9 The pesticide DBCP and male infertility

Eula Bingham and Celeste Monforton

Dibromochloropropane (DBCP) is a pesticide used against nematodes (roundworms or threadworms) that damage pineapples, bananas and other tropical fruits. It was introduced into US agriculture in 1955 and approved for use as a fumigant in 1964. By 1961 laboratory experiments had shown that it made the testicles of rodents shrink and significantly reduced the quantity and quality of sperm. Nonetheless, the compound was widely marketed and became a commercial success.

In 1977, workers at a production plant became worried that they were unable to father children. An emergency study by a US government agency discovered that in many cases the workers were suffering from deficient or absent sperm. While controls were improved at US facilities, the product continued to be marketed and sprayed in Latin America, the Philippines, some African countries, and elsewhere.

By the 1990s, tens of thousands of plantation workers in these countries had allegedly suffered adverse reproductive effects from DBCP use. The story continues today with contentious legal claims for compensation, contamination of drinking water and industry attempts to prevent a Swedish documentary on the issue from being screened.

This chapter looks at the knowledge available about the hazards and the actions taken, or not taken, to avert them. The DBCP story is significant as it is the first clear example of reproductive damage to workers who manufactured and used a synthetic chemical. This is one of many examples supporting the growing concerns about increasing rates of reproductive and developmental disease, and about the endocrine disrupting chemicals that seem to be playing a role in these disorders.

Protecting production workers, users, consumers and the environment from chemicals that may damage reproduction demands closer integration of scientific disciplines, as well as government action. The lessons of DBCP may help in ensuring timely protection from harm, based on precautionary approaches to scientific evidence.

'If anyone wants to use a male birth control drug, I think we have identified one, but it is not very pleasant to use.'

 Dr Charles Hine, Shell Contractor, c. 1960, quoted in Goldsmith (1997).

9.1 The discovery 1977: 'our union members are sterile'

In July 1977, one of the authors of this chapter (Eula Bingham, then US Assistant Secretary of Labor for Occupational Safety and Health) was contacted by Tony Mazzocchi, Vice President of the Oil, Chemical, and Atomic Workers International Union (OCAW) in the United States. He wrote:

> 'In a chemical factory in California during a lunch hour, several workers confided to each other that they were worried about not having children. One worker had a child, but had been trying for another for almost two years and two other young workers had no children and were concerned that there was something wrong. Their wives had been examined and now they thought it might be that they themselves had a problem. When the concerns were passed to other workers, the union arranged for the seven of them to have sperm counts performed. The sperm counts were either zero, or so low, that they showed the men sterile and the union was contacting the US National Institute of Occupational Safety and Health (NIOSH) to perform a Health Hazard Evaluation'.

The chemical responsible for causing sterility in the workers at the California chemical plant was 1,2-Dibromo-3-Chloropropane (DBCP). The compound was first produced in the United States in 1955 and used as a soil fumigant to control nematode worms in the soil. DBCP products carried trade names such as Fumazone, Nemagon, Nemaset, and Nematox (US EPA, 1979; OSHA, 1977a and 1977b; Misko et al., 1993; Clark and Snedeker, 2005; NIOSH, 1977) and were primarily used to protect crops, such as pineapple, bananas, sugar cane and other produce, mostly in the tropics. As a soil fumigant, DBCP was applied at a rate of 10–125 kg/ha, either injected directly into the soil or added to irrigation water.

The three main US manufacturers were Dow Chemical Company, Shell Chemical Company and Occidental Chemical Company, but DBCP was also produced in Europe (by International Chemical Company in the United Kingdom) and in Japan. At its peak an estimated 14.7 million kg of DBCP were used annually prior to its suspension in 1977. Most production was used in the US, Latin America, the Philippines and some African countries.

9.2 Early warnings: 1961–1975

The earliest research on DBCP toxicity was carried out by two chemical companies producing the compound for use as a nematocide. By 1958, both Shell and Dow had obtained toxicological data from experiments on rats showing that DBCP was absorbed through the skin and by inhalation and affected the liver, lung, kidney and testes.

Charles Hine, working then under contract for Shell, reported a variety of adverse effects in laboratory animals, depending on the dose of DBCP vapour administered. At an exposure to 5 ppm (5 parts of the chemical in one million parts of air), the testes in male rats shrank, at 10 ppm most of the male rats had testes half the normal size and at 20 ppm all the male rats were sterile. An internal memorandum prepared by Shell noted: 'We understand that Dow Chemical Company have similar data and are very upset by the effects noted on the testes' (Lykken, 1958).

At that time, scientists working for these companies were clearly worried about the results. John Goldsmith, epidemiologist, later wrote: 'I recall a conversation with the late Dr Charles Hine from the University of California at San Francisco about 1960 at a party, when he said, "If anyone wants to use a male birth control drug, I think we have identified one, but it is not very pleasant to use"' (Goldsmith, 1997).

In 1961, the industry toxicologists published their data from experimental studies (NIOSH, 1977), supporting the initial observations (see Box 9.1). These studies revealed that DBCP had two outstanding toxic effects: an antispermatogenic effect in males and damage to kidneys in both sexes of the rat.

9.3 Pesticide registration and inadequate 'hazard control' 1961–1977

The years 1958–1961 were a critical period for decisions on hazard protection and for the use and marketing of DBCP in the United States and globally. Charles Hine, working as an expert

consultant for Dow and Shell, supported a request for the US Food and Drug Administration (USFDA) to register DBCP as an approved pesticide. His report called for workplace concentrations to be less than 1 ppm and impermeable protective clothing to be used if skin contact was likely.

In a series of discussions between the USFDA, Shell and Dow, the regulator noted that at the lowest exposure level studied, 5 ppm, there were adverse effects after repeated exposures and that the current safety precautions therefore appeared inadequate. However, the Shell representative considered the Hine recommendations to be impractical.

By 1961, Torkelson, Hine and colleagues (Torkelson et al., 1961) recommended that occupational exposure to DBCP should be limited by keeping the airborne concentration below 1 ppm, and stressed that suitable analytical methods rather than sensory perception should be depended upon for control. These authors had interviewed men who had been briefly exposed to 1.7 ppm of DBCP and they described a definite, not unpleasant odour (NIOSH, 1978).

In 1961 the US Food and Drug Administration approved and registered DBCP as a pesticide and recommended the exposure limit of 1 ppm. Thereafter the US Department of Agriculture (USDA) was asked to approve the product labelling, which simply stated: 'Do not breathe vapours, use only in a well-ventilated area and avoid prolonged breathing'. As such, the warning label included no reference to testicular damage.

The USDA initially expressed reservations regarding the warnings on the label but Shell argued that at 5 ppm no adverse effects had been reported and that the odour threshold of 1.7 ppm therefore provided an adequate 'warning' of excessive exposure (Thrupp, 1991). USDA accepted these reassurances even though no studies had been performed to indicate that this approach was safe. Virtually no attempts were made to determine if the measures adopted were indeed safe for manufacturing workers or pesticide sprayers. Neither group was subjected to medical surveillance.

In fact, the 'odour threshold' for DBCP exposure was too high to ensure reliable protection against the toxicity reported in the animal studies. DBCP fumes are only a mild irritant and unlikely to be reported as potentially harmful. Workers could therefore be exposed to dangerous amounts of the chemical without being aware of it.

The labelling and workplace exposure precautions were therefore inadequate from the 1950s until 1977, failing to provide accurate information about the potential health effects of DBCP or to ensure safe working conditions, in the light of the animal evidence. The toxicological data available in 1961 on the potential adverse health effects of DBCP was sufficient to have required specific health warnings, personal protective equipment and medical surveillance. None was provided.

Box 9.1 Animal toxicity data for DBCP: 1958–1975

Torkelson et al. (1961) evaluated the effects of exposing rats to DBCP by inhalation for seven hours a day, five days a week for 10 weeks. The lowest concentration, 5 ppm (parts per million), produced an 18.6 % decrease in the mean weight of the testes, which was not statistically significant. Exposure to 10 ppm resulted in a statistically significant decrease (49 %) in the mean weight of the testes and a significant increase in the weight of the kidney (31.7 %).

In another study reported by Torkelson et al. (1961) male and female rats were exposed to 12 ppm DBCP for seven hours a day, five days a week for 10 weeks. Degenerative changes occurred in the tissue where sperm are formed, reducing the number of sperm cells and increasing the proportion of abnormal sperm cells. Significant increases in the weights of the kidneys occurred in both sexes and there were changes in the kidneys of the males. Changes in the livers of both sexes were also noted.

Exposing guinea pigs and rabbits to 12 ppm DBCP vapour inhalation resulted in statistically significant decreases in the mean weights of the testes in both species (Torkelson et al., 1961).

Toxicological studies by European laboratories also reported around 50 % reductions in the weight of testes and in sperm counts and motility (Rakhmatulayve, 1971; Reznik and Sprinchan, 1975). In female rats, the reproductive cycle was disrupted.

9.4 Actions to reduce exposure in DBCP manufacturing: 1977 and 1978

The National Institute for Safety and Health (NIOSH) was created in 1970. Responding to the suspicions of the DBCP manufacturing workers and a request by their trade union, OCAW, for government help, NIOSH (1977) conducted a health hazard evaluation at the Occidental Chemical Company's Lathrop plant. It reported airborne DBCP concentrations of 0.29–0.43 ppm, measured as an eight-hour time-weighted average. Of 13 workers in the production area, nine had no sperm (azoospermia) and another four workers had very reduced sperm counts (oligospermia). The researchers conducting the evaluation for NIOSH found a 'clear increase in the prevalence of oligospermia with increasing exposure' to DBCP (Whorton et al., 1977). These exposure levels were far below those used in the toxicological studies and also below the recommended 'safe' levels for workers of 1 ppm.

With this alarming information, the President of OCAW formally petitioned the US Occupational Safety and Health Administration (OSHA) on 23 August 1977 to take action to limit worker DBCP exposure to 1 part per billion (1 ppb) parts of air and to conduct medical testing to identify cases of sterility and cancer among exposed workers. This call for action was met with a flurry of activity at the federal government level. OSHA issued an Emergency Temporary Standard (ETS) on 9 September 1977 and proposed a permanent standard in November. Public hearings were held in December 1977 and a final standard was published in the Federal Register on 11 March 1978.

The results of the medical examinations of the OCAW workers provided compelling evidence for OSHA action, but the Administration also evaluated all other available information on DBCP as part of the rulemaking process. This included, for example, data from the Dow Chemical facility in Magnolia, Arkansas, where DBCP was manufactured. Air sampling results revealed concentrations of 0.04 ppm to 0.4 ppm of DBCP calculated as an 8-hour time-weighted average. Furthermore, medical tests revealed that 50 % of the 106 workers examined there had either oligospermia or azoospermia.

These data suggested that exposures below 1 ppm were associated with adverse reproductive effects. However, because DBCP is also absorbed through the skin, dermal exposures may have contributed an unknown but potentially significant amount to the workers' total dose of DBCP.

Based on the evidence of the serious adverse reproductive health effects in animals and humans, and its carcinogenicity in animals (see below), OSHA issued a final standard to limit workers' DBCP exposure to 1 ppb (based on an 8-hour time-weighted average), and to 10 ppb over any 15-minute period. OSHA also required employers to provide initial and annual medical examination for DBCP-exposed workers, respiratory protection and training, among other provisions.

The new rules took effect in April 1978 and were not effectively challenged by any interested party. The US National Peach Council did, however, attempt to delay the regulation with a direct plea to OSHA, expressed in a letter to Eula Bingham, co-author of this chapter. The Council argued that:

> While involuntary sterility caused by a manufactured chemical may be bad, it is not necessarily so. After all, there are many people who are now paying to have themselves sterilized to assure they will no longer be able to become parents. How many of the workers who have become sterile were of an age that they would have been likely to have children anyway? How many were past the age when they would want to have children? These, too, are important questions.

> 'If possible sterility is the main problem, couldn't workers who were old enough that they no longer wanted to have children accept such positions voluntarily? They could know the situation and it wouldn't matter. Or could workers be advised of the situation and some might volunteer for such work posts as an alternative to planned surgery for a vasectomy or tubal ligation, or as a means of getting around religious bans on birth control when they want no more children. We do believe in safety in the work place, Dr Bingham, but there can be good as well as bad sides to a situation' (US National Peach Council, 1977).

This argument found little favour with OSHA.

Meanwhile studies on exposed production workers were conducted in Israel. In a series of publications researchers discovered DBCP-induced sterility in the six workers at a DBCP-production facility that had been exposed for two to ten years (Potashnik et al., 1978). The workers also had an elevated serum concentration for one sex hormone (follicular stimulating hormone, FSH, which increases with testicular damage) and damage to the testicular tissue responsible for producing sperm cells.

In a related study, 18 of 23 workers (78 %) involved in DBCP production had abnormal sperm counts, including 12 workers with azoospermia. After several years without exposure to DBCP, some of the workers' testicular function improved but the men exposed for more than 120 hours experienced no improvement (Potashnik, 1984). Similarly, 17 years after being exposed to DBCP the extent of recovery from sperm damage was mixed (Potashnik and Porath, 1995).

9.5 DBCP and cancer?

In 1975, as part of a programme to test pesticides for carcinogenicity, the National Cancer Institute reported that DBCP was carcinogenic in rats and mice. Industry representatives criticised the study at an OSHA hearing in 1977 because of the high doses used. Subsequently, a rodent study of both sexes using much lower doses by Dow Chemical at the Hazleton Laboratory in 1977 resulted in carcinomas of the stomach, liver and renal tubules at the highest dose and a statistically non-significant increase in carcinomas at the two lower doses.

Today, animal experiments using high exposure levels are still employed to evaluate the safety of many chemicals. Industry and other interested parties often assert that such experiments are irrelevant to human exposures, which are usually much lower. There are, however, good reasons to doubt these claims. The small number of animals used in experiments (e.g. usually 20 per exposure group) mean that the doses have to be high in order to reveal any possible hazard that thousands of workers (or many more consumers) face at much lower exposure levels. As a result, high doses have been shown, in very many cases, to be reliable predictors of the hazards humans face at much lower doses.

Since 1992, the US Environmental Protection Agency (EPA) has classified DBCP as a 'probable human carcinogen' (¹). The International Agency for Research on Cancer (IARC) assessed the evidence as sufficient in experimental animals to classify DBCP as a 2B ('possible') carcinogen (IARC, 1999).

9.6 DBCP risks: from manufacturing to pesticide spraying

While OSHA was attempting to protect workers manufacturing DBCP, the US EPA took steps to protect the health of workers using the pesticide. In September 1977, US EPA administrator Douglas M. Costle announced that under the authority granted by the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) he was suspending distribution of DBCP. Costle noted that he had this special authority 'in situations where the use of that pesticide appears likely to pose an unreasonable risk to man during the period necessary to conduct and complete a more lengthy administrative proceeding.'

The US EPA examined the scientific evidence and, even though early studies of DBCP sprayers in California and Israel had produced mixed results (Glass et al., 1979; Karraazi et al., 1980), determined that the risk of harm was sufficient to take emergency temporary action to protect workers spraying the pesticide. The US EPA prohibition on using DBCP became permanent in November 1979 and applied to all crops, except for pineapples grown in Hawaii. In 1985 the US also prohibited use of DBCP on pineapples.

9.7 DBCP exports from 1969 to the 1980s: spreading sterility?

DBCP was developed for use against nematodes that attack pineapple plants, so it was not surprising that it was also effective on another tropical fruit: bananas. In the mid-1960s, the Standard Fruit Company began testing DBCP on its banana plantations in Central America; by 1969, DBCP was in full-scale use in Costa Rica and Honduras. The pesticide containers were boldly marked with the brand names Fumazon and Nemagon but, like containers in the United States, provided no warnings to workers about the risk of sterility. Moreover, the labels on the pesticides exported were in English. Even if they had been written in Spanish, there is no guarantee that pesticide sprayers could have read them, since many were illiterate.

The US EPA regulatory ban on using DBCP pesticide in the US did not ban Shell and Dow from manufacturing it and the Standard Fruit Company

⁽¹⁾ http://www.epa.gov/ttn/atw/hlthef/dibromo-.html.

Box 9.2 The Swedish film documentary, the Dole lawsuit and freedom of speech

Maria Albin

'Bananas!', a 2009 documentary by independent Swedish filmmaker Fredrik Gertten, addresses the attempts of 12 banana plantation workers in Nicaragua to sue Dole Food Company (previously named Standard Fruit) for DBCP-induced sterility. The film closely follows the workers and the controversial personal injury Californian lawyer, Juan Dominguez, who took on their claims.

In July 2009 Dole sued Gertten and the film's producer Margarete Jangard, claiming defamation and seeking a permanent injunction against them screening 'Bananas' in public, displaying the film website or giving interviews promoting the film 'in which any portion of the accusations made against Dole in the documentary film Bananas are republished'.

The lawsuit followed other steps by Dole to obstruct the film's release. Dole sought to have the film withdrawn from the Los Angeles film festival where it was due to be shown in June 2009 (it was moved to a 'special case study screening' to avoid possible legal action). Dole also sent a letter to the Swedish ambassador Jonas Hafström in Washington, asking him to take 'appropriate steps to limit its damaging impact, including urging the filmmakers, WG Film AB and Mr Gertten to act responsibly and halt dissemination of this film in the United States of America and Europe.

The media's response to Dole's efforts was robust. Filmmakers launched a petition for free speech during the Los Angeles film festival. The CEO of the German Documentary Film Association wrote a letter demanding that Dole cease its 'attacks on the freedom of information as well as stop your company's inhuman practices in Latin America which the film "Bananas" criticizes.' The International Federation of Journalists likewise condemned the use of the law to evade media scrutiny and public accountability as an unforgiveable violation of free speech.

Gertten and Jangard regarded Dole's lawsuit as a strategic lawsuit against public participation (SLAPP) - a deliberate attempt by a wealthy party to silence its critics by outspending them in launching a legal action. Accordingly, the filmmakers filed an anti-SLAPP motion and a cross-complaint.

Swedish reaction was strong and media coverage was extensive. The film was shown in Sweden's parliament, causing an exchange of letters between the executive vice-president of Dole and the two MPs responsible for the screening, Mats Johansson of the Conservative Party and Luciano Astudillo of the Social Democrats. Their unusually frank letter reflects Swedish public opinion at that stage:

'It seems clear to us that you are misled by your PR-firm on how to influence Swedish opinion, with a poor understanding of our tradition of free speech during more than two hundred years. As the saying goes: all business is local. We strongly recommend a change of bureau and tactics, if you are at all interested in the Swedish market. But first and most we urge you — in the name of free speech — to withdraw your lawsuit against Mr Gertten.'

MPs signed a cross-party petition urging Dole to withdraw their legal action in the name of free speech, and they were joined in these demands by the CEOs of leading food chains. The action was sufficient to make Dole withdraw its legal action in October 2009. It stated that it made its decision in view of the free speech concerns being expressed in Sweden, although it continued to believe in the merits of its case. The filmmakers withdrew their counter-claim but demanded that their legal fees be reimbursed. However, the threat that Dole would reinstitute the action hampered the distribution of the film.

In 2010, a Los Angeles court decided in favour of the filmmakers, stating that the lawsuit had been what is commonly known as a SLAPP, awarding them almost USD 200 000 in fees and costs and enabling Bananas! to be released in the US.

Having made a film about Dole being sued, Gertten has now made a new film about being sued by Dole. 'Big boys gone bananas!' premiered in October 2011 at the International Documentary Film Festival in Amsterdam.

Sources: http://www.bananasthemovie.com/wp-content/uploads/resources/slapp_ruling_nov17_10.pdf; http://www.bananasthemovie.com/wp-content/uploads/resources/bananas_defamation_complaint_july8_09.pdf; http://www.iidh.ed.cr/comunidades/ombudsnet/docs/doctrina/doc_ccpdh_resoluciones/resolucion%20nicaragua%20final.htm; http://www.bananasthemovie.com/wp-content/uploads/resources/letter_dole-hafstrom_june5_09.pdf; http://www.ifj.org/en/articles/ifj-condemns-attempts-by-embattled-food-company-to-censor-free-speech-in-us. and other growers continued to use it. When Dow informed Standard Fruit that it was halting shipments of DBCP, Standard Fruit threatened Dow with a claim of breach of contract. To settle the matter, Standard Fruit Company agreed to indemnify Dow for any injuries resulting from exposure to DBCP and implement 'applicable work standards in respect of protective clothing, training etc.' as outlined in the OSHA standard.

Shipping records and billing invoices made available through litigation on behalf of DBCP-injured workers reveal that Shell Chemical also sold the pesticide to growers in the Ivory Coast from 1977 to 1980. Another US manufacturer, Amvac Chemical Corporation, sold DBCP in 1979 to companies in the Philippines, Honduras and Nicaragua. DBCP was still used in Central American banana plantations until at least 1985 (New York Times, 2003).

In the Philippines, workers employed by subcontractors of Standard Fruit used DBCP until about 1986. According to reports collected by lawyers, some of these workers became sterile and reported that they had not been informed about the risk of using the chemical and had not been given appropriate personal protective equipment. In Costa Rica too, there was inadequate protection of DPCP sprayers (Thrupp, 1991).

Similarly, medical evaluations of 28 Panamanian banana workers in August 1993 diagnosed 25 with damaged sperm (Navaro, 1993).

9.8 Banana workers bring compensation cases: 1990-2010

In the early 1990s, more than 16 000 banana plantation workers from Central America and the Philippines filed a class action lawsuit in Texas against US fruit and chemical companies, demanding compensation for permanent sterility linked to DBCP. A 1992 settlement in Costa Rica provided USD 20 million for 1 000 workers. In another lawsuit involving 26 000 workers employed in Latin America and elsewhere, the total settlement in 1997 of USD 41 million provided an average compensation of USD 1 500 to each



Photo: © istockphoto/Francisco Orellana

worker. In 2002, a national tribunal in Nicaragua sentenced the American multinational companies to pay USD 489 million in damages and interest to 450 workers affected by Nemagon. In a lawsuit, filed on behalf of 13 Nicaraguan banana plantation workers, Amvac Chemical agreed in April 2007 to a total compensation of USD 300 000 to the now-sterile workers.

These settlements came 20 years after each of these firms knew about the potential reproductive health risks to DBCP pesticide spray workers, which had stopped their use in the US. Nevertheless, the firms marketed and sold DBCP abroad without ensuring that worker health would be adequately protected. Tens of thousands of banana workers still have suits pending in courts in the US and elsewhere but many of the relevant facts are still unclear and contested by the growers.

9.9 Environmental pollution of soils and water by DBCP

DBCP is a persistent and mobile chemical and has been found in the soil, and in ground and surface water in areas where it has been used. Torkelson et al. (1961) noted that 'its relatively low vapour pressure and high density assures a long residence in the soil', depending on the method of application.

Although DBCP has been banned for use in agriculture for more than 20 years it persists in the environment and in water supplies. Underground aquifers in the Sacramento Valley of California are contaminated with DBCP and, depending on the temperature and pH, the chemical can persist for over a century (Peoples et al., 1980; Burlington et al., 1982; California Environmental Protection Agency, 1999). It was the most frequently detected contaminant in California wells in the early 1990s (Bartkowiak et al., 1995).

The 2010 update of the California Well Inventory Database reported DBCP detections in 254 of 1 312 wells sampled. Concentrations of DBCP found ranged from 0.01–1.7 ppb compared to the US EPA and Californian maximum contaminant level of 0.2 ppb. Between 1986 and 2009 DBCP concentrations declined in about half of the wells sampled from 49 % above the maximum concentration level (MCL) of 0.2 ppb to 25 % being above the MCL.

The US-based interest group Environmental Working Group (EWG) analysed 20 million tap water quality tests performed by water utilities between 2004 and 2009 (EWG, 2009). Their investigations identified 191 water systems in 18 states with DBCP levels in drinking water above health guidelines set by federal and state health agencies. Of these, 48 water utilities had DBCP levels above the US EPA's legally enforceable maximum contaminant level of 0.2 ppb. The World Health Organization's guideline value for drinking water quality is 1 ppb (WHO, 2003).

More than 20 years after DBCP was banned, levels continue to exceed health limits in the tap water of over 4 million Californians.

EWG also noted that 'in 38 communities, the levels of DBCP in tap water are above the so-called 'negligible' risk for carcinogens. In 31 communities, the levels ranged from 20–200 times the amount associated with a 'negligible' risk. A particular concern raised was in the case of infants drinking formula prepared with the tap water.'

9.10 Some late lessons

Lessons for science

- 1. DBCP exposures below the lowest dose tested in animal studies were mistakenly assumed to be safe.
- 2. While adverse effects on crude testicular morphology and sperm counts were documented, further studies were not carried out to determine the exposure levels that could have provided more subtle indicators of early stage infertility.
- 3. The early toxicity studies were carried out before modern protocols became available, but continued application of DBCP in developing countries did not lead to the use of updated protocols to assess the toxicity in further detail.
- 4. Evidence of harm in animals was not seen by many scientists as relevant to humans.
- 5. Human reproduction may be sensitive to subtle derangements of physiological processes, thereby causing sub-fertility or infertility in the absence of obvious pathology.
- 6. No attention was paid to the possible effects on sons of exposed women.
- 7. Routine medical records and health statistics can be of limited use in regard to adverse effects on reproduction.

- 8. Skin exposure can contribute significant doses of DBCP: air monitoring alone, as with other skin penetrating chemicals, therefore underestimated total doses received.
- 9. Independent expert assessments e.g. by the US governmental body, NIOSH, were needed to identify harm and to better protect employees.

Communication and use of research evidence

- 1. The original evidence for DBCP effects on human male sterility came from the lay and local knowledge of the workers and their wives.
- 2. It was confidentially asserted that DBCP was safe to use without there being any studies of workers or relevant animal studies to confirm this assumption: an example of the '**authoritative assertion but without evidence**' which appears in other chapters.
- 3. The toxicity information was translated only into very general warnings on labels: no translations were provided for products exported.
- 4. The animal toxicological findings did not lead to any surveillance studies of men exposed to DBCP at the production plants until after evidence was observed by the workers.
- 5. No action was taken to avoid the earlier biological 'effects' in animals until they had become 'adverse effects' in people.
- 6. The application of DBCP was considered essential by growers, including multinational companies, and they considered the toxicity concerns were too small to be significant.
- 7. The early scientific warnings were not widely reported but confined to specialist scientific journals or internal company communications.
- 8. Knowledge from the manufacturing risks did not get taken up by the companies responsible for user risks.

- 9. National standards to control the risks of DBCP were not transferred into international standards to protect workers from globalised exposures to hazardous chemicals.
- 10. Early warnings about the persistence of DBCP in soils and water did not get acted upon until many years later.

Compensation for victims

- 1. Much information about the responses of DPCP producers and user companies only emerged via legal procedures in the compensation cases.
- 2. Compensation cases in the law courts can be difficult, expensive and very time consuming to pursue (see Chapter 24 on protecting early warners and late victims).

9.11 Conclusion

There is now widespread concern about male infertility, and related reproductive problems, such as testicular cancer and developmental defects, in wildlife, workers and consumers (e.g. WHO, in press; EEA, 2012; BCPT, 2008).

The lessons of DBCP are very relevant to these concerns and to the current exposures of many workers and consumers to the endocrine disrupting substances (EDSs) which seem to be playing a role in the reproductive ill health of both humans and wildlife (see also Chapter 13 on ethinyl oestradiol in the aquatic environment and Chapter 10 on BPA).

Protecting production workers, users, consumers, and the environment from chemicals that may damage reproduction needs the closer integration of scientific disciplines, and government actions if the timely protection from harm, using precautionary approaches to the evidence from science, is to be achieved. The lessons of DBCP may help in this.

1956	Manufacturing of DBCP began
1958	Two independent rodent studies document testicular toxicity
1961	DBCP is registered as an approved pesticide in the US
1961	Animal studies show effects on testes and sperm
1961	Medical examination of workers at a DBCP production plant takes place but testicular function is not examined
1961	Data on persistence and water solubility are published
1969	Use of DBCP at banana plantation in Costa Rica occurs without appropriate warning labels or safety precautions
1975	DBCP is found to be carcinogenic in two species of rodents, both male and female
1977	Episodes of reduced sperm counts occur in US manufacturing workers
1977	US authorities investigate DBCP toxicity, issue new guidelines, enforce new exposure limits and remove approval, except for special pineapple protection in Hawaii
1977-?	DBCP continues to be exported for use outside the US
1990s	Court cases are brought against employers and producers to compensate for adverse health effects in plantation workers in Latin America and the Philippines
1999	State of California decides to regulate DBCP contamination of drinking water
1990s-2010	Compensation cases for DBCP users in South America are won and lost
2007-today	DBCP remains a contaminant of drinking water in 38 cities in California and elsewhere

Table 9.1 Key early warnings about and recognition of DBCP toxicity

References

Albin, M. and Giwercman, A., 2009, 'Evidence of toxic effect of a chemical on reproduction ignored' *Lakartidningen*, (106/37) 2 296–2 297 (in Swedish).

Bartkowiak, D., Newhart, K., Pepple, M., Troiano, J. and Weaver, D., 1995, Sampling for Pesticide Residues in California Well Water, 1995 update of the Well Inventory Data Base, Report No EH95-06 (http://www.cdpr.ca.gov/docs/empm/pubs/ ehapreps/e9506.htm) accessed 12 April 2011.

BCPT, 2008, 'PPTOX Prenatal Programming and Toxicity', *Basic & Clinical Pharmacology & Toxicology*, (102/2).

Beasley Allen Law Firm, 2007, 'Pesticide company settles sterility lawsuit', 24 April 2007.

Burlington, N.E., Lee, L.A. and Rosenblatt, D.H., 1982, 'Kinetics and products of hydrolosis of 1,2-dibromo-3-chloropropane', *Environ Sci Technol.*, (16) 627–632.

California Environmental Protection Agency, 1999, *Public health goal for 1,2-Dibromo-3-chloropropane* (*DBCP*) *in drinking water* (http://www.oehha.ca.gov/ water/phg/pdf/dbcp_f.pdf) accessed 12 April 2011.

Clark and Snedeker, 2005, 'Critical evaluation of the cancer risk of dibromochloropropane (DBCP)',

J. Environ. Sci. Health C. Environ. Carcinog. Ecotoxicol. Rev., (23/2) 215–260.

EEA, 2012, *The impacts of endocrine disrupters on wildlife, people and their environments,* EEA Technical report No 2/2012, European Environment Agency.

EWG, 2009, 'National Drinking Water Database', Environmental Working Group, December 2009 (http://www.ewg.org/tap-water/home) accessed 5 January 2012.

Glass, R.I., Lyness, R.N., Mengle, D.C., Powel, K.E. and Kahn, E., 1979, 'Sperm count depression in pesticide applicators exposed to dibromochloropropane', *Am. J. Epidemiol.*, (109) 346–351.

Goldsmith, J.R., 1997, 'Dibromochloropropane: Epidemiological Findings and Current Questions', *Annals New York Academy of Sciences*, (26/837) 300–306.

Goldsmith, J.R., Potashnik, G., Israeli, R., 1984, 'Reproductive outcomes in families of DBCP exposed men', *Arch. Environ. Health*, (39) 85–89.

International Agency for Research on Cancer (IARC), 1999, 'Re-evaluation of some organic chemicals, hydrazine and hydrogen peroxide', *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*, (71) 470–500. Kharrazi, M., Potashnik, G. and Goldsmith, J. R., 1980, 'Reproductive effects of dibromochloropropane', *Isr. J. Med. Sci.*, (16) 403–406.

Kloos, H., 1996, '1,2 Dibromo-3-chloropropane (DBCP) and ethylene dibromide (EDB) in well water in the Fresno/Clovis metropolitan area, California', *Arch. Environ. Health*, (51/4) 291–299.

Lykken, L., 1958, 'Letter to M.R. Zavon, Kettering Laboratory, University of Cincinnati, June 4, 1958', in: Misko, F., Siegel, C.S. et al., 1993, *DBCP: The Legacy*, Dallas, Texas.

Miller, T.C., 2007, 'Pesticide company settles sterility suit for \$300,000', *Los Angeles Times*, 16 April 2007.

Misko F., Siegel, C.S. et al., 1993, *DBCP: The Legacy*, Dallas, Texas.

Navaro, C., 'Laboratorio Clinico Navarro, Changuinola, Panama, Agosto de 1993', in: Misko, F., Siegel, C. S. et al., 1993, *DBCP: The Legacy*, Dallas, Texas.

New York Times, 2003, 'Banana Workers Get Day in Court', 18 January 2003.

NIOSH, 1977, Health Hazard Evaluation: Occidental Chemical Company, Lathrop, California, United States National Institute of Occupational Safety and Health, August–October 1977.

NIOSH, 1978, A Recommended Standard for Occupational Exposure to DBCP, US Department of Health, Education and Welfare.

OSHA, 1977a, *Proposed Standard on Occupational Exposure to 1,2-Dibromo-3-Chloropropane*, Occupational Safety and Health Administration, 42 Federal Register 57267, 1 November 1977.

OSHA, 1977b, *Emergency Temporary Standard on Occupational Exposure to 1,2-Dibromo-3-Chloropropane*, Occupational Safety and Health Administration, 42 Federal Register 45536, 9 September 1977.

OSHA, 1978, Final Rule on Occupational Exposure to 1,2-Dibromo-3-Chloropropane, Occupational Safety and Health Administration, 43 Federal Register 11514, 17 March 1978.

Peoples, S.A., Maddy, K.T., Cusick, W., Jackson, T., Cooper, C. and Frederickson, A.S., 1980, 'A study of samples of well water collected from selected areas in California to determine the presence of DBCP and certain other pesticide residues', *Bull. Environ. Contam. Toxicol.*, (24) 611–618.

Potashnik, G., Ben-Aderet, N., Israeli, R., Yanai-Inbar, I. and Sober. I., 1978, 'Suppressive effects of 1,2-dibromo-3-chloropropane on human spermatogenesis', *Fertility and Sterility*, (30) 444–447.

Potashnik, G., Yanai-Inbar, I., Sacks, M.I. and Israeli, R., 1979, 'Effect of dibromochloropropane on human testicular function', *Israel Journal of Medical Sciences*, (15) 438–442.

Potashnik, G., Goldsmith, J.R. and Insler, V., 1984, 'Dibromochloropropane-induced reduction of the sex-ratio in man', *Andrologia*, (16) 213–218.

Potashnik, G., and Porath, A., 1995, 'Dibromochloropropane (DBCP): A 17-year reassessment of testicular function and reproductive performance', *J. Occup. Environ. Med.*, (37) 1 287–1 292.

Rakhmatulayve, 1971, 'Hygienic features of nematocide nemagon in relation to water pollution protection', *Hyg. Sanit.*, (36) 334–348.

Reed, N.R., Olson, H.E., Marty, M., Beltran, L.M., McKone, T., Bogen, K.T., Tablante, N.L. and Hsieh, D.P.H., 1987, *Health risk assessment of 1,2-Dibromo-Chloropropane (DBCP) in California drinking water*, University of California Davis, Department of Environmental Toxicology.

Reznik, I.B. and Sprinchan, G.K., 1975, 'Experimental data on gonadotoxic action of nemagon' (in Russian) *Gig Sanit*, (6) 101–102.

Thrupp, L.A., 1991, 'Sterilization of workers form pesticide exposure: The causes and consequences of DBCP-induced damage in Costa Rica and beyond', *International Journal of Health Services*, (21/4) 731–757.

Torkelson, T.R., Sadek, S.E., Rowe, V.K., Kodama, J.K., Anderson, H.H., Loquvam, G.S. and Hine, C.H., 1961, 'Toxicologic investigations of 1,2-dibromo-3-chloropropane', *Toxicol. Appl. Pharmacol.*, (3) 545–559.

US EPA, 1979, *Dibromochloropropane: Suspension order and notice of intent to cancel*, United States Environmental Protection Agency, Federal Register 65135, 9 November 1979.

US National Peach Council, 1977, Personal communication to Eula Bingham, September 1977.

WHO, 2003, WHO Guidelines for Drinking-Water Quality, undated, Water Sanitation and Health (WSH).

WHO, in press, *Global assessment of the State-ofthe-Science of Endocrine Disruptors*, World Health Organization. Whorton, D., Kraus, R.M., Marshall, S. and Milby, T.H., 1977, 'Infertility in male pesticide workers', Lancet, (2/8051) 1 259-1 261.

WSJ, 1997, 'Filipino workers receive compensation from banana pesticide settlement fund', Wall Street Journal, 12 December 1997.