

Preventing work cancers

A workplace health priority

—

Marie-Anne Mengeot, journalist

with Tony Musu and Laurent Vogel, ETUI

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Introduction

Cancer kills around 1.2 million people each year in the European Union. Between 65 000 and 100 000 of these deaths are believed to be directly caused by working conditions. Others are the result of environmental exposures which, in many cases, are themselves related to firms' business activities.

Exposures at work and from the environment play into one another to bring about huge social inequalities in health. Work-related cancer mortality is far and away the main cause of death by working conditions in Europe.

These tens of thousands of deaths each year are not from accidents. They are preventable. Most neither stem from malfunctioning production processes, nor disrupt normal production. They are to do with technical choices about substances, processes, and work organisation.

These cancers write the stamp of labour relations into human biology. They deepen social inequalities of health. By far most affect manual workers.

The main obstacle to preventing work-related cancers is lack of control over working conditions by the workers themselves. The current level of scientific knowledge and the existence of alternative technologies make much more effective prevention possible. This brochure sets out to identify the key issues in acting against work-related cancers. It lays no claim to offer an A-to-Z analysis of all the issues. It is a contribution to reviving the trade union agenda on health and safety at work. Its main focus is chemicals. Other causes of cancer – ionising radiation, biological agents, night work, etc. – are touched on but the specific issues in preventing them are not dealt with here.

The effective way to tackle work-related cancers is through trade union action. But it is no easy task for powerful interests are at play. The chemical industry pumps huge volumes of carcinogenic chemicals onto the market, which are used in many production processes. Business has no financial incentive to prevent cancers, which often do not appear until long years after exposure. The only pressure comes from workers – especially through their trade unions – or public policies, such as through laws that prohibit chemicals or work processes or try to minimise exposure levels. Action by health and safety inspectors is also crucial to see that the laws are followed.

Public policy action is often hampered by an unwillingness to clash with business interests. The two European Commissions headed by Mr Barroso (2004-2014) have a disastrous record on protecting workers against cancer risks. The revision of Community law has been blocked, while the existing rules are so flawed as not to allow of effective prevention. While workers are exposed to hundreds of different carcinogens, the list of binding limit values contains just three: the same ones as in 1990! For the first time in 35 years, the European Union no longer has an action programme on health and safety at work.

This booklet sets out to help inform the public debate and offer means to help workers and their unions analyse and act to push back the scourge of work-related cancers. While the initial thinking had been simply to update a previous booklet published in 2007, advances in knowledge and union schemes for tackling work-induced cancer required a much more radical revision

Marie-Anne Mengeot is one of the too few journalists who take a close interest in working conditions and their health impacts. She was a pioneering documentary-maker on Belgium's public service television. She came to prominence in the 1970s with reports on asbestos, gender inequalities in the workplace, occupational cancers and musculoskeletal disorders. As a journalist, she was able to put over in clear, layman's terms the knowledge needed by the main players in taking effective action against work-related cancers – workers themselves. This brochure also includes contributions by Tony Musu and myself, and was coordinated by Denis Grégoire.

This booklet is dedicated to the memories of two people who were both leading researchers and pioneers in getting rank-and-file action going against cancer: Henri Pézerat (1928-2009) and Simon Pickvance (1949-2012). Both Henri and Simon gave unstintingly of their assistance to the European Trade Union Institute's work through the benefit of their experience, contacts, suggestions and criticisms.

– *Laurent Vogel*

Researcher, European Trade Union Institute

Chapter 1

An unequal burden of disease

Cancer is the main killer after cardiovascular disease for all men and women in developed countries. Approximately 2.6 million new cases of cancer were diagnosed in the European Union in 2012, and some 1.2 million people have been killed by the disease. Cancer accounted for 29% of male deaths (approximately 700 000 cases per year) and 23% of female deaths (over 550 000 cases per year)¹. In some countries, like France and Denmark, cancer has become the leading cause of death. In 2008, nearly 34% of deaths among the French male population and 25% of female deaths were due to cancer (Aouba *et al.* 2011). EU statistics for 2006 report this proportion rising to 41% in the 45-64 age group, making cancer the leading cause of mortality in the median age group².

The incidence of cancer may be very high in developed countries, but it is also rising sharply in emerging economy countries. Worldwide, the most common and most deadly form is lung cancer in men and breast cancer in women.

Beyond these general findings, mortality atlases show that the incidences of death, disease and cancer differ with geographical region. They can also help identify why these differences occur. In the United States, the first cancer atlas pinpointed a surplus of mouth cancers in south-western states. Later, the cause was narrowed down to the habit of chewing tobacco. Likewise, the high lung cancer death rate found along the American coasts could be put down to the World War Two boom in shipbuilding work, where exposure to asbestos was particularly high.

1. For more information, see the International Agency for Research on Cancer's estimates: <http://eco.iarc.fr/eucan> > Cancer factsheets
2. Eurostat (2006) Causes of death in the EU25, press release, 18 July 2006.

Spain's mortality atlas shows that male lung cancer death rates are highest in the Extremadura, Asturias and south-west Andalusia regions. In the latter, it is 20% above the national average and double the rate found in Navarre. This part of Andalusia also has the highest rate of manual workers in Spain, up to 80% of the working population. The same pattern is repeated in Catalonia, with a highly specific geographic distribution of lung cancer. The highest rates are found in the Barcelona region and along the Catalan coastline. In Barcelona itself, they are concentrated in the old working class districts and the new outer suburbs populated by immigrant communities.

These geographic inequalities in illness and death tend to reflect social status inequalities.

Social inequality

The first studies on mortality and life expectancy emerged in the 19th century, notably through French doctor Louis René Villermé's observations that the poorest districts of Paris had higher mortality than the more affluent ones. Other medical men subsequently made similar findings in the United Kingdom and Germany.

The late 20th century saw a resurgence of interest in studies on health inequalities in England following the publication of a report showing that social inequalities in health and mortality were persisting despite across-the-board coverage by social security schemes and improved access to health care.

While the life expectancy of the worst-off has certainly improved, the gap between them and the most affluent has not closed. The inequality starts at birth and goes on throughout life, from the cradle to the grave. It is measured by shorter lifespans for the most economically-deprived groups: from 4 to 6 years less on average for men and 2 to 4 years for women. A 2006 report commissioned by the French Presidency of the European Union found that inequalities in mortality had even increased in recent decades (Mackenbach 2006). The life expectancy gap between the highest and lowest socioeconomic status men in England and Wales rose from 5.4 years in 1970 to more than 8 years in the 1990s.

These findings are borne out by a 2010 Belgian study. In 1991, males in Belgium with the highest educational attainments enjoyed five years more life expectancy at age 25 than those with the lowest; by 2001, the gap had risen to seven and a half years, while that in women's life expectancy by educational level increased from three to six years (Van Oyen *et al.* 2010).

Poverty, unemployment, and poor working conditions are big contributors to these inequalities. In Seville, for example, researchers showed that well-to-do men and women had life expectancies 8 years and 4.5 years longer, respectively, than those from poor backgrounds. The researchers argued that loss of a job and unemployment materially affect life expectancy and mortality. Unemployment black spots had excess mortality rates of 15% among males and 8% among females.

Another example comes from the Nord-Pas-de-Calais region in France, which has the country's highest cancer incidence rates – 669 in every 100 000 men and 372 in every 100 000 women, against national averages of 504 per 100 000 men, and 309 per 100 000 women. These high cancer rates affect life expectancy in the Nord-Pas-de-Calais, which on average is 3.6 years shorter for men and 2.8 years for women than in the south of France³.

3. Revue Prescrire (2007) Espérance de vie, cancers : les deux France, 279, 66-67.

But the kind of cancers found in the north are not different from those found elsewhere in France. The head of the regional health observatory attributes the regional gap to “the higher percentage of people vulnerable to poverty” in the north. “The pattern of excess cancer mortality reflects pockets of unemployment and poverty; a legacy of the collapsed industrial and mining fabric”, he adds⁴.

While unemployment may be a factor of social inequality in illness and death, so, too, is work. A study of the influence of social factors in cancer deaths in Cadiz shows that excess cancer mortality rises in an inverse relationship with social status. The excess cancer mortality here is due to a surplus of cancers of the larynx, lungs, bronchi and pleura. Alongside traditional factors like drinking and smoking, the authors point to occupational factors. This region of Andalusia is home to furniture, footwear, aluminium manufacture, and shipbuilding yards, where the workers were exposed to acids, paint, chromium, arsenic and asbestos.

Men aged 25-54 living in the Nord-Pas-de-Calais have a higher death rate from cancer than in other French regions for all social status categories, but in very different proportions: 9% higher for senior managerial staff; 30% higher for technician and skilled craft occupations/self-employed skilled workers/independent retailers; 60% higher for manual/office workers (Aïach *et al.* 2004). Nationally, if mortality among senior managerial staff and professionals is taken as 1, the excess mortality ratio for manual/office workers is 2.9 for mortality from all causes, and 4 for cancers. The ratios are higher in the Nord-Pas-de-Calais at 4 and 5, respectively. Work-related risks obviously have an impact on the excess cancer incidence reported there. It is telling that the rate of recognised occupational cancers in the Nord-Pas-de-Calais is nearly double that of the Paris region. In the 1960s and 1970s, half the region’s labour force consisted of manual workers, many working in the mining, iron and steel, and shipbuilding industries where exposure to different carcinogens – especially asbestos – was commonplace.

The national institute of health and medical research (Inserm) studied laryngeal and hypopharyngeal cancers – usually associated with smoking and drinking – in 15 French hospitals (Menvielle *et al.* 2004). It found that manual workers have a two and a half times greater probability than non-manual workers of developing these cancers. The study’s authors attributed a third of this excess risk to occupational factors.

This is unsurprising, given that exposure to all forms of asbestos is a cause of cancers of the larynx and pharynx, and that other substances like cement dust or silica also play a contributory role (IARC 2012a, Paget-Bailly 2012).

A report by the national institute of statistics and economic research (Insee) in October 2011 reviewed nationwide social differences in mortality for France (Blanpain 2011). The first finding was that among both male and female workers, managerial staff and professionals have the longest life expectancy and manual workers the lowest. So, in the period 2000-2008, male managers and professionals had a life expectancy at age 35 six years longer than manual workers, or 47 years against 41. The female gap was three years, with a life expectancy at age 35 of 52 years for managerial staff and 49 years for manual workers. The second finding was that improving life expectancy has mainly benefited the higher occupational categories. The Insee report found that mortality had declined steadily since 1976, but differentially for men across socioeconomic status categories. By contrast, inequalities in mortality have remained virtually unchanged since 1980. Inequality in mortality is particularly pronounced for premature death: at age 45, the probability of dying within one year is 2.5 times higher for a male

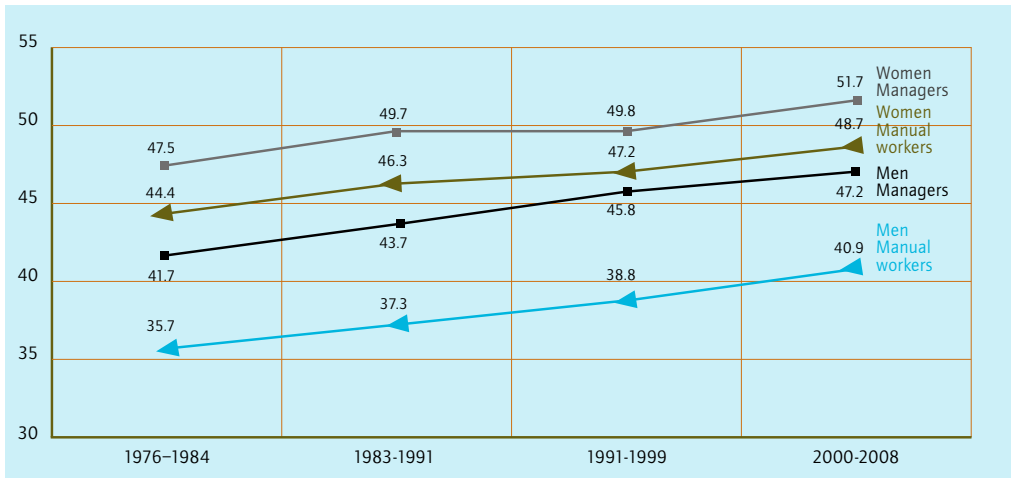
4. AFP (2007) Cancer : le Nord/Pas-de-Calais est la région française la plus affectée, 17 January 2007.

manual worker than a manager, and 1.3 times higher at age 90. The report’s authors argue that behaviours and lifestyles, but also physically harsher working conditions and a higher incidence of exposure to occupational hazards, work against manual workers.

Social inequalities in cancer obviously play a big role in these inequalities in mortality. Research reveals the following trends for France: marked social inequalities in cancer mortality are seen among men, especially for cancers of the upper aerodigestive tract (mouth, pharynx, larynx). Social inequalities are less marked among women, but are found for the uterus, stomach and lung. Social inequalities in cancer mortality increased among men between 1968 and 1981, showing little change since the 1980s. The increase is particularly marked for cancers of the upper aerodigestive tract. The lower mortality from breast cancer observed among women with lower educational attainment in the early 1970s gradually declined to zero in the late 1990s (Menvielle *et al.* 2008).

This linkage between cancer, life expectancy and social status is not specific to the Nord-Pas-de-Calais, France, or Spain. Manual workers in all European countries have a death rate at ages 45-59 – i.e., a premature mortality rate – higher than non-manual workers, ranging in some case up to double. Even in northern European countries noted for their advanced social welfare system and greater equality in access to health care, cancer risks are very much determined by socioeconomic status.

Graph 1 Life expectancy at age 35 by sex for managers and manual workers



Source: INSEE (2011) Permanent Demographic Sample

Global inequality

The International Agency for Research on Cancer (IARC) reports a higher cancer incidence and cancer mortality in low-income groups in all industrialised countries. In the past half-century, the incidence of lung cancer has fallen in the highest-income groups, but has risen steadily among the lowest-income groups. The IARC specialists argue that this difference is not just due to different smoking habits in the social groups. They also claim that exposure to carcinogens in the working environment may account for a third of the observed difference between the cancer incidences in higher and lower income groups, rising up to a half for lung and bladder cancer.

The undoubted social differences in the incidence of smoking cannot explain all the observed cancer inequality. While in the male population, there is a 20% differential in the prevalence of smoking between manual workers and managerial staff, manual workers have an excess premature cancer mortality rate of about 200% compared to managerial staff (Thébaud-Mony 2006).

Additionally, the available data on recognised cases of work-related cancer point to a concentration among manual workers and low-income groups. This is unsurprising, given manual workers' greater exposure to carcinogens, as reported by Sumer, the large-scale French survey on exposure to work hazards (see p. 25).

Chapter 2

Occupational cancers.

An over-long story with no ending

Diseases that are associated with certain occupations have been described by authors from ancient times onwards, but only as isolated observations. The first systematic descriptive account of different diseases affecting craft workers and labourers is that made by Bernardino Ramazzini, Professor of Medicine at the University of Padua, in his “Treatise on the Diseases of Workers”, published in 1700⁵. In the foreword, he writes, “Are we not forced to the conclusion that several trades are a source of illness to those who carry them out, and that tradesmen, finding the most serious illnesses where they hoped to find their and their family’s livelihood, die hating their thankless occupation? Having in my practise had frequent occasion to observe this misfortune, I have set myself to writing about the diseases of workers.”

This uncommon physician was not content merely to describe, but set about “suggesting medical precautions for the prevention and treatment of such diseases as usually affect workers”. He recommended that his colleagues should add to the list of questions that Hippocrates advises doctors to ask their patients, the question “What occupation does the patient follow?” Three centuries on, this is still a question that needs to be put. And as in Ramazzini’s time, it is still today unfortunately not being asked enough.

In his work, the father of occupational medicine describes in detail the diseases to which workers in more than 50 occupations are prone, including miners, quarry-workers, chemists, textile workers, glassmakers, painters, grave-diggers,

5. Ramazzini B. (1855) *Essai sur les maladies des artisans*, traduit du latin par M. De Fourcroy, nouvelle édition faite sur celle de 1778, Paris, Adolphe Delhays Libraire.

midwives, wet-nurses, and many more. He reports respiratory disorders, asthma, coughs, skin diseases, the risks of infectious and parasitic diseases, mercury, lead and antimony poisoning, but nowhere does the word “cancer” appear in his writings. It was not until nearly a century later that another doctor demonstrating the same observational bent would report the first occupational cancer.

Chimneysweep's cancer, the first identified occupational cancer

Cancer of the scrotum, known as “chimneysweep’s cancer”, is the first cancer attributed to work-related exposure. In 1775, an English surgeon, Percival Pott, gave an account of the incidence of scrotal cancer among men who had been chimneysweeps when young. Hitherto, it had been thought to be a purely venereal disease. In 18th century England, chimneysweeps were often children, climbing naked in sometimes narrow, blistering chimneys. Pott ascribed chimneysweep’s cancer to the build-up of soot and tar in clothing and the folds of skin covering the testicles.

The disease appeared to be unknown on the Continent. English doctors crossed the Channel and found that occupational cancers could be avoided by relatively simple preventive measures. They observed that chimneysweeps on the Continent, especially in Germany, had long worn a special head-to-toe covering that was fastened tightly about the wrists and prevented soot from entering into contact with their body. They also observed that these chimneysweeps were very careful about their personal hygiene.

A century after Pott’s discovery, other doctors reported cancers of the scrotum in textile workers exposed to mineral oils. A carcinogen in these oils caused a veritable epidemic of scrotal cancer among workers in the English cotton industry after 1910. But it was not until the 1930s that the carcinogen was identified as benzo[a]pyrene, along with a series of polycyclic aromatic hydrocarbons (PAHs) present in coal and oil by-products. In fact, PAHs are all around us. They are found in cigarette smoke, and many workplaces may be contaminated with them through fumes, gases, soot, and heat-degraded oils.

Scientific knowledge about their cancer-causing properties has not stopped coking plant workers, exposed to PAH-containing fumes, from continuing to have double the death rate from bronchial cancers.

The incidence of scrotal cancer among chimney sweeps may have fallen, but they continue to be affected by other types of cancer. A study of a period from 1960 to 2005 in five countries of northern Europe on the occupations of cancer sufferers suggests that chimney sweeps have one of the highest rates of cancer. The study – which collected data from more than 15 million people – is part of the Nordic Occupational Cancer (NOCCA) project (Pukkala *et al.* 2009) which sets out to identify the link between occupations and cancer (see also Chapter 3).

PAHs may cause not only skin cancers, but also cancers of the lungs, throat, larynx and oesophagus (food pipe). The Nordic study’s authors consider that PAH exposure may also partly explain the high rate of bladder cancer seen among chimney sweeps.

Chimney sweeps are not alone in being exposed to benzo[a]pyrene and other PAHs. Workers in aluminium production or in contact with bitumen during asphaltting and roofing with waterproofing membranes can also be highly exposed. In November 2012, the Lyon Court of Appeal held a major public works contractor, Vinci, liable for the death of a 56-year-old worker from skin cancer contracted after 20 years’ working in contact with bitumen. In Denmark, analysis of data from a cohort of 679 asphalters showed them to have a risk of lung cancer three and a half times that of the general male population (Hansen and Lassen 2011).

In 2010, more than 200 years after Pott's observations and 80 years after benzo[a]pyrene and PAHs were identified as carcinogens, the International Agency for Research on Cancer (IARC) classified benzo[a]pyrene as carcinogenic to humans based on its toxicity for many animal species (skin cancer and lung cancer), and its mechanisms of action. The IARC also classified exposure to bitumen during paving and roofing with coal-tar pitch as probably carcinogenic in 2011.

What does this tell us? Firstly, that while a carcinogen may preferentially target one organ it may still also invade others. Secondly, the same carcinogen can occur in very different work environments. Thirdly, preventive measures tend to be less systematically taken for carcinogens released during a production process than for substances identified as carcinogens used in the production cycle. Fourthly, working conditions influence whether cancer is contracted or not. So, very different bronchial carcinoma rates were found between coke oven plant workers in different plants and in different countries depending on the preventive measures taken or technological processes used. Thus proving that occupational cancers are not an inevitability.

Deadly dye!

The chemical industry developed around 1860 on the back of the dye industry. Some years before, a chemist had chanced on a way to synthesise aniline mauve, an arylamine present in coal tar. Before the 19th century was out, Ludwig Rehn a surgeon in Frankfurt was reporting carcinoma of the bladder among dye factory workers. Between 1895 and World War Two, hundreds of cases of bladder cancer were being reported among dyestuff and synthetic colour industry workers.

In 1938, William Hueper an American researcher demonstrated the potential animal carcinogenicity of certain arylamines, especially beta-naphthylamine, which was also used as an antioxidant in the rubber industry, while rubber itself was used in other industries like cable-making, where cases of bladder cancer were also being reported (Lower 1982).

After World War Two, the British chemical industry commissioned a workforce-wide survey (Case *et al.* 1954). It found that one in ten workers who were exposed to arylamines developed carcinoma of the bladder. The survey's authors concluded that with an average 18 year latency period, the final prevalence of bladder cancer would be 23% among workers exposed to arylamines, and 43% among workers exposed to betanaphthylamine only. Compared to the general population, this works out at a 30-times higher probability of dying of bladder cancer for all arylamines, and 60-times higher for beta-naphthylamine only. The United Kingdom halted manufacture of beta naphthylamine in 1949, and benzidine in 1962. And still continental Europe carried on regardless...

The trial of managers of the IPCA dye factory in 1977 caused an international outcry. Dubbed the "fabbrica del cancro" (the cancer factory), 132 of its workers and ex-workers had died of bladder cancer over a 20-year period. In 1990, Belgian journalists made the stupefying discovery that workers were still dying of bladder cancer from exposure to benzidine and beta-naphthylamine in the "Les colorants de Tertre" dye works in the Mons region⁶. No-one seemed to have alerted them to the cancer risks of the chemicals they were handling. The four most lethal aromatic amines were not outlawed across Europe until 1988 by

6. Attention à la couleur, RTBF (French-speaking public TV station), programme broadcast on 10 June 1990.

a directive⁷. But many other aromatic amines are still in use in synthetic dye and pharmaceuticals production, as well as the rubber and plastics industries, and may well be behind the raised bladder cancer risk among hairdressers who often use hair dyes with no special protection (Lower 1982).

Asbestos: a nightmare health scenario dawns

The industrial use of asbestos gradually developed from the latter half of the 19th century, with the discovery of major deposits in Quebec and South Africa. Outside the immediate big producers/users (mining, textiles, felts, paperboards, brakes, asbestos cement, asbestos flocking), a wide range of occupations were exposed to asbestos in the insulation, chemical, iron and steel and power generation industries, shipbuilding, transport, painting, joinery, decoration, etc.

In 1906, the first industrial era account of an asbestos-related pulmonary disease was given to a UK parliamentary committee. In the same year, a French occupational doctor recorded cases of pneumoconiosis, specific pulmonary tuberculosis and pulmonary tuberculous sclerosis in an asbestos textile spinning and weaving factory at Condé-sur-Noireau, in Normandy⁸.

In 1935, British doctors reported a case of lung cancer in a patient with asbestosis (pulmonary fibrosis caused by asbestos). In 1947, His Britannic Majesty's Chief Inspector of Factories recorded in his annual report that the autopsies of 235 people whose deaths were attributed to asbestosis revealed the presence of lung cancer in 13.2% of cases, but only 1.3% among workers who had died of silicosis.

In 1955, a British epidemiologist published what has since become a benchmark study (Doll 1955), reporting that in the study population of asbestos workers, the risk of developing lung cancer was 10 times higher than in the general population. This finding was to be reinforced by much other research.

1960 is another landmark date in the history of understanding of asbestos-related diseases. It was the year in which the findings of a South African pathologist were published, making a link between cases of mesothelioma – pleural carcinoma – and exposure to asbestos (Wagner *et al.* 1960). The connection between asbestos and mesothelioma was gradually confirmed to the point where mesothelioma has now become an epidemiological indicator of asbestos exposure.

Asbestos has been phased out of use in Western Europe. It was banned in Denmark in 1986, in Italy in 1992, in France in 1997, in Belgium in 1998, and in all European Union countries in 2005. It was not before time, but too late for many workers. As the International Labour Organisation (ILO) points out, the latency of asbestos-related diseases holds the world in an “iron grip”. One of its senior officials argues that “asbestos is one of the most, if not the most important single factor causing work-related fatalities, and is increasingly seen as the major health policy challenge worldwide”⁹.

Asbestos was a foreseeable disaster. And yet, unbelievably, the carnage goes on. In those countries that have outlawed asbestos – fifty-odd in 2013 – the material which has

7. Council Directive 88/364/EEC of 9 June 1988 on the protection of workers by the banning of certain specified agents and/or certain work activities, Official Journal of the European Communities, 9 July 1988.

8. Auribault D. (1906) Note sur l'hygiène et la sécurité des ouvriers dans les filatures et tissages d'amiante, Bulletin de l'inspection du travail, 14, 120-132.

9. ILO (2006) Asbestos: the iron grip of latency.

been used for a century still abounds in workplaces, office buildings, blocks of flats and houses, as well as in means of transport like railway carriages/trucks and ships. Workers in many occupations will be faced with it for long still to come.

Even in countries that have banned asbestos the mesothelioma epidemic rages on, as a study done in the state of Massachusetts in 2013 (Roelofs *et al.* 2013) shows. The authors examined 1,424 cases of mesothelioma occurring between 1998 and 2003. They found no decrease in the number of new mesothelioma cases in the shipbuilding and construction industries, and found the disease occurring in hitherto apparently untouched jobs such as: chemical engineers, machine operators, mechanical engineers in motor and machine tool manufacturing, railway employees and the U.S. Postal Service.

“Errare humanum est, perseverare diabolicum” (to err is human, to persist [in folly] is evil). This old adage fits the history of asbestos to a ‘T’. Globally, asbestos production remains high and broadly unchanged since the early 2000s at close to 2 million tpa. In 2012, Russia produced half, selling the bulk of it abroad, followed by China and Brazil. The biggest asbestos users are China (more than 30% of world consumption in 2011) followed by India (15% of world consumption).

The ILO estimates that up to 100 000 people die each year in the world as the result of an occupational exposure to asbestos.

Wood dust: a low profile killer

Asbestos fibres are not the only source of cancer. Some kinds of wood dust cause a specific kind of sinus cancer – ethmoid carcinoma. The discovery dates back to 1965, when doctors in the Oxford area began seeing an abnormally high number of sinus cancer cases. They observed that the patients were mainly carpenters and cabinetmakers. Puzzled by this, they consulted the regional cancer registry to find a concentration of nasal cavity carcinomas – mainly among woodworkers – in a small area of Buckinghamshire where many furniture factories are located. A large-scale national survey confirmed their findings (Acheson *et al.* 1972).

On the Continent, doctors in France, Belgium and Denmark were not long in coming to the same conclusions. Ethmoid adenocarcinoma became a recognised occupational disease in England in 1969, in Belgium in 1976, France in 1981, and Germany in 1987.

The British researchers’ investigations into ethmoid adenocarcinoma uncovered a higher rate of nasal cancers among leather and footwear industry workers. The highest risk was found among workers in the preparation and finishing shops, where cutting, polishing and sanding operations exposed them to high concentrations of leather dust. Hotly-disputed at first, the findings were definitively confirmed in 1988 by a Danish study (Olsen 1988).

This kind of joining-up of data, if extended to other European countries, could in future help bring to light as yet unidentified risks and confirm statistical associations. So, the Nordic occupational cancer study (NOCCA) found that dust from not just some but all types of wood could cause cancer. Both men and women employed in furniture manufacture in the Nordic countries have almost double the risk of developing nasal cancer as the rest of the population, and for exposed men it was 5.5 times higher for a particular type of nasal cancer (adenocarcinoma).

Carcinogenic chemicals: toxicity often adjusted upwards

Four aromatic amines were banned in 1988, but many other carcinogenic chemicals are still in production and use. The list of chemicals that are known and possible human carcinogens established and regularly updated by the International Agency for Research on Cancer (IARC) is a very long one. Here, we look at three whose toxicity has regularly been adjusted upwards over time.

Formaldehyde is a natural component of living systems. It is a product of oxidation and can be formed in plants, mammals and humans alike. Formaldehyde is released into the environment by the combustion of organic matter (incinerators, power plants, motor vehicles, etc.). As a chemical, it is used in manufacturing a wide range of building materials and consumer goods. It is found in adhesives and resins used as binders for wood products, pulp, paper, glasswool and rockwool; plastics and coatings, especially for textile finishing; in the chemical industry; and as a disinfectant and preservative (embalming). In the 1990s, up to a million workers were exposed to formaldehyde in the Europe of Fifteen (IARC 2012b). At the same time, approximately 2 million American workers were also exposed to it, nearly half in the clothing industry (National Toxicology Program 2011).

In the 1980s, researchers began seeing an excess mortality among workers exposed to formaldehyde for certain types of cancers. In light of published animal studies reporting nasal cancer cells, the IARC classified formaldehyde as probably carcinogenic to humans in 1995 (Pinkerton *et al.* 2004).

After a long series of epidemiological studies confirming the risk of cancers of the nasal cavity and paranasal sinuses among exposed workers, the IARC finally classified formaldehyde as a known human carcinogen in 2004. In Denmark, a study using data from the cancer registry showed that among those who developed cancer of the sinuses, the risk of developing such cancers was three times higher among workers exposed to formaldehyde (Hansen and Lassen 2011).

In 2009, the IARC reconsidered formaldehyde and confirmed the risk of nasopharyngeal cancer, but added leukaemias (particularly myeloid leukaemia, a cancer of the blood affecting the white blood cell) as known human carcinogens. A number of studies have shown a more than 50% increased risk of this form of leukaemia among workers exposed to formaldehyde.

By the criteria of the EU's CLP Regulation on the classification and labelling of chemicals, formaldehyde is considered as a category 2 "suspected" human carcinogen (see Chapter IV). The European Trade Union Confederation (ETUC) called for formaldehyde to be reclassified in 2011, while a proposal to reclassify formaldehyde in category 1A (known carcinogen) was submitted to the European Chemicals Agency (ECHA) at the French authorities' request in the same year. In November 2012, ECHA's Committee for Risk Assessment issued an opinion for the classification of formaldehyde as a category 1B carcinogen – a substance which is presumed to have carcinogenic potential for humans. A comprehensive EU assessment of formaldehyde is planned for 2014 and its classification in category 1B in the CLP (Classification, Labelling and Packaging) Regulation in 2015.

If formaldehyde is recognised by the EU as a category 1B carcinogen, it will be more stringently regulated in both consumer products and professional uses, including being replaced by less toxic substances if possible. It could also be subject to an authorisation requirement or restrictions on its use under the REACH Regulation, but these are long drawn-out processes that can take from five to eight years.

In 2011, the U.S. authorities took the decisive step to classify formaldehyde as a known carcinogen, adding it to a list of 240 chemical and biological substances which now includes 54 known carcinogens and 186 probable carcinogens.

Trichloroethylene and **perchloroethylene** are chemicals in the group of chlorinated ethylenes which also includes ethylene and vinyl chloride. Trichloroethylene (TCE) and perchloroethylene (PCE) were produced in large quantities and were the most commonly used chlorinated solvents in the 20th century in a wide range of applications: dry cleaning, medical uses as anaesthetics (TCE) and antiparasitic agents (PCE), for degreasing metals and manufacture of water supply pipe liners (PCE). The earliest information about their toxicity came from assessments of their therapeutic use. Their effects on occupationally exposed workers were first analyzed in terms of acute effects. Only since the 1970s, when vinyl chloride was discovered to be carcinogenic, did the toxicity of TCE and PCE begin to be looked at amid much controversy (Ozonoff 2013).

Trichloroethylene (TCE) caused cancers of the liver, kidneys, lungs, testes and blood system in animal experiments, while the main finding in humans was its renal toxicity. A French study showed that workers highly exposed to TCE had double the risk of kidney cancer to that of unexposed workers (Guha *et al.* 2012).

In 2012, the IARC classified TCE as carcinogenic to humans. In the European Union, it is considered as a category 1B carcinogen, i.e., it should be regarded as if it is carcinogenic to humans.

TCE is still used for degreasing, but its main use is in the production of chlorinated products.

Perchloroethylene (PCE), also called tetrachloroethylene has mainly been used in dry cleaning, where it is still widely used. It was also extensively used for degreasing metals and in the production of chlorofluorocarbons, or CFCs, the gases accused of destroying the ozone layer, most of which have been banned under the Montreal Protocol.

Epidemiological studies have linked perchloroethylene to a range of cancers. Most studies have focused on workers in the dry cleaning industry, where the link with bladder cancer was considered the most substantial. In 2012, the International Agency for Research on Cancer (IARC) classified perchloroethylene as probably carcinogenic to humans (Group 2A). The European Union considers it as suspected of causing cancer – Category 2 in the CLP Regulation.

Since the IARC's 2012 evaluation, a Canadian study has reported that workers exposed to perchloroethylene have double the odds of contracting lung cancer (Vizcaya *et al.* 2013). A study in four countries of northern Europe found a slightly increased risk of liver cancer and non-Hodgkin lymphoma (cancer of the lymphatic system) (Vlaanderen *et al.* 2013). The environmental risks of urban dry cleaning shops focused attention on perchloroethylene, and it has been unlawful to use the solvent in any new dry cleaning shop in Norway since 2005 and in California since 2008. It has been unlawful to install new dry cleaning machines using perchloroethylene in premises adjoining occupied premises in France since 1 March 2013, and existing machines in such premises will gradually be made unlawful. France has 4 500 dry cleaning shops and 5 200 machines using perchloroethylene. Approximately 25 000 people work in commercial drycleaners.

Perchloroethylene is still permitted in commercial drycleaners that do not adjoin occupied premises. What, therefore, of the health and safety of the French workers who remain exposed to it? Not to mention other workers elsewhere in the world? The European Chemicals Agency (ECHA) is to make a full assessment of perchloroethylene under the REACH Regulation.

These three examples of chemicals whose toxicity has been repeatedly revised upwards raise questions about the validity of the preventive measures taken and exposure

limit values set on the basis of often already outdated knowledge. When all is said and done, who are they protecting? Because these are not isolated examples. The carcinogenicity of a number of other chemicals such as polychlorinated biphenyls (PCBs), ethylene oxide, butadiene, acrylamide, epichlorohydrin, ethylene bromide, 2-nitrotoluene, polybrominated biphenyls, styrene, and more has been revised upwards by the International Agency for Research on Cancer (IARC).

An old killer still going strong: crystalline silica

Asbestos is one tragedy, silicosis another. It came before and is comparable to the asbestos disaster by the sheer number of victims. Sadly, lessons were not learned.

In European countries, many workers still bear the legacy of coal mining. In 2009, figures from the annual report of Belgium's Occupational Diseases Fund (FMP) indicate that up to 10 000 workers are receiving compensation for silicosis and silicosis combined with pulmonary tuberculosis. In Germany, 1 097 new cases of silicosis and 3 cases of silicosis combined with pulmonary tuberculosis were recognised in 2011.

Like asbestos diseases, silicosis is an irreversible lung disease. It is caused by exposure to crystalline silica dust, the main form being quartz. Exposure to silica occurs mainly during milling, sawing, drilling or polishing stone materials that give off respirable quartz dust. The amount of quartz dust given off during these operations depends on the composition and nature of the material.

Respirable quartz dust is very fine, barely visible. It consists of tiny insoluble particles that penetrate deep into the lungs. It is these fine particles that can cause silicosis. There are several types of silicosis: acute silicosis from massive exposure can cause death within three years; so-called early forms can appear within five years; delayed forms can occur after several years' exposure, sometimes many years after the last exposure. Silicosis is a progressive and irreversible incapacitating disease. Crystalline silica can also cause chronic obstructive bronchitis. And that is not the whole story.

In 1996, the IARC put crystalline silica on its list of known human carcinogens, being of the opinion that it could cause lung cancer.

Industries where workers are exposed to crystalline silica abound. They include: mining, quarrying, ceramics, glass, cement and pre-cast concrete, natural stone, foundries, jewellery, dental prostheses and all parts of the construction industry.

Many building trades are concerned: asphalters, concrete workers, screed layers, bricklayers, plasterers and plasterboard fitters, demolition workers, tilers, pavers, etc.

An EU-wide survey done in the early 1990s (Carex, Carcinogen Exposure) indicated that crystalline silica was among the most common workplace carcinogens. The survey estimated that over 3.2 million workers were affected in the Europe of Fifteen, 87% of them in the construction industry. A million workers were exposed to crystalline silica dust in Germany, about half a million in the UK and another half-million in Spain.

Crystalline silica is still not classified as carcinogenic in the European Union. In December 2012, the European Advisory Committee on Safety and Health at Work (ACSH) adopted an opinion calling for the Carcinogen Directive to be revised (see Chapter 4) to add EU-wide binding occupational exposure limit values (OELVs) for 20 substances, including crystalline silica. The OELV for crystalline silica proposed by the Scientific Committee for Occupational Exposure Limits to chemicals (SCOEL) is 50 mcg/m³ (micrograms per cubic metre). That proposal was made in 2003. It corresponds to the recommendation made in 1974 by the U.S. Institute for Occupational Safety and Health (NIOSH). Despite the health

issues involved, the European Commission has still not put forward a binding limit value for exposure to silica. In August 2013 – 30 years after NIOSH first recommended it – the 50 mcg/m³ standard finally featured in a bill put up by the U.S. Occupational Safety and Health Administration (OSHA), whose director claims that applying the standard could save 700 lives and prevent 1 600 new cases of silicosis each year in the United States.

In Germany, where lung cancer caused by crystalline silica is a recognised occupational disease, 898 cases were compensated between 1978 and 2010, and 69 new cases in 2011. Also in 2011, Germany compensated 2 000 cases of silicosis.

Chapter 3

Workers and carcinogens

Set up in 1971, the International Agency for Research on Cancer (IARC) evaluates the cancer-causing potential of substances and agents (chemicals, biological and physical agents), situations where exposure occurs, and industrial processes.

The IARC classifies these into 5 categories:

- Group 1: carcinogenic to humans
- Group 2A: probably carcinogenic to humans
- Group 2B: possibly carcinogenic to humans
- Group 3: not classifiable as to carcinogenic to humans
- Group 4: probably not carcinogenic to humans.

To date, the IARC has evaluated over 1 000 substances, approximately 460 of which have been identified as carcinogenic or potentially carcinogenic¹⁰. Of the hundred substances classified as group 1 – i.e., proven to be carcinogenic to humans – 60 are found in the working environment. Group 1 carcinogens include those cited earlier, plus arsenic, benzene, beryllium, cadmium, Chromium VI, formaldehyde, ethylene oxide, and vinyl chloride. The list of Group 1 carcinogens also includes certain mixtures, in particular tobacco products, wood dust, and tar as well as particular industrial processes like shoe manufacture and repair, rubber, iron and steel production, and jobs like painter or fire-fighter. Also on the carcinogens list are physical agents, like ionising radiation and ultraviolet radiation, as well as biological agents, like certain viruses, and even some medicines and medical treatments.

10. See IRAC website: <http://monographs.iarc.fr>

There are too few epidemiological studies on the cancer-inducing potential of non-physical aspects of working conditions. Factors such as stress, job insecurity and irregular working hours have regularly been named as potentially cancer-inducing. In 2007, the IARC classified shift work as probably carcinogenic to humans (Group 2A) (see also Chapter 6).

The IARC classification is established by committees of international experts in carcinogenesis. It does not have the force of regulations, but does give a “state of play” on what is known about the carcinogenicity of a particular substance. The European Union has compiled its own list of carcinogens. The European classification is laid down in regulations (see Chapter 4).

How many workers exposed to carcinogens?

The first study identifying work-related hazards was done in the United States in the early 1970s. It identified more than 9000 potentially hazardous work situations and categorised the groups exposed to carcinogens¹¹. The next investigation followed in the early 1980s, based on detailed on-site surveys¹², which enabled the U.S. National Institute of Occupational Safety and Health (NIOSH) to develop a database from which to estimate the number of people exposed and the sectors concerned for a given harmful factor.

In the European Union, the Carex (Carcinogen Exposure) study is the only comprehensive research to put figures on the proportion of workers exposed to carcinogens (Kauppinen *et al.* 2000). Carex is an initiative launched in the late 1980s as part of the “Europe against Cancer” programme¹³.

Europe, Carex 1990-1993: a quarter of workers exposed to carcinogens

The Carex project is based on the estimated percentage of exposed workers in Finland and the United States. Generally, the Finnish estimates were lower than the U.S. estimates because they excluded workers exposed to lower doses. One limitation of Carex was its failure to make gender-differentiated estimates. For each country, experts assessed the distribution of employment across economic sectors, from which they calculated the percentage of workers exposed to different risks, basing their estimates on the American and Finnish databases adjusted for their own assessment of actual conditions in their country. The Carex assessment also disregards within-career changes of job. It more resembles a point-in-time snapshot of the distribution of all employees between occupations. Factoring in the total duration of working life would yield higher percentages.

The overall result for the period 1990-1993 was for the fifteen countries in the EU in 1995. The share of workers exposed to carcinogens was 23%, ranging from 27% in Greece at the top down to 17% in the Netherlands at the bottom, and representing a total of 32 million workers. After 1995, the Carex project was extended to the three Baltic republics and the Czech Republic, where it found 28% of workers exposed to carcinogens at work. The project was never extended to the other EU countries.

11. See: www.cdc.gov/niosh/docs/89-103

12. See: www.cdc.gov/noes

13. Carex can be found at www.ttl.fi/Internet/English/Organization/Collaboration/Carex

The carcinogens to which workers were generally exposed were solar radiation (9.1 million people), passive smoking (7.5 million), crystalline silica (3.2 million), diesel engine exhausts (3.1 million), radon (2.7 million), wood dust (2.6 million), lead and its inorganic compounds (1.5 million), benzene (1.4 million). Below these were: asbestos, ethylene dibromide, formaldehyde, PAH, glass wool, tetrachloroethylene, Chromium VI and its compounds, sulphuric acid mists, nickel, styrene, chloromethyl and trichloroethylene.

The economic sectors where exposure to carcinogens was highest were: forestry work (solar radiation), fishing (solar radiation), mining (silica and diesel engine exhausts), the wood and furniture industry (wood dust and formaldehyde), ores (silica), construction (silica, solar radiation and diesel engine exhausts) and air transport (passive smoking and ionising radiation). Exposure to benzene was highest in the vehicle repair sector.

The Carex estimates cover all workers and allow for exposure to environmental carcinogens – like solar radiation, radon and passive smoking – which occurs for 75% of working time. Their big advantage is to provide public policymakers with a set of data with which to drive occupational cancer prevention policies.

Since Carex, a mixed series of changes have occurred. The percentage of workers exposed to second-hand smoke and asbestos has decreased due to stricter legislation. But the number of known carcinogens has increased. Any update of the estimates would have to extend the 139 carcinogens listed by Carex in Europe. An application of Carex currently underway in Canada is looking at 229 carcinogens or suspected carcinogens and conducting gender-differentiated assessments in different sectors of activity¹⁴.

The declining share of industrial and agricultural employment should contribute to bringing down the percentage of exposed workers. However, some service sectors (cleaning, health care, transport) may pose cancer risks that have traditionally been ignored. Contingent employment increases the probability of exposure during part of working life and the probability of exposures at different periods. All told, it is unclear whether the percentages calculated twenty years ago should be adjusted up or down.

France, Sumer 2010: 10% of workers exposed to carcinogenic chemicals

France's Sumer survey (the French acronym for medical monitoring of exposures to occupational hazards) designed in the early 1980s was conducted for the first time in 1987. It has been repeated every seven years since and extended each time to a larger population of workers. It is based on occupational doctors' questioning of individual workers about all their activities in the week preceding the survey, supplemented by the doctor's own knowledge of job-related exposures.

Unlike the Carex system, the Sumer survey percentages take no account of exposure to sunshine, second-hand smoke or radon, which explains the significant difference between the two sources' estimates.

14. See: www.carexcanada.ca/en

Main carcinogens identified in the Sumer 2010 survey (in descending order of the number of employees exposed)

- Diesel exhaust fumes
- Natural mineral oils
- Wood dust
- Crystalline silica, formaldehyde
- Lead and its derivatives
- Coal tar and its derivatives
- Aromatic hydrocarbons
- Halogenated and nitro hydrocarbons
- Chromium and its derivatives
- Nickel

- Asbestos and refractory ceramic fibres
- Metallurgical and electrometallurgical fumes
- Cobalt and its derivatives
- Trichloroethylene
- Aromatic amines
- Various cytostatic agents
- Cadmium and its derivatives
- Sintered metal carbides
- Benzene, perchloroethylene
- Acrylamide
- Phenol-formaldehyde resins
- Vulcanisation fumes
- Arsenic and its derivatives

Source: Dares Analyses (2013) Les expositions aux produits chimiques cancérogènes en 2010

The Sumer 2010 survey covered nearly 50 000 workers representing up to 22 million employees (DARES 2012). The published findings indicate that one in three employees is exposed to at least one chemical in the course of their work. Some sectors are more affected: in the construction industry in particular, more than half of employees are exposed to at least one chemical and nearly a third (29%) are exposed to more than three chemicals. In health care, half of hospital employees are exposed to at least one chemical and a quarter to more than three chemicals. Manual workers are most affected by this multi-exposure – 32% of skilled workers and 23% of unskilled workers compared to 14% of all private sector employees.

In 2010, 10% of all employees – nearly 2.2 million French workers – were exposed to at least one chemical carcinogen. Manual workers are still most at risk. They account for more than two-thirds of the employees exposed to at least one carcinogen although making up only 29% of the workforce. Employees in some big industry sectors are particularly exposed: maintenance (43%), building and public works (32%), metal working (31%).

Young people suffer more exposure than other age groups: 16% of employees under the age of 25 are exposed to at least one chemical carcinogen, compared to 7% of the over-50s. In the mechanical engineering/metalworking industry, 70% of apprentices are exposed versus 35% of all manual workers.

As in 2003, the 2010 survey focused on IARC Groups 1 and 2A carcinogens, with the most frequently mentioned being substantially the same as in 2003: diesel exhaust fumes, mineral oils, wood dust and crystalline silica still head the list (see box). While women generally suffer much less exposure than men, some categories of women workers (in personal and community services, in particular) have higher exposure than men to certain substances: cytostatic agents (used in chemotherapy treatment), formaldehyde and aromatic amines. Employees in very small businesses (fewer than 10 workers) have a higher exposure than those in firms with more than 500 workers.

About half of cases involve point exposures, but a quarter of cases involved more than 10 hours' exposure in the week preceding the survey.

Exposure intensity is usually measured not by sampling or analysis, but by assessment. While exposure intensity was judged low in 70% of cases, it was considered high or very high in 10% of cases (above the exposure limit value in 2%) and unknown in 18% of cases. Thirty-eight percent of workers have significant exposure to degradation and breakdown

products generated by the production process: smoke, dust, tar, diesel exhaust, crystalline silica derivatives, etc.

There is no collective protection in 35% of exposure situations. The most often cited forms of collective protection are extraction systems and general ventilation, but the latter cannot protect effectively against carcinogens. Closed systems are mentioned in only 1% of exposure situations. 57% of exposed employees in the building industry, and 37% in the maintenance sector, have no collective protection.

A comparison of the Sumer 2003 and Sumer 2010 data shows that the share of employees exposed to carcinogens in France has fallen from 13% to 10%. This decrease is partly due to a tightening-up of regulations which has improved awareness and encouraged prevention. Substitution seems to be more frequently used where the substances concerned are identified as carcinogens at the point of purchase. Where exposures result from the production process itself, the situation is probably more critical as is shown by the list of substances most frequently mentioned in the survey.

The Sumer findings also show that most of the reduction in exposed workers has been in big firms. In companies with more than 500 workers, the number of exposed workers is down 6% on 2003, compared to the very low percentage (less than 1%) in very small firms (fewer than 10 workers), pointing to the very poor risk control and the need to develop more systematic prevention in these companies. A lack of workers' health and safety representation may also be a contributory factor to poor prevention in these firms. Subcontracting is another issue to consider, as some of the progress made in large firms may be linked to the outsourcing of particularly hazardous activities, usually to small subcontractors.

Endocrine disruptors: a cocktail effect and questionable limit values

The Sumer survey shows that many workers are not just exposed to one specific chemical, but to several at once. Since the early 2000s, researchers have thought that research should be looking at mixtures of substances. Exposure to multiple chemicals present in the environment at very low doses, each individually below the dose considered safe or NOAEL (*no observable adverse effect level-dose*) could produce "cocktail effects" from additive or multiplicative doses.

The widespread theory that a mixture of substances is no more dangerous than the most toxic substance it contains, or that a mixture of substances each at less than the NOAEL is safe, has been questioned by some researchers. They argue that a NOAEL dose is not a "zero dose", i.e., each component of a mixture, however small its dose, contributes to the net effect. Even a very small dose does not mean no risk.

"Endocrine disruptors" (EDCs) are substances that interfere with hormones and can therefore disrupt the endocrine system which produces hormones. The endocrine system regulates many of the body's biological processes from the very moment of conception, including growth and reproductive function development. Researchers have shown that some of these substances, especially bisphenol A (see box p. 29), may have effects at very low doses, below the no observable adverse effect level-dose (NOAEL) fixed on the basis of "good laboratory practice" as defined by OECD (Organisation for Economic Cooperation and Development) criteria. OECD studies are very costly and usually financed by business. They are animal studies using high doses over a short period, whereas human exposure is often at low doses over very long periods. The potential low dose effects are derived from the observed high dose effects on the principle that "the dose makes the poison". The application of this principle to endocrine disruptors has been challenged, however, as academic

researchers and government laboratories have found endocrine disruptors having different and sometimes more acute effects at low doses than at higher doses¹⁵.

As well as their reproductive function effects (decreased sperm quality, birth defects, early puberty, etc.) EDCs, particularly in the case of embryo exposure, are suspected of causing various cancers later in life – testicular and prostate cancer, and especially breast cancer (European Environment Agency 2012). So parents' exposure to EDCs at work may have consequences for the health of their unborn children. This has been found in the children of farmers exposed to pesticides. The cancers concerned include reported cases of leukaemia, brain cancer, lymphoma, etc.

Sources of workplace exposure to endocrine disruptors abound as they are found in a wide range of products – pesticides, solvents, metals, medicines, etc. – across a broad swathe of industries, not least the pharmaceutical industry (hormone and corticosteroid production) and agriculture (pesticide handling) (Pilière 2002).

In 1999, the European Commission adopted a strategy for endocrine disruptors which included identifying substances capable of interfering with hormone systems. A list was finally established of 428 substances used in industry, agriculture and the production of consumer products, 194 of which are classed as category 1 – substances of highest concern¹⁶. Some chemicals known to be EDCs – like polychlorinated biphenyls (PCBs) and dioxins – are also known or probable carcinogens.

A report submitted to the European Commission in late 2011 recognises that identification of endocrine disruptors by OECD validated test methods captures only a limited range of their known effects (Kortenkamp *et al.* 2011) because these methods are not designed to study in detail the special “windows of vulnerability” that are pregnancy, infancy and puberty, or the mechanisms of chemical action. The report's authors argue that the exposure thresholds (“cut-off values”) currently defined for endocrine disruptors under the EU's CLP Regulation which lays down the rules for classification and labelling of chemicals, “are largely arbitrary and without scientific justification” and call for new methods for the evaluation of chemicals to be developed that take into account both their mode of action and their toxic effects.

In May 2013, 88 top scientists put their names to a declaration on endocrine disruptors, which was forwarded to the European Commission. They argued that the regulations were inadequate and urgently needed amending and that EU proposals “do not follow the best available science, and place commercial interests above the protection of human and wildlife health”¹⁷.

Trade unions also have concerns about endocrine disruptors. The social partner consultations undertaken for the revision of the Carcinogens and Mutagens Directive have seen the European Trade Union Confederation (ETUC) repeatedly call for the Directive to be extended to reprotoxicants.

Where prevention is concerned, the special case of endocrine disruptors should prompt workers to find out about all the chemicals they are exposed to, and list them regardless of their known level of toxicity or the level of exposure. They also need to remember that compliance with standards does not equal zero risk. The ALARA – “as low as reasonably practicable” – principle developed for exposure to ionising radiation should also apply to any exposure to chemicals.

15. Revue Prescrire (2013) Bisphénol A, un faisceau d'arguments pour réduire l'exposition, 33 (355), 375-380.

16. Revue Prescrire (2011) À la recherche des perturbateurs endocriniens, 31 (333), 541-542.

17. ChemSec (2013) Scientists express strong concern over EDCs and urge EU to act, press release, 24 May 2013.

Bisphenol A - also a problem for workers

Bisphenol A is a chemical produced in large volumes. Its hormonal and estrogenic properties have been known since 1936. It is classified by the EU as a substance suspected to be a reproductive toxicant due to its adverse effects on sexual function and fertility (category 2). It is mainly used in the manufacture of polycarbonates used in a wide range of applications, most notably the production of plastic containers (feeding bottles, other plastic bottles, containers) and epoxy resins used in many products (drinks and food can linings) for their corrosion protection and thermal stability properties. The bisphenol A present in containers made of polycarbonate or containing epoxy resins migrates into the contents, thus contaminating the entire population through food and drink.

It was banned in the EU for the manufacture of baby feeding bottles in 2011. At the end of 2012, a proposal to revise its classification was lodged with the European Chemicals Agency (ECHA). Studies in humans have shown that bisphenol A at doses sometimes well below the acceptable daily intake established by the European Food Safety Authority had adverse effects: cardiovascular disease, diabetes, reproductive health and child development problems. The same effects were observed

in animals along with lesions of the prostate gland and mammary gland, predisposing to cancer in adulthood following exposure in the womb.

Workers are exposed to bisphenol A across a range of industries. In the retail and distribution sector, checkout operators are exposed by repeated handling of thermal paper containing bisphenol A (till receipts and labels). This route of exposure was taken more seriously after 2010 when researchers in the United States found that the urine of shopworkers and checkout operators contained higher levels of bisphenol A than that of other workers or the general population (Lunders *et al.*). The European Food Safety Agency (EFSA) has since concluded that thermal paper was the second most important source of bisphenol A contamination of the general public after diet.

Several countries, including Japan, have already banned BPA in thermal paper. Others are preparing to do so. In October 2013, France asked the European Commission to follow suit.

Further reading

Revue Prescrire (2013) Bisphénol A : un faisceau d'arguments pour réduire l'exposition, 33 (355), 375-380.
Revue Prescrire (2013) Le bisphénol A imprègne toute la population, 33 (354), 294-300.

Chapter 4

European legislation

European laws on carcinogens can be classed in two groups: those on marketing of such substances, and those on protecting workers exposed to them. The legal bases underlying these two types of legislation are different: articles 94 and 95 of the EC Treaty for the former, article 137 for the latter. What this means in practise is that where placing carcinogens on the market is concerned, the Member States cannot, in theory, add extra restrictions on top of the rules laid down at Community level. This is known as full harmonisation. But where worker protection is concerned, the Member States can impose national rules that are more stringent than the European rules. This is known as minimum harmonisation.

Both types of legislation exist in parallel, and those who produce or use cancer-causing substances have to comply with the obligations arising under both.

The Carcinogens Directive

The Carcinogens Directive, the first version of which dates from 1990, lays down the Community rules for protecting workers from the risks related to exposure to carcinogens or mutagens at work. It is one of the first individual directives adopted under the 1989 Framework Directive on health and safety at work. It covers all chemicals that “meet the criteria for classification as a category 1 or 2 ‘carcinogen’ or ‘mutagen’”. This wording is important, because it covers not only substances

actually classified as category 1 or 2 in the Community legislation¹⁸, but also more broadly any substance or agent that meets these classification criteria.

This means that substances which for some reason have not been included in the Community classification, but are nevertheless known carcinogens, like crystalline silica, can be brought within the Directive's scope. The Directive also covers carcinogenic and mutagenic preparations and emissions that form in certain production processes listed in an Annex to the Directive. Unfortunately, although it should have been regularly updated to reflect advances in scientific knowledge, the Annex is limited to five processes (some of them historical or affecting only a tiny number of workers), and so takes no account of the risks faced by millions of workers across Europe like diesel particulate matter, the conversion of mineral oils used for machining and processing of metals, or leather dust.

The Directive, which has been carried over into the national law of all 28 EU countries, lays down an order of priority in employers' obligations to reduce the use of carcinogens in the workplace. First among these measures is the obligation to replace the carcinogen or mutagen by a substance which is not, or is less, dangerous. Where a safer alternative exists, the employer must use it instead, whatever the cost to the business. If replacement is not technically possible, the employer must ensure that the carcinogen or mutagen is manufactured or used in a closed system. If he cannot take this safety precaution, the employer must ensure that the level of exposure of workers is "reduced to as low a level as is technically possible".

The Directive also provides for occupational exposure limit values (OELVs) to be established. Unlike the OELVs adopted under the Chemicals Directive (98/24/EC), which are indicative and therefore leave each EU country free to set the value that will be implemented in national law, the limit values adopted under the Carcinogens Directive are binding. This means that EU countries have no choice but to apply at least the value set at EU level in their workplaces.

Since the Carcinogens Directive was adopted in 1990, only three substances (benzene, vinyl chloride monomer and hardwood dusts) have been assigned a binding OELV, whereas indicative OELVs have been adopted for 122 substances under the Chemicals Directive. In practice, each EU Member State has the (indicative or binding) OELVs defined by the EU and implemented into its laws plus national OELVs for many other substances (including carcinogens) adopted in accordance with its own specific rules¹⁹.

This EU law also requires employers to inform their workers about the health risks of substances in the workplace and provide them with training to reduce these risks to a minimum.

Workers are not all equally protected against carcinogens. It depends on the country they work in. The occupational exposure limit value (OELV) for crystalline silica, for instance, ranges from 50 mcg/m³ in Italy, to 75 mcg/m³ in the Netherlands, 100 mcg/m³ in Sweden and up to 300 mcg/m³ in Poland. These wide variations stem from the differing methods and practices used by countries to define and/or review existing national OELVs. The only way to protect workers better against cancer risks, therefore, is by revising the Carcinogens Directive to lengthen the list of chemicals with a binding OELV (preferably harmonized upwards) at EU level.

¹⁸. Only substances that are marketed have a classification and labelling harmonised by the EU. They are listed in Annex VI of the CLP Regulation (Regulation (EC) No 1271/2008) which came into force on 20 January 2009.

¹⁹. See <https://osha.europa.eu/en/topics/ds/oel>

Revision of the Directive and tentative stocktaking

Directive 2004/37/EC is the codified version of the original Directive (90/394/EEC) which it repealed along with all its successive amendments (Directives 97/42/EC and 1999/38/EC). It makes no substantive changes, but merely consolidates the pieces of legislation it replaces.

The European Commission proposed revising the Carcinogens Directive in the early 2000s to adapt it to changes in scientific knowledge, technical progress and the world of work. The revision process got formally under way in 2004 with the first of the two rounds of social partner consultations organised by the Commission in line with the European treaty requirements for changing or introducing laws that affect the protection of workers.

The two Commissions headed by José Manuel Barroso have raised one obstacle after another to fend off a revision of the Directive, despite the urgings of the European Parliament and the points of agreement reached by the unions, employers and governments. On 2 October 2013 – almost ten years since work on revising the Directive got under way – the Barroso Commission made the bald announcement that it would not be coming forward with proposals to improve the legislative framework for the prevention of occupational cancers. This setback gives an idea of the interests at play. The two main stumbling blocks between employers and unions are extending the Directive's scope to reprotoxins and setting binding occupational exposure limit values (OELVs) for a bigger number of carcinogens.

While the social partners are still at odds over the first point, they managed to agree in 2012 and 2013 on adopting binding OELVs for over a score of additional carcinogens including respirable crystalline silica, refractory ceramic fibres, particles emitted by diesel engines, hexavalent chromium and trichloroethylene. These advances, however, are unlikely to be endorsed by EU lawmakers before 2015 or 2016.

Assessing the impacts of European legislation on workplaces is a dicey operation. The difficulty is less purely legal than political. Most Member States and the European Commission visibly lack the political will to process the relevant information.

The Directive provides that the information collected in companies must be supplied to national authorities on their request. In practice, only a minority of Member States have reliable information on occupational exposure to carcinogens and the preventive measures implemented in workplaces. Unlike with most HSW Directives, States originally had no obligation to report to the Commission on the implementation of the Carcinogens Directive. That reporting obligation has now existed since 2007, but the first reports planned for 2013 have not yet been made public. And while the Commission did order a study on the application of the Directive, it has never released it.

From the available data, it can be suggested that wide disparities exist between EU states. Firms in states with a firmly established tradition of chemical safety seem to achieve better compliance with the Directive's requirements. The political upset of the asbestos scandal has also played an important role. Sadly, its impact has differed widely from one country to another. So, while national differences must be emphasised, actual enforcement of the law seems to vary more with the sector and type and size of undertaking. Broadly-speaking, big pharmaceutical and chemical companies, machinery manufacturers, hospitals and research laboratories have better safety records than small firms in the leather, furniture-making, waste collection and recycling sectors. The situation in the building industry is worrying.

The failings in employee information and training in the specific risks of carcinogens, and the trend towards outsourcing hazardous work – more and more often to migrant workers – add to the “invisibility” of these substances. But tackling occupational cancers is a major public health challenge that will not easily be addressed by sidelining the issue.

Implementation of the REACH regulation could drive the spread of practices that will really prevent the risks of carcinogens in workplaces of all sizes.

Current health surveillance of workers under the EU Directive is badly lacking, being required only during the period for which they are exposed. But cancer may not develop until a very long time after exposure, which makes it vital for workers who have been exposed to a carcinogen to continue getting health surveillance throughout their lives. Early detection of tumours is key to surviving most cancers.

REACH, the new EU chemicals legislation

After several years' fierce debates and lobbying, the reform of European legislation on chemicals use and marketing, known as REACH (Registration, Evaluation and Authorisation of Chemicals), was finally adopted by the EU in December 2006. The regulation comes into effect in the 27 EU countries on 1 June 2007, and will replace the jumble of close to 40 existing pieces of legislation that were seen as no longer capable of effectively protecting human health and the environment from chemical hazards.

REACH requires chemicals manufacturers and importers to prove that the risks related to the use of their substances can be controlled before they are allowed to market them. They must do this by drawing up a registration dossier. Chemicals that are produced or imported in volumes of more than 1 tonne a year onto Community territory – approximately 30 000 substances – will have to be registered over an 11-year period up to 2018 with the European Chemicals Agency based in Helsinki.

REACH is huge in scope, therefore, and can be considered as the main piece of EU legislation for regulating most uses of chemicals and their marketing. But some uses of chemicals fall outside REACH because they fall within other specific EU laws which cover closely-defined areas of use (see box p. 37).

REACH and carcinogens

Registration rules for manufacture or import

In order to continue being manufactured or imported in the EU in quantities above 1 tonne a year, a class 1A or 1B carcinogen, mutagen or repro- toxin (CMR) will have to be accompanied by a registration dossier giving information on its properties, uses and classification, plus guidance on how to use it safely. For chemicals produced in quantities of 10 tonnes a year and more, the registration dossier will also have to include a chemical safety report describing the risk management measures necessary for adequate control for each identified use of the substance. This means that it will no longer be permitted to manufacture or import a CMR substance in Europe without a registration dossier, except in quantities of less than 1 tonne a year.

Authorised use rules

As well as registering with ECHA, manufacturers will have to get European Commission authorisation for each proposed use of those CMRs included in Annex XIV of REACH (the authorisation list). To get authorisation, applicants will have to show that the risks associated with the use of the chemical concerned are “adequately controlled”. Even if they

are not, authorisation may still be granted if it is shown that the risks are outweighed by socio-economic benefits and there are no suitable alternative substances or technologies. Authorisations will be granted in 2014 for a specific period on a case-by-case basis. In theory, authorisation must be sought for all REACH-registered category 1A and 1B CMRs regardless of the quantities they are produced in. In practice, a prioritising system has been implemented because the European Chemicals Agency cannot process more than twenty-odd applications for authorisation a year. At the start of 2014, Annex XIV contained only 22 substances, selected as priorities because of the large quantities they are produced in²⁰. This means that many CMRs (especially those produced in small quantities) will continue to be used while waiting to be included in the REACH authorisation list. In early 2014, upwards of 1 100 chemicals had a harmonised classification as category 1A or 1B CMRs in EU law.

Restriction rules

As well as the registration and authorisation system, REACH also includes a system of restrictions. This means that the marketing or use of particular hazardous substances may be prohibited or allowed only subject to conditions if the Commission considers that there are unacceptable risks to human health or the environment. This is the case for category 1A or 1B CMRs which can be used in the workplace but cannot be used as substances or in mixtures intended for sale to the general public. This general prohibition does not, however, apply to cosmetics, medical or veterinary drugs (see box, p. 37) or fuels.

An EU-wide system for restricting the use or marketing of a series of hazardous substances had existed since 1976, long before the REACH reform was adopted. All the restrictions brought in before REACH (prohibition of asbestos, heavy metals in batteries, etc.) obviously remain in place and are added to Annex XVII with those introduced since the REACH Regulation came into force. Sad to say, however, since REACH's entry into force, the list of restricted substances has been updated much more slowly than before. Under the old system, 59 entries were added to the EU list of prohibitions in 33 years, compared to four new entries in four years under REACH.

Classification, labelling and packaging rules

Like all substances classified as hazardous under EU law (CLP (Classification, Labelling and Packaging) Regulation No. 1271/2008), category 1A, 1B or 2 CMRs must be labelled in the regulation manner. They must carry a hazard pictogram and hazard statement (see box p. 36). As a general rule, manufacturers or importers are responsible for classifying and labelling their own substances (self-classification) unless it has a harmonised classification at Community level, in which case the supplier must apply that. Under the CLP Regulation, manufacturers had to notify ECHA before the end of November 2010 of the classification and labelling of all their marketed substances that were classified as hazardous regardless of what quantities they were produced in. ECHA has compiled this information into a regularly updated inventory which is publicly available on its website²¹.

20. <http://echa.europa.eu/addressing-chemicals-of-concern/authorisation/recommendation-for-inclusion-in-the-authorisation-list/authorisation-list>

21. <http://echa.europa.eu/web/guest/information-on-chemicals/cl-inventory-database>

This inventory should make it possible to identify where different classifications have been given to the same substance and encourage its different manufacturers to agree on a single classification.

According to this inventory, over 3 700 category 1A or 1B CMRs are present on the European market (about 1 100 of which have a harmonised EU classification).

The EU's three categories of carcinogens

Category 1A: substances known to be carcinogenic to man. There is sufficient evidence to establish a causal association between human exposure to a substance and the development of cancer.

Category 1B: substances which should be regarded as if they are carcinogenic to man. There is sufficient evidence to provide a strong presumption that human exposure to a substance may result in the development of cancer, generally on the basis of appropriate long-term animal studies and/or other relevant information.

Category 2: substances which cause concern for man owing to possible carcinogenic effects but in respect of which the available information is not adequate for making a satisfactory assessment. There is some evidence from appropriate animal studies, but this is insufficient to place the substance in category 2.

The International Agency for Research on Cancer (IARC) – a WHO agency – has also established its own list of carcinogens (see Chapter 3).

While the IARC and EU classifications broadly overlap, they are not identical on all points. For example, diesel engine exhausts are classified as Group 2A by the IARC, but are not included on the EU list. As was seen in the previous chapter, crystalline silica was classified as a Group 1 carcinogen by the IARC in 1996, but by early 2014 had still not been classified as a hazardous substance by the EU. This therefore makes it advisable to refer to both classifications as using the EU classification alone would significantly underestimate the percentage of workers exposed to carcinogenic chemicals. France's

SUMER survey shows that of the 10% of exposed French workers identified by occupational doctors in 2010, only 4.3% were exposed to substances classified as EU category 1A or 1B carcinogens.

Unlike the IARC list, the European classification is part of a regulatory framework to govern the marketing of chemicals. This framework is vital to prevention but not enough: exposures to carcinogens can come about from the processing of a substance that is not itself a carcinogen, or from a reaction between two non-carcinogenic substances used in the production process.

Labelling of carcinogens and mutagens under the CLP Regulation

Hazard codes and statements to be used



Categories 1A or 1B

- *Carcinogens* classified in category 1A or 1B must be labelled with the pictogram shown above, the code "H350" and the hazard statement "May cause cancer".
- *Mutagens* classified in category 1A or 1B must be labelled with the same pictogram, but with the code "H340" and the hazard statement "May cause genetic defects".

Category 2

- *Carcinogens* classified in category 2 must be labelled with the same pictogram, but with the code "H351" and the hazard statement "Suspected of causing cancer".
- *Mutagens* classified in category 2 must be labelled with the same pictogram, but with the code "H341" and the hazard statement "Suspected of causing genetic defects".

How the Carcinogens Directive and REACH interface

The different business concerns with obligations under REACH – e.g., manufacturers and importers – are often also employers. So they have to meet both their REACH obligations and those laid down in the worker protection legislation.

If a carcinogen has to be used at a workplace, the employer must as a rule first apply the order of priority of obligations laid down in the Carcinogens Directive (elimination, replacement, control) before using it. Employers who then go on to use such carcinogens must abide by the rules laid down for authorisation under REACH.

The obligation to get authorisation for carcinogens included in Annex XIV of REACH should encourage producers to replace them by less hazardous alternatives, which will promote implementation of the substitution principle which is mandatory in the Carcinogens Directive. But the authorisation procedure finally adopted in REACH will allow some carcinogens to be authorised for use even though a safer alternative exists²². This will then create the perverse situation of a clash between the two pieces of legislation, with one authorising the use of a carcinogen, and the other requiring it to be replaced by the safer available alternative. When REACH comes in, the trade unions will need to ensure that the different actors – European Commission, social partners, business, etc. – implement the regulation with the guiding principles and spirit of worker protection legislation intact. Otherwise, which of the two pieces of legislation prevails could well end up being a matter for the courts.

Other specific EU laws

The use of carcinogens in cosmetics, biocides, pesticides and medicines (areas not covered by REACH) is generally prohibited. However, looked at closely, each of the laws governing these specific uses of chemicals contain derogations that are likely to be instrumental in workers and consumers being exposed to carcinogens.

The Cosmetic Products Regulation

Most of the provisions of the European Regulation on cosmetic products (1223/2009) have applied since July 2013. It contains a list of colorants, preservatives and UV filters that can be used in cosmetic products. The Regulation's Annexes also contain a list of substances whose use is prohibited (Annex II) or restricted (Annex III). The Regulation prohibits the use of substances classified as CMR (category 1A or 1B) apart from in exceptional cases. For a CMR substance to be allowed to be used in a cosmetic product, it must comply with food

safety requirements (e.g., occur naturally in food), no suitable alternative substances must exist, and its use as a cosmetic must have been found safe by a Commission scientific committee. This is the case for formaldehyde, which may be used up to a certain concentration in hair products.

The Biocidal Products Regulation

The Biocidal Products Regulation (BPR Regulation 528/2012) is ECHA's responsibility. It concerns the placing on the market and use of biocidal products, which are used to protect humans, animals, materials or articles against harmful organisms (fungi, bacteria, viruses, rodents). Biocides are to be distinguished from pesticides which are mainly used in agriculture. All biocidal products must obtain an authorisation before being marketed and the active substance they contain must be EU-approved. To date, there are 49 substances on the approved active substances list. In principle, CMR substances classified 1A or 1B cannot be included on

22. Such as carcinogens for which it can be shown that there is an exposure threshold below which there is no demonstrable adverse effect on human health.

this list, but derogations are foreseen where the active substance is needed on grounds of public interest and when no alternatives are available. This is the case for five active substances used for wood preservatives or for the control of rodents, for example.

The Pesticides Regulation

A vast body of European legislation overseen by the European Food Safety Authority (EFSA) regulates the marketing and use of plant protection products (commonly known as pesticides) and their residues in food. The main law is Regulation 1107/2009 concerning the placing on the market of pesticides, which cannot be used or placed on the market without prior authorisation. A two-stage system is in place by which the European Community evaluates active substances used in plant protection products and the Member States evaluate and authorise plant protection products themselves at the national level. Active substances classified as category 1A or 1B carcinogens are not authorised unless the exposure of humans to that active substance in a plant protection product under realistic proposed conditions of use is negligible. Member States can also grant a derogation for the use

of an unauthorised pesticide for up to 120 days where necessary because of a danger which cannot be contained by any other reasonable means. Environmental NGOs regularly allege misuse of this derogation option by some Member States. For example, dichloropropene (a genotoxic carcinogen) is claimed to be in continued illegal use in Europe in large quantities.

EU medicines legislation

The regulatory framework for medicinal products for human use is managed by the European Medicines Agency (EMA). It is based on the principle that manufacturers must obtain marketing authorisation from the competent authorities. The main marketing authorisation requirements and procedures, and the rules on the constant safety-monitoring of authorised medicines, are set in the Directive on medicinal products for human use (2001/83/EC) and the Regulation laying down procedures for the authorisation and supervision of medicinal products for human and veterinary use (726/2004). The use of carcinogens in medicines is avoided as far as possible, but is possible if the therapeutic benefits outweigh the risks of developing cancer.

Chapter 5

Cancer is also a power issue for the unions

At first glance, cancer touches the innermost privacy of the individual. It is a condition that people are not naturally forthcoming about. Sufferers undergo an experience which in some ways cuts them off from the world. Physical pain, mental distress, the feeling of being betrayed by one's own body where vital cell regeneration processes are warped into health-destroying ones. The way our societies see cancer adds to this isolation.

It can be put down to modern forms of predestination – faulty DNA or personal fault – what are too readily accused of being unhealthy lifestyle choices. It is not easy to develop a strategy for collective defence. But nor is it impossible, as feminist lobbying on breast cancer, the opposition to nuclear weapons mounted by the Hibakusha, the Hiroshima and Nagasaki atom bomb survivors, and the exemplary fight by asbestos victims worldwide show. Each of these experiences showed how direct engagement by victims could act as the binder for collective action.

In acting on working conditions that create a cancer risk, the trade union movement has a big job on its hands.

Obstacles include:

- the lack of public attention for the role played by working conditions in cancer. From deliberately organised industry manipulation to the comparative lack of interest from large swathes of medical research, a wide array of factors contribute to a lack of knowledge and social visibility;
- the trade union movement is fixated on immediate action to improve working conditions. Generally, there are long latency times between work exposure and the development of cancer. In most cases, the victims are no longer working for the same company. This makes it harder to establish the linkage between working conditions and cancer;

— acting against cancers requires the ability to examine critically all the technical choices that make up a production system. This is no easy matter. One way or another, workers come to identify with their work. This makes it hard to stand back from it and visualize alternatives. This is compounded by an ever-present blackmail: employers have consistently responded to demands to eliminate carcinogens with threatened job losses. The union movement is also susceptible to the dominant ideologies of the society it operates in, often partaking of a belief in high productivity that imbues economic growth with virtues that it does not possess.

This chapter does not cover all the problems that trade union action faces. It merely offers some ways forward and raises questions to set a wider-ranging debate rolling.

Why take a stand against working conditions-related cancers?

We saw in preceding chapters that cancers are behind wide social inequalities of health. In the same way, the unequal distribution of cancers reflects conditions of exploitation and domination. It goes with and worsens other inequalities in the distribution of wealth, access to knowledge and information, and empowerment. This by itself is grounds for trade union action. But two other things must also be said. Working conditions are a big factor in these social inequalities in cancer.

Directly, through the large-scale exposure of workers to cancer-causing chemicals; indirectly, through production and technology choices that put large volumes of carcinogens on the market. This latter fact means that trade union strategy must not only be about addressing exposures to carcinogens at work, but also preventing exposures at home and in the environment. The issue of asbestos illustrates this relationship very clearly. With asbestos production totalling over 170 million tonnes throughout the 20th century, tens of millions of workers were directly exposed at various stages: extraction, manufacture, use or processing of asbestos-containing products, and destruction or recycling.

Hundreds of millions of people were also affected by exposures in their workplaces, homes, schools, etc. The trade union movement's fight against cancers can form part of a strategic alliance with environmental protection groups, public health agencies and other actors concerned to improve cancer prevention.

Public health policies on cancer disregard working conditions and production processes. They tend to consider workplaces as “private spheres”, and the indisputable preserve of employers. They are reluctant to call into question trade secrets and the marketing of hazardous products.

Only exceptionally do they ban particularly dangerous substances and harmful production processes. Notwithstanding the public pronouncements, and probably even against the agenda, of a large section of those that run them, these public health policies remain generally powerless to tackle the growing social inequalities in health.

Tackling cancers in workplaces

The evidence is that preventing exposure to carcinogens is seldom a priority for company management. The effect of exposures is seen only after a fairly long latency period, when the victims are usually no longer working for the company. There is therefore no direct economic gain for the employer in implementing a prevention policy. This is particularly

so for sectors that are bulk chemicals users, like the construction, cleaning and textile industries. This makes worker participation in setting prevention priorities a key factor. There are many barriers to such participation, not least among the workers. Exposure to cancer-causing factors is often not seen as an immediate risk. In many cases, health damage will be detected only years afterwards, and the linkage between the ill-health and working conditions will not be clearly established. Tackling work related cancers therefore requires trade unions to work in a systematic and organised way to develop collective awareness and action.

Mapping the lie of the land

The union will usually start by doing a survey, either because exposure to carcinogens is uncharted territory, or because management downplays it. The survey should aim to identify all possible points of exposure in a particular production cycle. It is hard to cover all situations in one go, so it can be helpful to start the survey by looking at a specific problem, and then widen it to other situations afterwards.

The trade union survey is a form of risk assessment which must be actively supported by the workers themselves in order to succeed. At the same time, management and preventive services must also be required to do their part. At the survey stage, that means they must come up with all necessary information on carcinogens and include them in their own risk assessment. Special care must be taken to see that the company has the safety data sheets for all chemicals used and that its preventive services report regularly to workers' reps on the measures taken to prevent occupational cancers. If there is no safety data sheet available for a chemical in use, the health and safety or labour inspectorate must be called in immediately to put a stop to the breach. If the use is creating a serious danger, there should be no hesitation in downing tools until a solution is found.

But it would be over-trusting to rely on this source of information alone. So the union has a vested interest in having its own sources of expertise. It may be "in-house" to the union, such as by enlisting the experience developed in other workplaces. Or it may be external, drawing on the labour inspectorate, scientists with trade union connections, preventive services (if operating as they should), etc. Workers may know little about toxicology, but they have vast knowledge when it comes to analysing working conditions, checking how work is actually done under conditions that minimise the risks of exposure. The finding in many companies is that jobs in which carcinogens are used or produced are poorly isolated from other jobs, that product transport, handling or cleaning operations can endanger the health of workers doing other jobs, and that pressure of work leaves too little time to deal with unexpected occurrences or exchange information and advice. All these aspects of work organisation are crucially important to preventing occupational cancers.

Checking the validity of the information supplied by company management is a key aspect of the trade union risk assessment, which should aim to identify all the physical and non-physical contributory factors to the development of cancers. It should also audit the practical working conditions of exposed workers, and critically assess the prevention policies pursued (or lack of prevention). Other relevant steps include assessing management and the preventive services' attitudes, and identifying what may work for and against creating a bargaining position. The appendix on p. 79 shows the key aspects to be considered.

The risk assessment is never done just for its own sake. It is only the first stage of an action that is intended to change working conditions. The union assessment is therefore logically carried through into two additional things: a trade union action plan, and negotiating a carcinogen exposure prevention plan with company management.

RISCTOX: a trade union database on hazardous substances

RISCTOX aims to provide clear and concise information on hazardous chemicals that workers may be exposed to in the workplace. The database was developed by Spanish trade unions in association with the ETUI, and provides information on over 100 000 chemical

substances including: classification of the substance under the EU's CLP Regulation, the specific risks to human health and the environment, as well as other European laws and regulations governing these substances. It can be searched by substance name or any of its identification numbers (CAS, EC, EINECS/ELINCS). The database is updated regularly, and is available on www.istas.net/risctox/en

Change working conditions: substitution is the non-negotiable priority

The workers' safety reps can use the trade union assessment to call the company to account. The idea is to check whether management's risk assessment is comprehensive and detailed, whether it results in a prevention plan, whether that plan follows the priorities for effective preventive activity, and whether it is put into effect with sufficient resources.

The top priority is to eliminate a carcinogen from the workplace whenever it is technically possible to do so. The concept of technical possibility is important and usually a source of conflict.

Many arguments are ranged against substitution, and it is important to knock them back:

1. **The technical argument.** Many heads of firms that use dangerous substances have only limited technical knowledge. For example, if their workers use trichloroethylene to clean metal parts, they see that as the only possible technical solution. It can be helpful to collect information on substitution practices to show that there are alternatives to using dangerous chemicals. In some cases, the carcinogen forms part of the end product, like the asbestos in asbestos cement, formaldehyde in insulating foams or furniture, for example. This will mean raising the question of an alternative production method. Other products with similar technical characteristics can generally substitute for carcinogen-containing products.
2. **The cost argument.** The prospective cost of substitution is often cited as a barrier. In some instances, the cost is heavily inflated. In others, it may be real. It is important not to give in to blackmail and to emphasise that not substituting puts human lives at risk.
3. **The controlled risk argument.** Company management will often claim that preventive measures are adequate, such that there is no need to go to the "extreme" of substitution. Regardless of how good the preventive measures may be, experience tells us that there are always critical points at which these measures fail. This may be one of the main lessons to be learned from the idea of "controlled asbestos use". Critical points may be connected with abnormal situations, such as a chemical leakage from a closed system, a fire, etc. They may be upstream (mining or primary manufacture, transport, storage, inputs to the production chain) or downstream (subsequent processing of the product, whether or not planned, deterioration or destruction, waste recycling or processing, etc.). This product lifecycle overview is key to

an effective cancer prevention policy. It joins up protection of health at work with protection of public health and the environment. It puts a workable gloss on two fundamental principles of union action: solidarity (the bottom line for us is to eliminate the risks for all workers potentially affected, whether employed in the workplace or elsewhere) and equality (we are fighting for better living and working conditions for everyone in society, which involves tackling the harm that a given product can do to public health and environment).

You should go on the attack where substitution is concerned. It is the top priority. It is also a legal obligation on the employer. If the situation cannot be resolved, you should not hesitate to call in the labour/health and safety inspectorate or exercise your right to stop work where a serious and imminent danger exists. It is not up to workers' reps to prove that substitution is possible and say exactly how it can be done. Company management must face up to its responsibilities, and is for them to prove that substitution is technically impossible.

Market rules are only a bottom limit. It is obviously illegal to use a product which has been banned from being marketed, like asbestos or some aromatic amines. But even if a carcinogen is allowed to be marketed, it is just as illegal to use it if there is an alternative. It is reasonable to infer that once REACH is implemented, the number of carcinogens put onto the market will gradually fall. But it is equally certain that some of these chemicals will continue to be produced and marketed. The important thing here is to avoid them being used at workplaces. That can be achieved through company prevention plans, but also through industry collective bargaining or national bans on their use in workplaces in each EU Member State.

When identifying substances of particular concern that have to be substituted, it is not enough to look only at those classified as 1A or 1B under the EU rules as these are not really concerned with prevention in the workplace: they are commercially-oriented, and about providing information for marketing purposes. Also, the classification process is slow and influenced by industry lobbies. This is why crystalline silica is not classified and formaldehyde is improperly classified, for example, even though on the basis of scientific evidence they should be recognised as known human carcinogens. There is still no harmonised classification for many substances: this means that manufacturers choose the risk category for the substance they are placing on the market.

Effective prevention needs to do at least three things:

1. Include substances classified as category 2 (see page 36) in the list of substances of very high concern regarding the potential exposure of workers.
2. Substitute, and where this is not technically possible, minimise exposures to the endocrine disruptors that are implicated in a number of cancers.
3. Take into account interactions between two substances which, though not carcinogens themselves, can react with each other to generate a carcinogen. The dyeing and printing industries, for example, use azo compounds which are now classed as non-carcinogenic. But carcinogenic amines may be formed during the use of bleaching soaps for hand washing: the soap on the azo dye creates a carcinogenic amine which is absorbed through the skin because it is fat soluble.

Where substitution is technically impossible, any risk of exposure must be eliminated by collective protective measures. The priority then must be to have production carried out in a closed system. If that is not possible, preventive measures must be taken to reduce exposures to the lowest levels technically possible.

**Bladder cancer among airport workers:
union campaign to cut pollution**

A group of European trade unions has been campaigning since 2009 to minimise the exposure of airport workers to fine particles. The "Clean Air" campaign links together the European Transport Workers' Federation (ETF), the British-Irish union Unite, the Swedish Transport Workers' Union and the Danish union 3F which set the campaign going.

In 2008, a case of bladder cancer in a baggage handler set alarm bells ringing for 3F's Kastrup branch – the Copenhagen suburb where the national airport is located. The Danish National Occupational Diseases Board was quick to recognise the link between this cancer and the worker's exposure to air pollution at the airport. 3F tackled airport management on it and the two sides agreed to measure air pollution on the airport tarmac. A series of readings would be taken by specialists.

In 2011, a report from the Danish Centre for Environment and Energy confirmed the suspicions. The readings found a concentration of fine particles at the airport three times higher than on Copenhagen's busiest main road in rush hour. The report estimates that airport baggage handlers breathe in up to 50 times more ultrafine particles than an office worker. Since 2008, two other cases of bladder cancer among airport workers

have been recognised by the occupational disease compensation system.

There are an estimated 1 million airport workers across Europe, about 20% of whom are exposed to massive air pollution due to working on the tarmac near aircraft and the different diesel-powered vehicles used to refuel, unload or push them to the hangars.

To reduce pollution levels around runways, the Clean Air Campaign recommends turning off the engines of handling equipment when not working and replacing diesel-powered by electric-powered handling machinery or fitting them with more recent diesel engines, fitting upgraded particle filters to snow removal vehicles, using electric towing vehicles to move the aircraft onto runways or, if that is not possible, using only one of the aircraft's two main engines once it is on the tarmac.

Knowing this to be a Europe-wide problem, the campaign organizers are trying to extend the Copenhagen experiment to other European airports. Clean Air has received financial support from the European Union's Social Fund to explore the scope for European-level social dialogue on the issue. A party of MEPs visited Copenhagen Airport in June 2012, and a conference was held in the European Parliament in January 2013.

Read more: <http://www.project-cleanair.eu>

Two surveillance measures must be carried out systematically to check whether preventive measures are effective:

1. Surveillance of exposures, paying particular attention to the most critical points in the production cycle. This requires intervention by competent and professionally independent preventive services, and oversight of their activity by the workers' safety reps. Meeting exposure limits is the rock-bottom minimum required. Whenever it is technically possible to reduce exposures below these limits, it must be done.
2. Surveillance of workers' health by occupational doctors. The health surveillance arrangements must be spelled out in detail. Health surveillance too often stops short at a general check-up or tests not directly connected with working conditions. Health surveillance must never be turned into a means of employee selection. This is why trade unions want employment-related genetic screening made illegal. Workers who have been exposed to carcinogens must continue to get health surveillance even after the exposure is at an end. Almost no EU country has organised post-exposure health surveillance.

The results of health and exposure surveillance must be supplied to the workers' safety reps. Anonymity of health surveillance information must be guaranteed. This information

can help shed light on work-related health problems and improve prevention plans. The data must be preserved and used in a broader framework than the workplace (sectoral or national) if a public policy of prevention of work-related cancers is to be pursued. Each exposed worker must be able to keep track of their exposures and the results of the health checks. It is also important to check the quality of the information and training for workers.

If personal protective equipment (PPE) does have to be used, two questions must be asked and answered:

1. How efficient is this equipment, really? It must be assessed in light of workplace realities – what is sometimes called the ergotoxicology approach – and not make do with standardised tests.
2. Do the working conditions need to be adapted to take account of the constraints inherent in wearing certain equipment? Do regular breaks need to be provided where equipment is burdensome? Use of PPE can never be used as an excuse for putting off or not bringing in more effective preventive measures (substitution, collective prevention).

Intervention in workplaces can be fully effective only if combined with action in broader society. Workplace exposure to carcinogens is also a major public health issue.

The trade union movement can act on several fronts here:

1. For a more effective public policy on health and safety at work. Workplace prevention depends very much on whether there is a public policy on health and safety at work. Producing detailed, independent information on chemicals, carrying out toxicological and epidemiological research, and implementing policing and enforcement systems obviously go beyond the capacity of a single company.
2. For a public health policy that incorporates working conditions. Public health policies in most EU countries do not at present act on working conditions, and have little effect on social inequalities of health.
3. To put work-related cancers in the public spotlight and labour action to put them at the top of the political agenda. Asbestos showed how far prevention depended on putting work-related health damage in the public arena. It was the result both of work done day-to-day by unions and labour action on specific issues. No avenue must be left unexplored: trade union press, mass media, lawsuits, calling political authorities to account, etc.
4. From workplaces out to society: the trade union contribution to environmental protection. Preventing cancers is a litmus test for imposing democratic control on production choices. Profit maximisation and meeting human needs, including that of preserving our ecosystem, are irreconcilable opposites. By increasing workers' control over their working conditions, the trade unions can also move towards social control of production, and thereby reduce the harm it causes.

Working with cancer

For most people battling cancer – both those undergoing treatment and survivors – the trial of the illness is made worse by losing their job or taking a cut in job quality. A 2011 survey by the Curie Institute in France found that half of returners have problems from the consequences of the illness (fatigue, pain, anxiety, etc.) but also from being penalised in terms of working conditions and the stigma attaching to the disease.

The difficulty of adapting working conditions to the situation resulting from the disease causes direct job discrimination. Chemotherapy often involves alternating periods of extreme fatigue when work is impossible and spells of relative normalcy. Many women operated on for breast cancer find repetitive arm movements acutely painful. This makes it virtually impossible to continue working on an assembly line or supermarket check-out. Physical challenges are often made worse by prejudices and rejection that can lead to isolation. A former patient says, "The day I started back, when I got in, I was greeted with: What the hell are you doing here?"

The Share European survey suggests that cancer reduces men's employment rate from 63% to 43%, and women's from 43% to 34%.

A French survey has found that among people aged 57 or under, 83% were in employment when their cancer was diagnosed. Two years later, that rate was down to 59% of men and 56% of women. The difference is not explained by sick leave, taken by 14% of men and 11% of women. The percentage of registered unemployed had increased by 60% while that of "other reasons for not working" had doubled. Inequalities within the workforce are very wide. Two years after being diagnosed with cancer, barely 45% of farmers and 54% of manual workers were still in work compared to 73% of craftworkers and independent retailers and 74% of associate professionals.

Trade unions must provide support to these workers and keep up pressure in workplaces for adapted jobs to ensure that they can stay working.

Chapter 6

Under-estimating and under-reporting occupational cancers

Epidemiological studies done in the decades following World War Two demonstrated the cancer-causing effects of several substances used on a large scale in industry: aromatic amines, asbestos, benzene, vinyl chloride, wood dust, and so on. To address the concerns raised, work was done to determine what percentage of cancer cases were linked to occupational exposure. Because, as the title of an English publication (Rushton *et al.* 2008) points out, estimating the scale of work-induced cancers is the first step towards preventing them.

Percentage wars

The first large-scale study, long taken as gospel in the matter, was done in the United States by two English epidemiologists, Richard Doll and Julian Peto, whose findings were presented to the US Congress in 1981 (Doll and Peto 1981).

Doll and Peto argued that 4% of all cancers could be regarded as work-related (8% in men, 1% in women). This figure of 4% seems on the low side compared to the large number of workers exposed to carcinogens, and has often been used to play down the impact of occupational causes in the development of cancers. In 1998, Samuel Epstein, Professor at the University of Illinois School of Public Health, highlighted a number of flaws in Doll and Peto's estimates of occupational cancers. He singled out the failure to consider the multifactorial nature of cancer and synergies between multiple carcinogens, as well as the failure to allow for the increased number of carcinogens in the working environment.

Legitimate questions are now being asked about the conflicting interests the British epidemiologists may have had, in light of the revelations made in an article

published in the November 2006 issue of the *American Journal of Industrial Medicine* bringing evidence of the financial links between Richard Doll and the chemical industry multinationals Monsanto, ICI and Dow (Hardell *et al.* 2006).

Doll and Peto argued that, over and above the overall figure of 4%, the fraction of cancers attributable to a work-related cause varies by sex and type of cancer. Among men, therefore, Doll and Peto estimated that 25% of sinus cancers, 15% of lung cancers, 10% of bladder cancers and 10% of leukaemias could be put down to work factors, falling to 5% for the same type of cancers among women.

Very comprehensive cancer mortality estimates published in 2001 by a Finnish team produced figures higher than Doll and Peto's. The Finnish researchers claimed that the share of occupational cancers among all cancers was as high as 8% (14% for men and 2% for women), and that in the male population, 29% of lung cancers, 18% of leukaemias, 14% of bladder cancers and 12% of pancreatic cancers were arguably work-related (Nurminen and Karjalainen 2001).

Behind the percentages lie a number of workers which in the Finnish study may range up to double those estimated by Doll and Peto. The number dying of occupational cancer each year in the United Kingdom is estimated at between 6 000 and 12 000, and the annual number of new work-related cancers between 12 000 and 24 000²³. In Spain, annual deaths from occupational cancers could range from 4 000 to 8 000, and the number of new cases of work-related cancers from 6 500 to 13 000 (Kogevinas *et al.* 2005).

Going beyond the "attributable fraction"

Whatever the "estimate wars", it has to be said that in the past thirty years the fraction of cancers attributable to working conditions has been regularly revised upwards. The traditional epidemiological approach by "attributable fractions", however, must be viewed with caution on several counts.

The data on women's work is patchy. Epidemiological study has neglected female-dominated occupations and sectors, and the most common female cancers. Breast cancer, the leading cause of cancer death among women, has received far less study in terms of occupational hazards than lung or bladder cancer in male populations.

Many collaborative epidemiological studies have been done with industry in order to access sample populations. A critical review of the scientific literature shows that the partnership between research and industry has often been associated with biases that underestimate the role of working conditions.

The "attributable fraction" concept is based on shaky foundations. Cancers are multi-causal diseases to which different factors may contribute at different times of life. There is no single model that can account for these interactions. In some cases, the synergistic effect comes more from a multiplication than a simple addition of factors. Most epidemiological studies take too little account of multiple exposures throughout working life. The calculation of "attributable fractions" seeks to exclude lifestyle causes. But such causes (smoking, drinking, diet, etc.) are anything but purely individual variables. They may themselves be linked to working conditions. Job insecurity, fear of danger, stress, and night work can influence such behaviours.

The "attributable fraction" approach considers that while some populations are exposed to a risk factor, others are not. The reality is often more complex. Industrial pollution tends to spread risks diffusely. A detailed analysis of actual work activities shows that they

23. Health and safety executive. Statistics. See: www.hse.gov.uk/statistics/index.htm

do not fit apparently well-established exposure scenarios. An excess cancer incidence may even be found in the base population presumed not to be at risk of a given exposure. As a result, the risk ratio for exposed workers is underestimated.

Calculations of “attributable fractions” can only be approximations, therefore. Their effect is to underestimate the role of working conditions in cancers, and they frequently prevent prompt action being taken by public authorities when the introduction of new rules has to wait on cost-benefit assessments.

Looking specifically for occupational cancers - examples

Looking specifically for occupational cancers is key to showing up how much working conditions contribute to contracting cancer. Studies in several countries since the 2000s have highlighted the key role of working conditions in cancer inequality. They also throw into question the traditional view that working conditions have an all-but marginal role in women’s cancers.

The Nordic countries - the Nocca project

The Nocca (Nordic Occupational Cancer) project processes a common database for the five Nordic countries (Iceland, Norway, Sweden, Finland and Denmark) (Pukkala *et al.* 2009). It is a particularly powerful statistical tool, recording 2.8 million cancer cases in occupations pursued by 15 million people over four decades (from the early 1960s to the late 1990s), and enabling new studies to be run identifying the factors that contribute to these higher cancer risks. Most are chemicals, but other factors like exposure to sunlight or work organisation factors (e.g., night work and shift work) can play a significant role. In some cases, the findings further substantiate established links like skin cancer among outdoor working fishermen and farmers, cancers of the nasal cavities among woodworkers, a variety of cancers in the construction industry where workers are exposed to many carcinogens. But the project has also made new findings, such as by identifying a higher prevalence of oral and vaginal cancers among women chemical industry workers; skin, breast (both female and male) and ovarian cancers among print-workers; and thyroid cancer among women farm-workers. Women working in tobacco growing, the chemical and printing industries, hairdressing and sales occupations are also found to have an increased risk of bladder cancer.

Italy – the occupational cancer registers and the Occam project

Italy maintains a mesothelioma register (ReNaM) and a nasal and sinus cancer register. It also has a register of low aetiological fraction occupational cancer (ReNaLOC) based on voluntary GP reporting of cases of cancer of probable occupational origin. An analysis of 936 (non-mesothelioma) cancer cases recorded between 1995 and 2008 indicates that the most common cancer sites in men are the lungs (58%), nose and sinuses (17%), and among women, the nose and sinuses (21%) and breast (21%). The highest incidences by sector are in metal manufacturing, followed by construction, health care and transport. The most frequently mentioned carcinogens are silica, polycyclic aromatic hydrocarbons (PAHs), asbestos and ionising radiation, the latter being implicated in 60% of reported cases among women (especially passenger airline cabin crew). Three-quarters of all recorded cases are from three regions: Lombardy, Piedmont and Veneto.

The study's authors consider that registration data is still very limited and must be augmented through adding more information, improving registration procedures and developing links with the information on occupational cancers in what is known as the Occam system (Occupational Cancer Monitoring) (Scarselli *et al.* 2010, Crosignani *et al.* 2009).

The Occam project was launched in 2001 in Italy's most heavily-industrialized region, Lombardy, and has since been extended to other regions and cities (Umbria, Genoa, Venice). The 35 000-plus cases covered identify companies in which cancer sufferers have worked. A fairly detailed description of actual working conditions can be had. The analysis is immensely valuable for prevention. All cases of cancer in patients between the ages of 35 and 69 are reported by hospitals. Older individuals were excluded due to the problems in getting detailed information about their entire working lives. A statistical comparison can then be made of the frequency of each cancer site in the population of a firm and industries in a province compared to the general population of that region. The Occam project has also reviewed the literature on the cancer-work link to produce working interpretations of the findings. The database provides an overview of more than 900 articles and is prompting active investigation into the work-related origin of cancers by both public health authorities and trades unions. Querying the database for dry cleaning, for example, turns up 25 references to around a dozen cancer sites.

France – the Giscop 93 project

The Giscop 93 project (from the French acronym for a scientific interest group on occupational cancers) appeared in 2001 in an industrial *département* of the Paris suburb of Seine-Saint-Denis. It came about as a collaborative venture between academic researchers and three hospitals, strongly supported by the *département* local authorities and trade unions. Cancer patients reconstitute their working lives with help from a team of investigators. So far, Giscop has focused on three groups of cancer sites: respiratory tract, urinary tract and blood/bone marrow/lymph nodes. In-depth interviews with patients identify potential exposures to carcinogens. Of more than 1 070 cancer sufferers every stage of whose working lives was analyzed between 2002 and 2011, 897 had been exposed for at least one period of their life to a carcinogen in their work, in proportions of 89% of males and 62% of females (Leconte and Thébaud-Mony 2010). Only a quarter of women for whom an occupational exposure was identified had been given a medical certificate as to a possible occupational origin of their disease versus 64% of men. The Giscop project affords a detailed analysis of exposure conditions and gives a basis to highly critical conclusions about the real state of prevention provision. It pinpoints “black spot” areas like outsourcing, and contingent jobs that result in multiple exposures and deny access to effective prevention provision. Giscop peers into the grey areas of work, retracing countless stories of exploitation, denial of rights, and endangerment of health for maximum profit.

Occupational cancers go unseen

Another big obstacle to the “social visibility” of work-related cancers lies in the fact that for many diseases, there is no medical difference between those that are and are not job-related in origin. Cancers often present long – up to 20 or even 40 years – after the initial exposure to toxic chemicals, making it hard to identify potential risk factors. The reason why attention has focused on certain cancers, like pleural and peritoneal mesothelioma or liver angiosarcoma, is because of their infrequency in the general population relative to their

frequency among workers exposed to a particular carcinogen, namely asbestos and vinyl chloride. Lung and bladder cancers are considerably more common types that can also be caused by smoking – and tobacco often gets the blame.

Researchers looking more specifically at lung cancer among men in 1987 reviewed the data published in the literature available at that time, and calculated that the fraction of job-related lung cancers varied from 2.4% to 40%, according to branch of industry (Simonato *et al.* 1987). They also concluded that smoking was not a confounding variable, i.e., it does not change the relation between the disease and the occupation. Since then, the list of recognised lung carcinogens has continued to lengthen: ionising radiation, chromic acid, PAHs, arsenic, asbestos, nickel, iron and iron oxides, cobalt and tungsten carbide Bis(chloromethyl)ether, etc.

But do doctors ask cancer patients about the products they have handled or breathed in during their working lives?

Generalised under-reporting

Whatever percentages are taken, the number of compensated occupational cancers is well below even the lowest estimates. The consensus view is that compensated diseases are only the tip of the iceberg in all EU countries.

There is no EU legislation on occupational diseases. Since 1962, the European Union has adopted only recommendations that have no binding force on Member States. This is part of the reason for the huge inconsistencies in recognition of cancers as occupational diseases.

While there is a general trend to under-recognition in all countries, the levels of it vary widely, as revealed by a Eurogip study (see Table 1).

Table 1 Recognised cancers relative to the insured population in 2006

Country	Recognised cases	Insured population	Recognition per 100 000 people insured
Austria	84	3 089 167	2.72
Belgium	245	2 483 948	9.86
Czech Republic	38	4 497 033	0.85
Denmark	135	2 710 462 (2005)	4.98
Finland*	139	2 129 000	6.53
France	1 894	18 146 434	10.44
Germany	2 194	33 382 080	6.57
Italy	911	17 686 835	5.15
Luxembourg	13	279 810	4.65
Spain**	4	15 502 738	0.03
Sweden	43	4 341 000	0.99
Switzerland	128	3 651 709	3.51

* Finland: the ratio is calculated based on claims for recognition (failing available data on recognised cases).

** Spain: the number of cancers recognised began to increase from 2007 on; if the 2008 data for population and recognised cases were taken, the ratio would be 0.39.

Source: Eurogip (2010)

On top of these aggregate data are the facts that the vast majority of recognised cancers are caused by asbestos and that women find it much harder than men to get their occupational cancers recognised.

The number of recognised occupational cancers has risen steadily in France from 1 033 cases in 2000 to 1 898 in 2008, more than three quarters of them asbestos-related. With 10.4 cancers per 100 000 insured, France had the highest rate of recognised occupational cancers in 2006.

In most other European countries, the figures are still very low: just 19 cases of occupational cancer recognised in Sweden, 62 in Spain, and 168 in Finland in 2008.

Mesothelioma is the most typical occupational disease and asbestos the most universally used and recognised carcinogen. Asbestos cancers, including mesotheliomas, make up more than three-quarters of compensated occupational cancers in the EU. However, some countries report no mesothelioma cases, and even in those countries that report the most, the numbers still nowhere near reflect the real scale of asbestos cancers.

Denmark prides itself on having a first-rate system for reporting occupational cancers. And yet, in 1990, a study which set out to analyse the reporting of cases of pleural mesothelioma and ethmoid and sinus adenocarcinoma – two cancers associated with occupational exposure to asbestos and wood dust, respectively – estimated under-reporting at approximately 50%. An examination of the medical records of patients who had not reported their disease revealed that, in most cases, the records held too little detailed information on occupational exposures. Recommendations were made following the study. A fresh evaluation was done in 2000, in which Danish cancer registry data were compared with those from the national industrial injuries office. The comparison revealed that the cancer registry had recorded 49 cases of ethmoid carcinoma and 73 pleural mesotheliomas, while the national industrial injuries office had received only 11 recognition claims for ethmoid carcinoma and 48 for mesothelioma. New measures have since been taken to improve reporting of occupational diseases.

Reasons for the invisibility of occupational cancers

The reasons why occupational cancers remain invisible are many and of different orders: regulatory, economic, social and medical.

Regulatory

One reason for under-reporting could be the limited number of cancers recognised as being work-related and the chemicals apt to have caused them. Most EU countries operate a dual system combining a “schedule” or “closed list” of compensable occupational cancers, and an “additional” or “open” system²⁴.

Those with “scheduled” diseases must prove the disease and working conditions described in the schedule but causation is presumed. Those with “open” system diseases must make a personal application and provide proof not only of the disease but also a causal link between their working conditions and the disease.

In most European countries, the “open” system seems to be no more than a marginal way of getting recognition for an occupational cancer. No cancer bears the distinctive mark of the carcinogens that may have contributed to its development and growth.

²⁴. Iceland and Sweden have no fixed schedule of occupational diseases but instead compensate them on a case-by-case basis.

Improved recognition in Germany

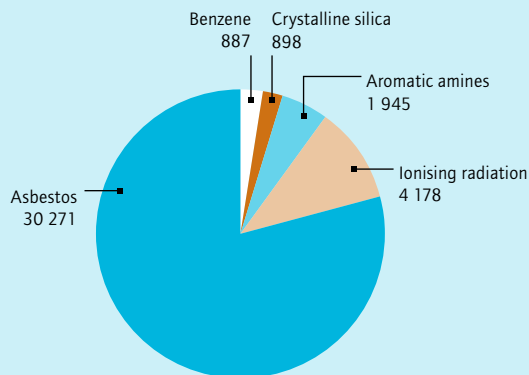
Germany is one of the EU countries with a good rate of recognition of occupational cancers, and has been keeping detailed figures on the percentage of compensated occupational cancers compared to estimated occupational cancers since 1978. The situation has improved over time.

Table 2 Trend in the compensation of occupational cancers in Germany (1978-2010)

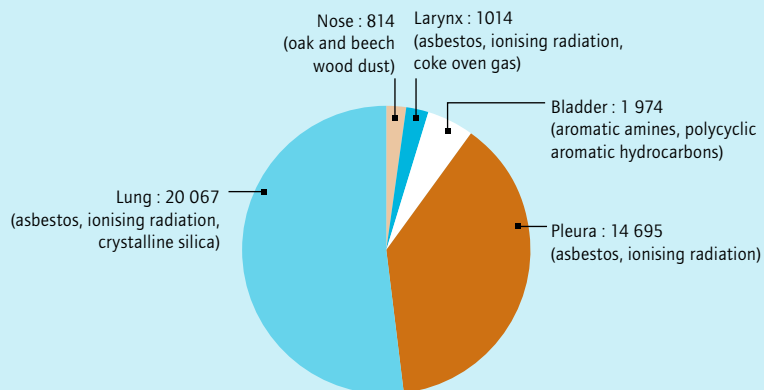
Year	Number of compensated cases of occupational cancer	Estimated total number of occupational cancers	% of compensated cases
1978	96	13 214	0.7
1988	455	7 637	6.2
1998	1 913	18 614	10.3
2008	2 074	12 244	16.9
2010	2 144	14 612	14.7

Graph 2 Origin of occupational cancer cases recognised in Germany, 1978-2010

Total number of cases
40 555



Graph 3 Main sites of occupational cancers recognised in Germany, 1978-2010



Sources : Butz M (2012) Beruflich verursachte Krebserkrankungen, Eine Darstellung der im Zeitraum 1978 bis 2010 anerkannten, Berufskrankheiten Deutsche Gesetzliche Unfallversicherung (DGUV); Recognised cases of OD www.dguv.de

Requiring a worker to prove a causal link between their cancer and an occupational exposure is a grossly inordinate demand.

This explains why no case of an occupational cancer has been recognised in recent years under the additional system in Belgium and Luxembourg; a single case in Switzerland between 2000 and 2007; only two cases in Austria between 2000 and 2008; and 1.1% and 2.2% in Germany and France respectively of the cases recognised in 2008. Italy is the only exception, with a rate of 13% in 2008 (Eurogip 2010).

The Netherlands has no system for the recognition of occupational diseases: sufferers are catered to by the health branch of the social security system whatever the origin of their disease and must sue the employer for damages in the civil courts²⁵.

A comparison of the schedules of occupational diseases in different European countries shows a measure of consistency. Skin cancers are a case in point, as are bone cancers, bronchopulmonary leukaemias and cancers, where the causative chemicals – like chromium, asbestos and nickel – are universally accepted. By contrast, iron oxide, cobalt and silica are accepted in only a handful of countries. Brain tumours are listed only in the French schedule. Bladder and liver cancer tend to be recognised only in connection with one chemical each: aromatic amines for the former, vinyl chloride for the latter.

An agent will often be recognised as causing only one type of cancer. Vinyl chloride, for example, is recognised for liver angiosarcoma, but not for other cancers of the liver or the other tumours described in the medical literature.

The International Labour Organisation (ILO) list

The International Labour Organisation (ILO) approved a new list of occupational diseases in 2010 listing 20 substances, groups of substances, and physical and biological agents as causes of occupational cancer²⁶. The ILO also opens up the list to cancers caused by other agents not mentioned among the 20 cited “where a direct link is established scientifically (...) between the exposure to these agents arising from work activities and the cancer(s) contracted by the worker”²⁷. The ILO list is not binding; it is a recommendation to the Member States.

The European Schedule

Likewise the European Commission Recommendation of 19 September 2003²⁸, Annex I of which contains a European Schedule of 108 occupational diseases or groups of diseases, and Annex II an additional list of 48 diseases or groups of diseases suspected of being occupational in origin, 36 of them involving chemicals. The European Trade Union Confederation (ETUC) has challenged the composition of these lists, pointing out that asbestos-caused cancer of the larynx, for example, is on the EU’s additional list when it already has recognised occupational disease status in several EU countries.

25. On which see: Heuts P. (2013) Dutch FNV union makes employers pay up for work-related diseases, HesaMag, 7, 35-40. Downloadable on: <http://www.etui.org/en/Topics/Health&Safety/HesaMag>

26. Asbestos, benzidine and its salts, Bis-chloromethyl-ether, chromium VI compounds, coal tars, coal tar pitches or soots, beta-naphthylamine, vinyl chloride, benzene, toxic nitro- and amino-derivatives of benzene or its homologues, ionising radiation, tar, pitch, bitumen, mineral oil, anthracene, or the compounds, products or residues of these substances, coke oven emissions, nickel compounds, wood dust, arsenic and its compounds, beryllium and its compounds, cadmium and its compounds, erionite, ethylene oxide, hepatitis B virus and hepatitis C virus.

27. List of occupational diseases (revised 2010) ILO Occupational Safety and Health Series, 74.

28. Commission Recommendation of 19 September 2003 concerning the European schedule of occupational diseases.

Economic, social and medical

Other, non-legal factors – not least social and medical – are also in play in the under-recognition of occupational cancers. In some countries, procedures are complicated, unsuitable, uncertain, off-putting and the occupational disease is poorly compensated, while in others, pressure from employers on occupational doctors or employees is a deterrent to reporting an occupational disease (European Commission 2013).

Two French surveys on the fate of occupational asthma victims showed that a big reason for under-reporting of occupational diseases was that victims frequently failed to report their disease because of the danger of losing their job and income (Eurogip 2002). Another survey showed that even in a teaching hospital where exposure to well-known carcinogens was involved, cancers had not been reported as occupational diseases. Analysis of the causes revealed doctors' disinclination to look for an occupational cause of medical conditions, and attending practitioners' and employees' lack of information or misinformation about the procedure for recognition of occupational diseases.

The sociologist Annie Thébaud-Mony highlights the problems doctors face with regard to work-related cancers (Thébaud-Mony 2006). They have to identify exposure to one or more carcinogens, which involves tracing back careers, having access to an individual's work history. Patients often do not know what products or dust they have been exposed to. Anything from 10 to 40 years may elapse between the time of exposure to a carcinogen and the development of a cancer. But above all, she argues, they must get away from the overriding perception of cancer as a disease related only to risk behaviour.

Even a cancer like pleural or peritoneal cancer (mesothelioma) incontrovertibly linked to a carcinogen may face other obstacles. Of 2,407 mesothelioma cases recorded between 1999 and 2009 by the National Mesothelioma Surveillance Programme in France, no application for recognition as an occupational disease or compensation from the compensation fund for asbestos victims (FIVA) was received in 30% of cases. An investigation into the reasons why found that gender, age at diagnosis, social status and type of health care coverage influence the claim for compensation of the disease (Chamming's *et al.* 2013).

Many countries have long-established oversight over workers' health and working conditions. Safety services take workshop air measurements, the occupational health service does urine and blood tests on workers exposed to toxic substances. The occupational doctor could play a key role in identifying occupational cancers, but is often left out of the information loop.

The impact of occupational doctors in preventing occupational cancers is uncertain. Often, their lack of independence from the employer makes it difficult for them to get involved in a risk prevention culture, especially where prevention has to compete with big industrial and economic concerns, as some particularly telling examples show.

Women often ignored

Women die less frequently from cancer than men. The standardised cancer death rate was 212 per 100 000 for men, and 128 per 100 000 for women in the EU in 2012. Might this be why women are found to be so little in evidence in the scientific literature? An American literature review of all articles on occupational cancers published between 1971 and 1990 found that only 35% included women, and only white women (Niedhammer *et al.* 2000). In 2000, an Inserm survey of health and safety at work research published in 1997 found that 31% of articles were concerned exclusively with men against 7% with women; 51% covered

both genders, but generally without distinguishing between them, even though the biological mechanisms that result in cancer may be gender-differential.

The explanations given for this situation are firstly, that men are more frequently exposed than women to serious risks in their work, and more so to carcinogens, and secondly that they more frequently work in large firms (metallurgical and chemical industry), which facilitates epidemiological research. None of these explanations is entirely satisfactory. That the gendered division of labour may produce a greater concentration of men in particular high cancer-risk jobs does not mean that women are immune from it.

Women will often be found in “peripheral” jobs, like premises cleaning, final assembly or finishing operations, packaging and packing, etc., for which almost no data are available. Account must also be taken of the interaction between the different carcinogens not just in paid work (where exposures linked to basic production interact with exposures linked to cleaning products) but also in unpaid house work, still overwhelmingly done by women. It is nevertheless surprising that so little research has been done into connections between the most common cancer among women, breast cancer (more frequent even among women than lung cancer in men), and the occupation of those affected by it or the products they have handled.

But female manual workers have at age 35 a life expectancy three years less than that of female managerial staff; while women manual workers have a death rate 40% higher than managerial staff between the ages of 35 and 80. In France’s heavily female-dominated service and domestic staff sector, 28% of workers are exposed to carcinogens like formol and chlorinated solvents. But as neither of these is included in the regulations on recognised occupational diseases, any attempt to report an occupational disease would be doomed to failure. This hardly contributes to making female occupational cancers visible!

In recent years, however, researchers have been looking more closely at the work link to female cancers. Progress is slow-going. A recent literature review of 122 articles published between 2006 and 2012 in thirteen international gold standard epidemiological journals – all original studies on the role of occupational factors in lung cancer, a disease with a high incidence among men and women alike – found that barely 4% of the articles focused on an exclusively female base population, 45% on a mixed base population and 51% on an exclusively male base population. In the mixed populations, men were significantly over-represented relative to women²⁹.

29. Communication from Mr Charles-Olivier Betansedi (GIS COP 93), October 2013.

Breast cancer and work

A French survey of more than 1 200 cases of breast cancer done in 2013 found over-representation of women workers in the textile and clothing, rubber and plastics industries and the nursing profession (Guénel and Villeneuve 2013).

A Canadian study of a thousand women done in 2012 which looked at the link between breast cancer and workplace exposure to carcinogens and endocrine disruptors (Brophy *et al.* 2012) found a risk of developing breast cancer on average 40% higher in sectors where women were exposed for at least ten years to carcinogens and endocrine disruptors. The most affected sectors are agriculture, metal manufacturing, the production of plastic parts for the car making industry and food preservation, with the latter two carrying a 5-times higher risk of developing breast cancer before menopause.

The authors establish a link between the breast cancer risk and exposure to carcinogens and endocrine disruptors, and stress the importance of detailed information on the personal and work history of victims.

A 2013 Italian study which analyzed 11 188 cases of female breast cancer contracted in Lombardy between 2002 and 2009 compared to a control population of 25 000 women (Oddone *et al.* 2013) found a higher risk of breast cancer for women employees in the electrical manufacturing and rubber industry (25% increased risk).

Since the IARC classified shift work as a probable carcinogen in 2007, researchers have studied the effects of shift work and night work on women's health. A Danish study of 7 000 women with breast cancer found a 50% increased risk for those who mainly worked nights. In Denmark, 38 women with a long history of night work had their breast cancer recognised and compensated as an occupational disease (Hansen and Lassen 2011).

Chapter 7

Going for gold – and a toxic industrial mindset

Manufacturers do not like it known that workers die of work-related cancers in their factories. If some do something by way of prevention, or replacing dangerous substances with less dangerous ones, it is often because legislation forces them to. Most would rather try to delay a ban on dangerous substances, and the need to take measures that are judged to be too costly even though they protect workers' health. The asbestos industry offers a striking example of this type of mindset.

"Controlled asbestos use"

The asbestos industry geared up very early on to secure the long-term future of their highly profitable businesses, under threat from ever more damning epidemiological surveys. Dr Irving Selikoff reported his findings of a high number of mesothelioma and lung cancer cases among the asbestos insulation workers to the New York Academy of Sciences Congress in 1964³⁰. The industry was quick to mount its counter-attack, as a result of which the United States and most European countries have asbestos lobbies that are backed by the Asbestos International Association (AIA). The AIA's membership includes firms like Johns-Manville, Cape Asbestos, Turner and Newall and Eternit. From the turn of the 1960s, the asbestos industry fashioned a strategy to enable them to continue using the material, successfully arguing for "controlled asbestos use". In 1976, the "Chambre syndicale de l'amiante" – the French asbestos industry trade association

30. In 1982, Irving Selikoff produced new findings that the reported deaths in the group of insulation workers included 45% from cancers, 20% from lung cancer alone and 10% from mesothelioma (Selikoff 1982).

– took out a full page advertisement in newspapers, putting over the message, “the odd problems created by asbestos pale into insignificance beside the immense service it does for you each day, without you even knowing it. (...) let’s learn to live with asbestos³¹.”

This, even though in 1977, the International Agency for Research on Cancer (IARC) had said that it was not possible to assess an asbestos exposure level below which there would be no increased cancer risk. The IARC classifies all varieties of asbestos as carcinogens. In the same year, France set its first asbestos exposure limits, more than 45 years after the United Kingdom. Even though hardly revolutionary, they went largely unapplied. In the shipyards, for example, exposure levels were found of 100 to 1 000 times above the regulation levels.

A French Senate report of 20 October 2005 described the French State as “paralysed” by the asbestos lobby. The Standing Committee on Asbestos (CPA) established in 1982 was an informal committee of business leaders, doctors, scientists, trade unionists and Ministry of Labour and Health officials. It was a particularly effective tool for the asbestos industry. In the words of the French Senate report, “By playing on scientific uncertainties, which are steadily receding over time, the CPA has succeeded in sowing doubt about the significance of the risk of exposure to asbestos, thereby delaying the banning of asbestos in France for the longest possible time”³².

Similar agencies also emerged in other countries. The lobby most active in Belgium, the Netherlands and Luxembourg was the BAIC – Benelux Asbestos Information Centre – which put out brochures for the specific purpose of clearing the name of asbestos cement. Belgium is home to the multinational Eternit – one of the world’s biggest asbestos cement producers.

In North America, the Chrysotile Institute – a propaganda and disinformation tool to support chrysotile asbestos mining in Quebec – was funded by the Canadian authorities until 2012. The Institute claimed chrysotile (the only form of asbestos still used) to be less dangerous than other forms of asbestos. In 2013, the last working asbestos mine in Quebec shut down on economic and health grounds.

Russia, however, which still possesses working asbestos mines, took up the lobbying baton, carrying other pro-asbestos countries along (Ukraine, Kazakhstan, Zimbabwe, Kyrgyzstan, Vietnam and India). The “dirty 7” as they are dubbed by asbestos sufferers’ associations are peddling the sedulously propagated myth about the supposed safety of chrysotile by using mining communities in fear of losing their livelihood.

Russia’s annual asbestos sales earn it some \$550 million. The 38 500 workers employed by its asbestos industry often have to carry on working in the mines to keep body and soul together. In the aptly named Urals town of Asbest, 17% of the population works for Uralasbest, the owner of a gigantic asbestos mine sprawling over 50 square km. “Every normal person is trying to get out of here. People who value their lives leave”, says a former asbestos factory employee³³.

Cover-ups

In the mid-1960s, Belgian occupational doctors reported a new disease seen among workers involved in cleaning autoclaves used to polymerise vinyl chloride into polyvinyl chloride³⁴. The new disease – acro-osteolysis – causes destruction of bone at the fingertips.

31. “À propos de l’amiante”, advert published in *Le Monde* daily newspaper, 17 November 1978, p. 8

32. Dériot G. and Godefroy J.P. (2005) *Le drame de l’amiante en France : comprendre, mieux réparer, en tirer des leçons pour l’avenir*, Report by the French Senate, 26 October 2005, tome I, p. 79.

33. Kramer A. (2013) City in Russia unable to kick asbestos habit, *The New York Times*, 13 July 2013.

34. An autoclave is a thick-walled, hermetically-sealed pressurised vessel used either for producing industrial reactions, or steam-cooking or – sterilising.

The discovery threw the chemical industry into turmoil. It was the time of a burgeoning awareness in the United States of the risks related to the growing use of chemicals.

Suspicion focused on PVC, hitherto seen as harmless. PVC is used in the manufacture of hundreds of consumer goods. Manufacturers feared that the reputation of their products would be tainted.

The University of Michigan carried out an epidemiological survey, backed by the big world chemical groups. The findings were that the disease also attacks conjunctive tissue, and is not confined to the fingers. The authors showed that workers were actually exposed to levels well above the then-accepted threshold limit value of 500 ppm, and recommended that the level be reduced to one-tenth of that value to ensure workers' safety. Industry took issue with the recommendations, and when the study was published in 1971, it contained no reference to the threshold limit values, and left a question mark over whether vinyl chloride was in fact the cause of the disease.

The chemical industry was soon to receive further bad news. Animal studies done in Europe by the Italian researcher Pier-Luigi Viola showed vinyl chloride to be carcinogenic at high doses. This fuelled concerns among vinyl chloride manufacturers, as no substance which is or is suspected of being carcinogenic had been allowed in food in the United States since 1958. But PVC provided the packaging of many foodstuffs.

Even so, the chemical industry did not seem minded to reduce exposure levels, arguing that vinyl chloride was dangerous only at high doses.

In 1972, the initial findings of a study commissioned by the European chemical industry from another Italian researcher, Cesare Maltoni, to check Viola's work dealt a savage blow to the chemical industry lobby. It showed that vinyl chloride does cause cancer in animals, even at low doses. European producers demanded that their American colleagues keep the findings under wraps.

The silence was broken soon afterwards by an article in an Italian newspaper written by one of Viola's former associates, speaking out against the rash of cancers thought to be caused by vinyl chloride among European workers. Industry could no longer hide the facts. In January 1974, news leaked out of the deaths of four workers at the Goodrich factory in Louisville (Kentucky) from a rare cancer – liver angiosarcoma – linked to their exposure to vinyl chloride. It was the same type of cancer as those reported in Maltoni's rat studies. Cases were then identified at all production sites.

In 1974, the U.S. Occupational Safety and Health Administration (OSHA) set the occupational exposure limit for vinyl chloride at 1 ppm. Vinyl chloride is claimed to be responsible for hundreds of cases of hepatic angiosarcoma across the world. Subsequent studies would show that vinyl chloride can also cause other liver cancers, bronchial cancers, brain tumours and leukaemia (Soffritti *et al.* 2013).

A Directive³⁵ set the limit for workers' exposure to vinyl chloride at 3 ppm in the European Union in 1978. As of 2013, this limit had still not been revised (Directive 2004/37/EC under revision). Some countries, however, have lowered it unilaterally - France and Sweden, for example, have reduced it to 1 ppm.

35. Council Directive 78/610/EEC of 29 June 1978 on the approximation of the laws, regulations and administrative provisions of the Member States on the protection of the health of workers exposed to vinyl chloride monomer.

Delaying the application of more binding standards

Benzene is an example of the crucial issue that exposure standards represent in terms of profit for some, and lost lives for others. Benzene is originally a by-product of the gas and tar recovered in coke ovens. It is an aromatic hydrocarbon. It is a solvent regarded as one of the most dangerous products that workers may encounter. Benzene is particularly toxic to blood cells and the organs that produce them, including bone marrow.

The extent of the damage depends on the dose of benzene to which the worker was exposed. Exposure to benzene, even at very low but continuous exposures, can cause leukaemia. The European Carcinogens Directive now makes 1 ppm a binding occupational exposure limit. But it was a standard long – too long – in the making.

Although the first reports of blood cell damage due to benzene date from the late 19th century, benzene use continued to spread in first the rubber, then the inks, adhesives and paint industries after 1910. This commercial success was accompanied by a rise in cases of what was then called “benzene poisoning”. Some victims fell ill very soon after starting work, dying within months. It was thought that poisoning occurred only at benzene levels above 200 ppm.

A 1926 study done in 12 US benzene using firms reported that 44% of their employees had abnormally low white blood cell levels. This high rate of blood ailments was found with exposures above 100 ppm. Two years later, the link between benzene and leukaemia was made (European Environment Agency 2001).

By the turn of the 1930s, cases of benzene poisoning were being seen pretty much worldwide. Some analysts called for benzene to be replaced by another solvent. A 1939 study of 89 cases of benzene poisoning and three of leukaemia found that two cases had occurred after exposure below 25 ppm. From the late 1940s, the American industrial hygiene association continued to press for exposure limits to be brought down to 100 ppm, 50 ppm, 35 ppm and, in 1957, 25 ppm. But workers in many countries continued to work in exposures of hundreds, not to say thousands, of ppm. In the 1960s, several publications called attention to benzene-related diseases, especially leukaemia in the Italian and Turkish footwear industries where benzene-based adhesives were used. The International Labour Organisation (ILO) adopted a Recommendation (No. 144) on the use of benzene in 1971, but without setting an exposure limit.

The allowable concentration was reduced to 10 ppm when, in 1977, the first large-scale epidemiological study done in a plastics packing factory found that benzene-exposed workers were from 5 to 10 times more likely to develop leukaemia at exposure levels of between 10 and 100 ppm. The US Occupational Safety & Health Administration (OSHA) then decided to lower the workplace exposure limit to benzene to 1 ppm. The American Petroleum Institute took issue with this, arguing that there was no increased leukaemia risk below 10 ppm.

The dispute went to the Supreme Court, which held that before making any change to the standard, OSHA must show that a “significant risk” exists at an exposure of 10 ppm which may be reduced by lowering the exposure. The Supreme Court considered a risk to be significant where the calculated probability of harm is increased by 1 case in 1000 workers over a working life. This is a crucial decision, because this definition of significant risk is now the rule in the United States, and has lengthened the time in which OSHA can publish new standards. The Supreme Court ruling had implications for all U.S. government agencies, including those with public health responsibilities, which have to prove the benefit of any change in statute law.

It was not until 10 years later, in 1987, that the 1 ppm standard was finally promulgated as the occupational exposure limit for benzene. Researchers calculate that the delay

in applying the standard in the United States probably resulted in an additional 275 deaths – 198 from leukaemia and 77 from multiple myelomas. But the probability of dying of leukaemia remains high even at 1 ppm. It has been calculated that this level of exposure results in from 4 to 15 deaths per 1 000 exposed workers (Nicholson and Landrigan 1989).

US oil industry internal documents show that as early as 1948, industry heads regarded the only safe level of exposure to benzene as being zero. The exposure limit recommended today by US hygienists is 0.5 ppm.

The regulatory limit value of 1 ppm for benzene in the EU was set in a 1999 Directive³⁶, but did not come into effect until 2003, fifteen years after the United States. Many lives would likely have been saved had this limit value been imposed earlier.

Benzene remains a very widespread source of air pollution because unleaded petrol and diesel can still contain up to 1% of it (by volume).

The “delay game” goes on

The “delay game” is how a U.S. non-governmental organisation, the Natural Resources Defense Council (NRDC), has described the business tactics for holding up the introduction of tighter standards or recognition of the real dangers of certain substances.

The NRDC gives a colourful description of the basic steps in “the four dog defence”:

- First step: *my dog does not bite*. The company denies that its product is harmful. This may include attempts to discredit scientific studies or their authors.
- Second step: *my dog bites, but it didn't bite you*. Industry concedes that the product is potentially harmful but that no one is exposed to it. Absence of data is used to argue that there is no exposure.
- Third step: *my dog bit you, but it didn't hurt you*. Industry admits that people are exposed, but denies that the exposure caused harm. Industry concedes that the product is harmful at very high doses, but not at the lower levels to which people are actually exposed.
- Fourth step: *my dog bit you and hurt you, but it wasn't my fault*. Industry tries to avoid legal liability by blaming past exposures, improper use or shifting the blame to other chemicals, medicines, smoking, etc.

It is a well-established, yet still effective strategy which the NRDC has analyzed in detail as used in three specific cases involving trichloroethylene, formaldehyde and styrene. The U.S. environment agency (EPA) has been struggling to finalise its evaluation of trichloroethylene for 22 years, and that of formaldehyde for 14 years. For styrene, the EPA's re-evaluation started in 1998 is still uncompleted (Sass and Rosenberg 2011).

Industry has no compunction about using the services of reputable scientists to play down the health risks of highly toxic substances, even those classified as human carcinogens by the International Agency for Research on Cancer (IARC). In December 2013, French broadsheet *Le Monde* unveiled the close ties that the highly influential epidemiologist Paolo Boffetta has with industry³⁷. The prestigious daily reported that former International Agency

36. Council Directive 1999/38/EC of 29 April 1999 amending for the second time Directive 90/394/EEC on the protection of workers from the risks related to exposure to carcinogens at work and extending it to mutagens.

37. Foucart S. (2013) Épidémiologie: des liaisons dangereuses, *Le Monde*, Science and Technology supplement, 16 December 2013.

for Research on Cancer (IARC) researcher Mr. Boffetta has in recent years provided consultancy services to a growing number of companies and business associations. His name also appears on articles in scientific journals denying or downplaying the carcinogenicity to humans of formaldehyde, styrene and diesel engine fumes, among others.

The EPA and OSHA's setbacks with benzene are not wholly down to corporate belligerence, but were enabled by the official mechanisms put in place that water down their powers to protect human health and the environment. Underlying these changes is the principle that a legal rule is justified only if cost-effective. Before bringing new rules in, therefore, the public authorities must conduct impact assessments that generally involve weighing up the supposed costs against the expected benefits. These calculations are very largely based on questionable assumptions and extrapolations from very limited data. Behind the guise of scientific rationality actually lie political and social/labour choices.

The watershed moment in the United States dates from the Reagan era. During his election campaign in 1980, the Republican candidate had even proposed scrapping OSHA. As President, however, he took the much subtler tack of keeping federal agencies going but obliging them to calculate the costs and benefits of their regulatory proposals. This was an obstacle course that basically blocked any progress in the regulatory framework for three decades. Subsequent administrations, Democrat and Republican alike, have taken the same tack and added to the roadblocks.

Recent years have seen the EU go down the same road, holding back and blocking any ambitious regulation in the name of "better regulation" and "smart regulation".

REACH and chemical industry lobbying

The REACH regulation to try and control chemicals produced or marketed in the European Union was adopted by second reading approval in the European Parliament on 13 December 2006. It was the finishing line of an obstacle race beset by ferocious lobbying from the chemical industry both in Europe and the United States.

The report written for US Congressman Henry Waxman (Democrat) published in April 2004, shows that US chemical industry lobbying was played out at the highest level (Waxman 2004). The report draws on internal documents (cables, memoranda, emails) from various US government agencies.

The Waxman report disclosed that the US chemical industry had given US\$ 21 million in electoral campaign contributions between 2000 and 2004, 80% of which had gone to the Republican Party. President Bush had been the top recipient, having received \$900 000 between 1999 and 2004. The report also shows that several federal agencies and senior government officials, like former Secretary of State Colin Powell, intervened at the same time to thwart the proposal for a REACH regulation.

Right from coming to power, the Bush Administration canvassed the US chemical industry's views and concerns. Meetings were held in the United States and Europe between Bush administration officials, US diplomats posted to Europe, organisations representing the different sectors of the chemical industry, firms like Dupont and Dow, to build a case focused on the cost, complexity and bureaucracy of the draft regulation. That case was then to be argued to Member State governments and the European Commission. In September 2003, Jacques Chirac, Gerhard Schröder and Tony Blair wrote to the then European Commission President Romano Prodi urging the Commission to take the legitimate interests of European business into account.

The Waxman report notes the changes that were made between the White Paper as published by the European Commission in February 2001, and the proposal for a REACH regulation

laid before the Parliament and European Council on 29 October 2003. Changes that enabled the American Chemistry Council's 2003 report to welcome the "significant concessions in the draft" achieved by the opposition to the Commission's preliminary draft regulation.

The European chemical industry lobby waged its own all-out assault on REACH, with employers' federations, especially the European Chemical Industry Council (CEFIC) and the Union of Industrial and Employers' Confederations of Europe (UNICE) keeping up unrelenting pressure both before and after the draft was tabled.

German chemical industry firms, especially BASF and Bayer, were the most active and influential at both national and European level. A Greenpeace special report entitled *Toxic lobby* reported that BASF had confirmed to the German press that it had 235 politicians "under contract" (Greenpeace 2006).

The environmental group even supplies examples of former BASF and Bayer employees who went on to occupy senior posts in UNICE and CEFIC and even, in some cases, the Commission or European Parliament departments in charge of REACH. But it was also a two-way traffic.

According to Inger Schörling, a Greens group MEP until June 2004, the industry lobbies campaigned towards MEPs using "seminars, workshops, meetings, lunches, dinners, letters, mailouts, phone-calls, visits to plants, media releases and any other component that could be used" (Schörling 2004).

Just ahead of the European Parliament's first vote on REACH in November 2005, Environment Committee rapporteur Guido Sacconi spoke of the "incredible pressure exerted on MEPs by big business" (Corporate Europe Observatory 2005).

The Internal Market Committee rapporteur, Harmut Nassauer, received direct assistance from a German chemical industry employee. On 13 December 2006, following the second reading vote, the ETUC condemned pressure from the chemical industry for having reined in the reform. The European trade union confederation lamented that information vital to protecting workers' health given in the chemical safety reports would now only be required for a third of the chemicals originally planned.

REACH is being phased in. An eleven-year, multi-stage transition period was provided from 2007 to 2018. Contrary to the forecasts of the chemical industry's hired consultants, REACH has not driven the European chemical industry into a black hole. Rather, most of the problems that surfaced in the REACH negotiations are still besetting its implementation. The chemical industry is still trying to persuade the public authorities to cut down the scope of REACH, limit the information made public and delay banning the most hazardous chemicals³⁸.

38. On which, see HesaMag (2013) Chemical hazards: state of play 6 years into REACH, 8, 12-44. Downloadable at: <http://www.etui.org/en/Topics/Health-Safety/HesaMag>

Chapter 8

A global issue

The social inequalities described in this brochure are obviously magnified many-fold if the scope of analysis is extended planet-wide. The globalisation of capital flows is all about maximising the return on investment. With this, human life and the environment become mere economic variables that shape the factors of competitiveness. One very simple fact is clear from an examination of the lifecycle of any product chain: the activities most harmful to health and the environment tend to concentrate in countries least resistant to exploitation. This is true for traditional sectors like agriculture and raw materials extraction, but no less so for high technology sectors like electronics and advanced chemicals. Multinationals systematically operate double standards. The European trade union movement has a responsibility here to workers in countries where European multinationals operate. It should develop ways of supporting the trade union struggle for health and safety at work in the countries affected, and fight double standards as operated by business and in the EU's international activities.

The REACH regulation bears recent witness to the pressing need for international trade union solidarity to thwart attempts to export the most dangerous industrial activities or products to developing countries. In the discussions leading up to the adoption of REACH, industry pressed for the regulation's scope to be restricted to chemicals for the European market only³⁹. Not only was this demand deeply cynical and grossly unethical, it was also unworkable.

Since Rachel Carson's book *Silent Spring* was published in 1962, there has been a general awareness that the use of chemicals, like pesticides such as DDT, has

39. Cefic (2005) New proposals to improve workability of REACH, 24 February 2005, p. 4.

effects across the world. “For the first time in the history of the world”, she writes, “every human being is now subjected to contact with dangerous chemicals, from the moment of conception until death. In the less than two decades of their use, the synthetic pesticides have been so thoroughly distributed throughout the animate and inanimate world that they occur virtually everywhere. (...) They have entered and lodged in the bodies of fish, birds, reptiles, and domestic and wild animals so universally that scientists carrying on animal experiments find it almost impossible to locate subjects free from such contamination. They have been found in fish in remote mountain lakes, in earthworms burrowing in soil, in the eggs of birds, and in man himself. For these chemicals are now stored in the bodies of the vast majority of human beings, regardless of age. They occur in the mother’s milk, and probably in the tissues of the unborn child (Carson 1962).”

Global pesticides regulation is essential

Citizens in developed countries have battled to get dangerous pesticides banned only to find later that they may be present as residues in food imports from developing countries where they are still used, often by American or European multinationals. The death toll from pesticides in the world is estimated at 10 000 today. Three in four of them are in developing countries.

After a series of scandals, the United Nations Food and Agriculture Organisation adopted a code of conduct on the export and sale of pesticides in 1985. Later on, in 1987, it accepted the principle of prior informed consent (PIC), subsequently taken up and administered by the United Nations Environment Programme. It was a voluntary system. The prior informed consent procedure has since been incorporated in the Rotterdam Convention, which came into force in 2004 and is now binding on the countries that sign up to it. In theory, the Convention covers all hazardous chemicals. The European Union approved it by a Council Decision of 19 December 2002. The Convention lays down as a general principle that a chemical covered by the Convention can be exported only with the “prior informed consent” of the importing country.

One big limitation of the Convention is that it does not automatically apply to all a producer country’s dangerous substances. For a chemical to be subject to the prior consent procedure, it must be listed in Annex III of the Convention.

At present, this only lists 39 chemicals – 24 pesticides, 11 industrial chemicals, and 4 severely hazardous pesticide formulations. As of early 2014, only 47 substances or groups of substances are listed: 33 pesticides or groups of pesticides and 14 products for industrial use (including five types of asbestos, but not chrysotile asbestos⁴⁰). The practical effect is that a State may regard a product as particularly hazardous, yet continue to export it without even informing the State that is receiving the hazard, provided the chemical is not listed in Annex III. So, Canada consumes only minute quantities of the asbestos it produces, and exports the rest to countries in Asia, Africa and Latin America.

But the developing countries are now themselves producing pesticides. India has become the foremost world producer, and its population – 56% of whom work the land – is suffering the direct consequences in the form of acute poisoning and chronic diseases like cancer. A survey in southern India disclosed that most of the peasants who use pesticides take no safety precautions (Chitra *et al.* 2006).

40. Rotterdam Convention, PIC Circular XXXVII, June 2013.

No global asbestos ban yet in sight

Asbestos has been banned throughout the EU since 2005, but the long latency of asbestos cancers means that its effects will be felt for long to come. In 1999, the English epidemiologist Julian Peto forecast approximately 250 000 deaths in Western Europe from asbestos-related cancers in the first 35 years post-ban. Asbestos consumption fell sharply in the United States from the early 1970s. Epidemiologists believe that the mesothelioma epidemic has already begun to decline and are forecasting a return to “normal” by 2055!

Notwithstanding the 100 000 deaths a year estimated by the ILO, world asbestos production remains high. It stood at 2 080 000 tonnes in 2003 – 60% of its 1970 all-time high. The biggest producer countries include the Russian Federation, China and Canada. Russia and Canada have so far managed to stop chrysotile asbestos being included on the Rotterdam Convention list of chemicals. Asia – especially India, China and Thailand – is the asbestos industry’s market of choice today. After 2000, production levelled off at 60% of its 1970 level but has not fallen further. In 2013, as in previous years, it stood at close to two million tonnes.

Thirty-odd other countries have joined the European Union in banning asbestos⁴¹. Nongovernmental organisations have been campaigning for a world asbestos ban for several years through the International Ban Asbestos Secretariat (IBAS). They lobby each Conference of the Parties to the Rotterdam Convention – which brings the signatories to the convention together at regular intervals – to get chrysotile asbestos included on the list of dangerous substances.

At the 6th conference in May 2013, these NGOs were out-manoeuvred by Russia, carrying with it countries of the former Soviet Union, India and Vietnam, to prevent chrysotile asbestos being put on the Convention list. Listing is done by consensus, and would not have meant the trade in chrysotile asbestos being outlawed; it would merely have placed an obligation on exporters to provide information on the risks of the product to the importing country.

The NGOs believe that the asbestos lobby hijacked the Convention whose credibility, they argue, is now on the line. They are working to draft an alternative international protocol that could effectively influence trade in asbestos and what the IBAS dubs its attendant miseries.

Addressing the fourth Conference of the Parties to the Rotterdam Convention as long ago as 2008, an Indian delegate working with workers and their families exposed to chrysotile asbestos said that the parties “had not disagreed to the science or the scientific process that conclusively proves this substance’s ability to harm (...) the opposition to its listing has been based purely on domestic commercial and political interests, undermining the larger public health and human rights interest (...) there will be so many more lives lost, uninformed and unprotected due to this deadly substance”⁴².

The blocking strategy successfully deployed by Russia and its allies for chrysotile asbestos could happen for other chemicals that also pose a threat to human health.

This is concerning, because the global risks from toxic substances come not only from “old” products and technologies but are central to the way we live now.

41. South Africa, Algeria, Australia, Argentina, Chile, Egypt, Israel, Japan and Turkey to name but a few.

42. Intervention at Plenary Session of COP4, October 31, 2008 by Madhumita Dutta, Rotterdam Convention Alliance delegate.

Global risks of the E-economy

The E-economy may be hazardous for workers who, as in India, China, California or Scotland's "Silicon Glen", manufacture printed circuit boards, computers and microchips. The micro-electronics industry employs about a million workers world-wide. It is a technology that uses highly intensive complex chemical processes. When National Semiconductor UK located in the small town of Inverclyde, near Glasgow, in the early 1970s it had a guaranteed rural female labour pool still deeply imbued with a patriarchal culture and lacking a trade union tradition. In the early 1990s, after several warnings had gone ignored, a handful of Scottish trade union activists met senior officials from the UK's health and safety inspectorate, the HSE. They explained the fertility problems and miscarriages experienced by women semiconductor industry workers, especially the "clean room" workers. A post-meeting survey of five semiconductor manufacturers in seven factories across the UK concluded that clean room work posed no risk to pregnant women. But three previous US studies had found evidence of an increased number of miscarriages among women clean room workers.

By 1996, the union was hearing complaints from male workers about health problems they believed were due to the chemicals they were handling. The toll rapidly rose to 60. They were unable to name the chemicals concerned, often knowing only the product brand names. The union decided to set up a support group, called Phase Two. The issue attracted media attention, which prompted the HSE to launch the first really independent study into the semiconductor industry. During this time, Phase Two collected personal accounts from more than 200 workers.

It received support from networks that had been formed two decades earlier in Silicon Valley, and from an American occupational medicine specialist. Together, they mounted the International Campaign for Responsible Technology, holding briefing meetings across Scotland. They were supported by a handful of academics who helped them puzzle out the scientific terminology. But, local health officials and GPs seemed uninterested in their actions. In 2001, the HSE finally acknowledged that the survey findings clearly pointed to an excess incidence of several types of cancers in the semiconductor industry.

The workers and their union now believe that had they not campaigned with help from the media and independent experts, the excess cancer incidence among workers in the UK semiconductor industry would have gone unremarked. The use of many carcinogens would have gone unregulated and uncontrolled. They also believe that the health and safety agency failed to fulfil its sentinel role. The industry approach focused on playing down and casting doubt on the information put out.

In 2012, the International Journal of Occupational and Environmental Health reported the difficulties faced by Korean researchers in investigating cases of cancer contracted by workers in a semiconductor production unit owned by the multinational Samsung (Lee and Waitzkin 2012).

A Korean NGO had publicised cases of cancer reported to it as occurring in the unit. The case caused a stir in Korea. Researchers persisted in digging deeper despite obstruction by Samsung and the South Korean authorities. They were able to document 17 cases of leukaemia and non-Hodgkin lymphoma diagnosed between 2007 and 2011 among workers in Samsung's Giheung factory (south of Seoul) (Kim *et al.* 2012). Lacking access to company data, however, they were unable to establish a causal link between the cancers and the semiconductor industry. In 2013, a victim support organisation (Sharps) provided the Korean government with a list of 180 cases of cancer and chronic diseases occurring among Samsung's young workers, including 104 in the semiconductors business line.

Risks are also present at the other end of the computer chain. These are all the more shocking for involving a particularly poor and uneducated population. 80% of the electronic

waste collected in North America is “recycled” in Asia, in primitive, dangerous and polluting conditions. Despite EU directives banning this kind of export trade, 60% of European electronic waste is thought to follow the same route. Nongovernmental organisations condemn the abuse of freedom of trade and the irresponsibility that allow the electronics industry to evade the social, health and ecological costs associated with its end-of-life products. They argue that consumers also need to be aware of these hidden costs. Men, women and even children work in makeshift shacks, sometimes in their own homes, trying to recover tiny amounts of a wide variety of often highly toxic materials (antimony, arsenic, cadmium, chromium, cobalt, lead, mercury, rare metals, etc.) from electronic waste.

Make toxic waste producers responsible

In the 1980s, the increased cost of processing hazardous waste in industrialised countries brought by regulations and legislation prompted a shift towards the developing countries. The 1989 Basle Convention initiated by the United Nations Environment Programme laid down the principles for controlling transfers of toxic waste, and organised a prior information system similar to that of the Rotterdam Convention. The Basle Convention came into effect in May 1992, and has been ratified by over 130 exporting and importing States as well as transit countries, including the European Union. But the signatory countries still have to observe and police what they have signed up to.

In August 2006, more than 500 tonnes of highly toxic waste stored in the hold of a chemical tanker, the *Probo Koala*, were dumped at several points in the city of Abidjan (Côte d’Ivoire). This fly-tipping of toxic waste reportedly caused the death of 17 people in Abidjan and led to tens of thousands of others being poisoned. “The *Probo Koala* affair is a clear case of violation of European and international law (...) It’s important to make sure that criminal cases like this will not go unnoticed and will not be repeated in the future,” said the-then European Environment Commissioner Stavros Dimas.

The *Probo Koala* was owned by a Greek shipping company, registered in Panama, and chartered by Trafigura, a company with its tax address in Amsterdam, registered office in Lucerne and operational centre in London... It was Russian-crewed and was carrying a mix of oil, hydrogen sulphide, phenols, caustic soda and organic sulphur compounds. The local Abidjan company that had offered to “process” the waste for 20 times less than that charged by a specialist company in the Port of Amsterdam was both recent and inexperienced.

In December 2011, Trafigura was fined a million euros on appeal in the Netherlands for the illegal exporting of waste. Trafigura paid out 200 million euros to compensate victims – much of which was misappropriated and never reached the intended recipients, claims Amnesty International⁴³.

The *Probo Koala* had a chequered six year history. It was initially destined for the breaker’s yard, but was put back into service under a different name, and is now reportedly to be broken up in China.

Cases like that of the asbestos-laden French vessel *Clemenceau*, sent to India to be broken up, but recalled in the face of strong international pressure, are still too few and far between.

43. Amnesty International (2011) Côte d’Ivoire: missing millions must reach Trafigura toxic waste victims, press release, 19 August 2011.

The lethal legacy of PCBs

While measuring levels of DDT – an insecticide now banned in many countries – in marine animals, Swedish chemist, Sören Jensen subsequently discovered other substances that are also pervasive in the environment: PCBs (polychlorinated biphenyls). Production of these in the EU was halted in 1986 after researchers found that 25% of the total world output of PCBs (2 million tonnes) had already accumulated in our environment, and will pollute rivers and oceans for a very long time due to their resistance to degradation. Freshwater fish in industrialised countries are so contaminated with PCBs that pregnant women, young girls and adolescents are advised to eat no more than one portion every two months.

Significant amounts of PCBs are still to be found in many electrical appliances, transformers or capacitors. Used PCBs are often contaminated with dioxin, and are costly to dispose of safely. If uncontrolled, there is a great danger of PCBs being disposed of fraudulently, not only to contaminate the environment, but also to enter the food chain on a large scale, as happened in Belgium in 1999 with the so-called “dioxin” scandal.

PCBs are endocrine disruptors, and some are now considered by the International Agency for Research on Cancer (IARC) as known human carcinogens, just like the dioxin that contaminates them.

Conclusion

The rise in cancer deaths seen after World War Two went together with increased life expectancy, prompting the long-held belief that it was a consequence of longevity.

Then, in the 1960s, epidemiological evidence implicating tobacco in the development of lung cancers put a focus on individual lifestyle causes of cancer, like smoking, drinking and poor diet. All these explanations had the political benefit of throwing responsibility for illness back onto the individual.

But examined closely, the rising cancer toll has tracked industrial development. Burning coal created the soot that caused chimneysweep's cancer. The development of carbon chemistry products – benzene, aromatic amines, polycyclic aromatic hydrocarbons – was to make exposure to carcinogens part of everyday life for industrialised country populations. Chlorine chemistry and petrochemistry would in turn lead to the creation of thousands of products, some of which are known to be mutagenic and cancer-causing. Occupational cancers are now an undeniable fact of life.

Despite the publication of studies evidencing excess cancer mortality among workers exposed to certain chemicals, the understanding that these cancers are not inevitable was too long in coming, and is still not satisfactory in industrialised countries, and even less so in developing countries. Bitter struggles are waged on pay, working hours and unemployment, rallying the mass forces of workers - work-related diseases and cancers have not drawn the same response. Barring the odd event like the sentencing of a former officer of asbestos-cement producer Eternit to 18 years in prison by the Court of Appeal in Turin on 5 June 2013, occupational cancers do not grab media headlines. Yet, with their attendant agonies, grief, and lives cut short, work-related cancers affect almost exclusively the most vulnerable workers. It is one of the great social injustices of our time. They should be tackled on the same basis as other inequalities, and top the policy agenda.

It can never be over-emphasised that occupational cancers are avoidable. The REACH regulation gives the opportunity for a new start. But it alone will not be a sure

recipe for improved working conditions. The key, here as elsewhere in health and safety at work, is the ability of trade unions to rally workers to take ownership of this debate. The workers on every factory-floor and in every company must be positively involved in the coming identification and assessments of workplace chemicals. They must unite to demand that the most toxic products be replaced, and if this cannot be done quickly, to demand working conditions that will give them the best possible protection.

Then, too, work must be done at the European level and in each country to secure better recognition and compensation for those occupational cancers that are bound to occur. All workers should have a certificate of exposure to carcinogens. They should also have a record that sets out the dates and reports of checks on their physical condition made while working. Any anomalies related to the carcinogenic agent or process should be noted in it. Finally, it is vital that they should be given health surveillance even after their working lives are finished.

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Appendice

Check-list for a trade-union assessment of workplace carcinogen hazards

Physical factors in the production cycle

- Carcinogens used
- Carcinogens related to the processing of physical agents used in production. E.g.: respirable wood dust in the furniture industry, crystalline silica in the building trade, fumes and vapours containing carcinogens
- Carcinogens in production processes/equipment. E.g.: use of a source of ionising radiation, filters with asbestos, use of diesel for transport, etc.
- Do not overlook "peripheral activities": maintenance and cleaning, storage, transport, etc. E.g.: cleaning metal parts with trichloroethylene

Environmental factors and work

- *From the environment to work.*
E.g.: asbestos in buildings, solar radiation on building sites, tobacco smoke in public places, contact with diesel engine exhausts, etc.
- *From work to the environment:*
discharges (liquids, solids, gases) that may be cancer-causing agents in the environment
- *From the product of work to the environment:*
carcinogens in the end production or a later phase of the end cycle of the end product; carcinogens related to the use of the end product

Work organisation factors

- Factors that may contribute to the development of some cancers: night work; contingent employment
- Factors that undermine prevention: conflict between productivity and safety; lack of information and training
- Problems created by use of temporary agency staff, subcontracting; other factors of insecurity

Organisation of prevention

- Compliance with the order of priority of preventive measures; regular evaluation of the situation and review of prevention plans to include the evaluation conclusions
- Activity of the preventive services: aptitudes (esp. toxicology, ergonomics and occupational medicine); professional independence; quality of the relations with workers' reps; quality of exposure measurements; quality of health surveillance
- Information on cancer-triggering factors, training, proper functioning of workers' health and safety representative bodies
- Systematically record exposures
- Take the gender dimension into account
- Take the health follow-up of previously-exposed workers into account

Taking the health surveillance data into account

- Check data on currently exposed workers. In particular, assess whether the health checks carried out are appropriate to the exposures and medical conditions that may develop: are there suitable biological indicators?
- Use information from outside the company: epidemiological research, data collected by sector, occupation or exposure by trade unions, research institutes or preventive services, outside contacts to collect information on carcinogens and possibilities of substitution
- Use data on previously exposed workers, check whether post-employment health surveillance is adequate, and its outcomes.

Incorporating cancer prevention in company policy decisions

- Production as process: how far are workers' health needs taken into account in decisions about the process?
- Production as end product: check whether the production is likely to create cancer hazards downstream of actual in-plant production. How much weight is given to the needs for health and safety at work and public health in the search for less dangerous alternatives?
- Create a bargaining position in the company and society: awareness-building campaigns; calling in the labour inspectorate; use of the right to stop work in the event of serious and imminent danger
- Incorporate the problems found in the strategy of demands and collective bargaining