive until the passage of landmark laws, including the Metal and Metallic Mine Act of 1966, the Federal Coal Mine Safety and Health Act of 1969, and the Occupational Safety and Health Act of 1970 (Lemen et al., 1989). What brought these broad legislative initiatives about is probably best explained as being the result of political action on receptive executive and legislative branches of a democratic government. In the United States, unions formed the grassroots structure that led to public admonishment and created a sense of urgency and awareness of a growing health crisis. This culminated in the passage of the Occupational Safety and Health Act of 1970. The law gave the mandate "To assure safe and healthful working conditions for working men and women; . . ." (OSHA, 1970). Such a law, however, is only effective if the government supports it by enacting regulations that are effective and then enforced. Frequently, the simple promulgation of standards will effectuate change and compliance; however, the ability and the willingness of government is a necessary adjunct for an effective occupational safety and health program. Asbestos played a key role in the United States, giving impetus for these governmental initiatives. Dr. Irving Selikoff stated to the U.S. Congress, in support of the Occupational

Safety and Health Act of 1970, that "It is depressing to report, in 1970 that the disease [asbestosis] that we knew well 40 years ago is still with us just as if nothing was ever known" (U.S. Senate, 1970).

In the United States, occupational health first became a part of the agenda of the federal government with the 1914 creation of the U.S. Public Health Service Office of Industrial Hygiene and Sanitation. The early development of a federal role in occupational safety and health was most likely the result of grassroots interest, forcing congressional action in response to the health problems of miners who were principally suffering from silicosis. During World War II, occupational health activities received temporary impetus and financial support, as they had during World War I. Although this impetus was placed by the Secretary of War with the Public Health Service, it was principally dedicated to providing emergency medical services that were aimed at winning the wars. While the depression of the 1930s caused a virtual cessation of field investigation, it did allow time to analyze and publish some major research findings on the determination and control of industrial dust, a reference still considered to be a landmark in the control of industrially-induced dust diseases (Bloomfield and Dallavalle, 1935).

During the early part of this century, many of the asbestos-manufacturing companies had medical departments (Castleman, 1984; Kotelchuck, 1989; Lilienfeld, 1991). Most of the medical departments practiced traditional medical care and were not involved in the practice of preventive occupational medicine, a specialty which has vastly expanded since the late 1960s as a result of the passage of public laws regulating industry in occupational safety and health areas. While hospitals and health care facilities did exist during the early history of asbestos-related diseases, the lack of specialists in occupational safety and health limited the extent of recognition of an occupational problem. Although Ramazzini suggested, as early as the beginning of the eighteenth century (Ramazzini, 1713), that the physician ask his patients the nature of their work, it was, and still is, a practice not fully implemented by physicians. By the mid-1900s, medical programs in industry were fragmented and often consisted of medical staffs who were not trained to recognize occupational hazards or diseases. Many of the health programs provided by industry were contracted to local general medicine clinics and consisted of pre-employment examinations, emergency medical responses, or routine sick calls. In any event, because the physician was on the company payroll, the perception by the worker was usually one of distrust (Davidson, 1970). Training of physicians and other health and safety professionals in the areas of occupational safety and health was very limited before passage of legislation. While the specialty had many recruits, the training of medical students and nurses to recognize occupational/environmental hazards was slow to be implemented.

All of the aspects of exposure to asbestos were present: mining, milling, and the manufacturing of asbestos containing products. Similar events were occurring in other industrialized world countries, including Sweden, England, and Germany.

4. There were not adequate mechanisms for surveillance of the workforce to determine if diseases such as asbestosis, lung cancer, mesothelioma, or other asbestos-associated diseases were occurring. Adequate surveillance for work-related disease did not exist before the passage of legislation protecting worker safety and health in the United States. Mechanisms for surveillance have improved to a limited extent. Much of the research conducted on asbestos was at the sole expense of the industry, in their own facilities. Additionally, research records were controlled by the asbestos industry. Findings of adverse health effects were not published. As A.S. Rossiter, editor of Asbestos Magazine, clearly stated in his letter of 1935 to Summer Simpson, president of Raybestos-Manhattan, "Always you have requested that for certain obvious reasons we publish nothing, and naturally your wishes have been observed." Simpson, then in a subsequent letter to Vandiver Brown, an attorney for Johns-Manville and brother of the Manville president, Lewis H. Brown, said, "I think the less said about asbestos [disease], the better off we are." Brown, in his reply, said, "I quite agree with you that our interests are best served by having asbestosis receive a minimum of publicity" (Kotelchuck, 1989). It was not until the 1960s, when Dr. Irving Selikoff and his associates used union supplied records, that public disclosure became freely available without the control and review of the affected industry. The OSHA Act of 1970 further opened to public accountability unhealthy and unsafe conditions in industry, when it gave government the "right-of-entry" and access to industry's records (OSHA, 1970).

CONCLUSION

Four factors were key to the development of an epidemic of asbestos-related disease in the industrialized world. First, the lethargic action by key policy makers in the medical community, who had the ability to set into motion events that would have weakened or possibly broken a link in the chain of causality. Second, the use of existing mechanisms to support policy change occurred too late to affect the course of the ensuing epidemic. Third, the limited support infrastructures were so fragile that their use was of minimal value to curb the epidemic. Fourth, even if the first three key factors been maximally operational, the lack of an adequately implemented surveillance system did not allow the detection of an epidemic. Contrast this series of events with the actions taken after the detection in 1974 of angiosarcoma of the liver from exposure to vinyl chloride. Each of these four elements were operational and, when effectively utilized, curbed the potential epidemic (OSHA, 1974).

Technology transfer to the developing world is in progress and will most likely continue until an equilibrium is reached between the technology of the industrialized world and the developing world. Asbestos is an example of one substance that is, and will likely continue to be, transferred. All technologies, including the example used in this document, should be analyzed to determine the known hazards and to what extent the agent can and should be developed or handled. There are many differences between the developing world and the industrialized world, including climate, culture, demographic, anthropometric, population diversity, labor force, etc., each of which can contribute to problems for technology transfer.

Such problems can result in adverse health effects, not only to workers, but to the population as a whole, and to the overall public health within the country receiving the new technology.

Many of the developing countries are at the very early stages of receiving new technologies, while others are much further along. Some developing countries are still trying to improve their basic public health infrastructures by improving sanitation, reducing infant mortality, improving health care delivery, etc. For these countries, the introduction of new technologies can represent a very critical, and oftentimes overwhelming, adjustment. The difficult lessons learned by the industrialized world can be critical in averting similar experiences in the developing world.

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