GENERAL REMARKS

This ninety-second volume of *IARC Monographs* contains evaluations of the carcinogenic hazard to humans of 60 polycyclic aromatic hydrocarbon (PAH) compounds and several occupations involving exposures to coal-derived PAHs. This is the first of several volumes related to agents that contribute to air pollution; subsequent volumes will cover certain particulate or chemical agents, indoor emissions from household combustion of solid fuels and high-temperature frying, asphalt/bitumen, motor vehicle emissions, and ultimately outdoor air pollution. Many of the PAHs and occupations evaluated in this volume were last reviewed more than 20 years ago in Volumes 32–35, a four-part series on polynuclear aromatic compounds. Newer experimental and epidemiological information have been published since that time and are reviewed in this volume.

There is a long history to the identification of PAHs as human cancer hazards. In 1775 Sir Percivall Pott identified soot as the cause of scrotal cancer in chimney sweeps (Brown and Thornton, 1957), the first attribution of an occupational cancer to a specific cause. In the early 1900s laboratory scientists began the search that led to the isolation of benz[*a*]anthracene, dibenz[*a,h*]anthracene, benzo[*a*]pyrene, and other PAH compounds and the demonstration that they can induce cancer in experimental animals (Phillips, 1983).

The evaluations of carcinogenic hazard in this volume are qualitative assessments of the evidence that a PAH congener or mixture can increase the incidence of cancer. The same evidence also demonstrates that PAH congeners and mixtures vary widely in the level of carcinogenic response induced by a given dose. Although benzo[*a*]pyrene is the marker of PAH exposure that is most often used, there is evidence that a few PAH congeners, for example, dibenzo[*a,l*]pyrene, are more potent in their ability to induce lung cancer or skin cancer in experimental systems. These potent congeners should be measured in environmental and biological samples, as they may contribute substantially to the risk of human cancer attributable to PAH mixtures.

Some occupations evaluated in this volume may entail exposures to carcinogens other than coal-derived PAHs, and it is possible that the observed cancer risks could be attributable in part to these other exposures. In aluminium production, for example, there is an excess of bladder cancer, and potential confounding by other occupational exposures could not be ruled out with reasonable confidence.

The *IARC Monographs* use the term 'carcinogen' to denote an exposure that is capable of increasing the incidence of malignant neoplasms, reducing their latency, or increasing their severity or multiplicity. In reviewing bioassay data for this volume, the Working Group considered mouse skin initiation-promotion studies (single or multiple applications of a PAH followed by repeated applications of a tumour promoter such as 12-*O*-tetradecanoylphorbol-13-acetate) to be insufficient for assessing the carcinogenicity of PAHs *per se*. The Working Group also reviewed a number of newborn mouse assays in which the PAHs were administered to infant mice (e.g. days 1, 8, and 15 of life). Some of these newborn treatments led to neoplasms, which were in some instances malignant. The Working Group considered positive newborn mouse assays to be indicative of the genotoxic potential of the compound under investigation rather than as unambiguous proof of carcinogenic potential. The Working Group further felt that results of newborn mouse assays had to be viewed in light of other available data before making an assessment of the overall carcinogenic potential of an exposure.

A significant source of PAH exposure in the general population is the consumption of particular foods, notably toasted cereals and grilled meats. These foods contain measurable levels of benzo[*a*]pyrene and other PAHs that are *carcinogenic, probably carcinogenic,* or *possibly carcinogenic to humans*, and there is strong evidence that some of these compounds, including benzo[*a*]pyrene, induce digestive-tract tumours in experimental animals when administered by ingestion. There are, however, few epidemiological studies investigating directly the possible association between dietary PAH intake and cancer. The studies conducted to date used questionnaires about meat-cooking in conjunction with benzo[*a*]pyrene as a marker for total PAH intake. Three case-control studies observed a small-to-moderate increase in the risk of colorectal adenoma, a precursor of colon cancer, with higher estimated intake of benzo[*a*]pyrene. There was, however, no association with benzo[*a*]pyrene intake in a case-control study of colon cancer. One case-control study of pancreatic cancer also observed a moderate increase in risk with benzo[*a*]pyrene intake. There was no association with benzo[*a*]pyrene in one study of prostate cancer and non-Hodgkin lymphoma. This volume reviews the epidemiological studies that suggest a possible association between consumption of PAHs in foods and increased risks of colorectal adenoma and pancreatic cancer. These epidemiological studies, however, are limited to one geographical area and are too small to be considered conclusive. Large-scale independent cohort studies are needed to more definitively investigate these associations.

A summary of the findings of this volume appears in *The Lancet Oncology*.

References

Brown JR, Thornton JL. (1957) Percivall Pott (1714–1788) and chimney sweeper's cancer of the scrotum. *Occup Environ Med* 14: 68–70.

Phillips, DH. (1983) Fifty years of benzo(*a*)pyrene. *Nature* 303: 468–472.