5. Summary of Data Reported

5.1 Exposure data

A large proportion of the emissions generated during cooking is steam from the water contents of the food. However, during frying (with oil), fatty acid esters that make up edible oils and fat can decompose and produce volatile organic compounds, as well as semi-volatile compounds that can condense to form particles. A wide variety of organic compounds have been identified in cooking emissions, including alkanes, alkenes, alkanoic acids, carbonyls, polycyclic aromatic hydrocarbons and aromatic amines. The main volatile compounds generated during frying were aldehydes, alcohols, ketones, alkanes, phenols and acids. Of particular concern in relation to carcinogenicity are polycyclic aromatic hydrocarbons, heterocyclic amines and aldehydes. The contribution of commercial cooking operations to outdoor levels of polycyclic aromatic hydrocarbons can be substantial.

Cooking also increases the concentrations of fine and ultrafine particles.

The chemical composition of cooking emissions varies widely depending on the cooking oils used, the temperature, the kind of food cooked, and the method and style of cooking adopted.

5.2 Human carcinogenicity data

To examine the potential association between emissions from cooking oil and the risk for lung cancer, the Working Group considered studies to be more informative when cooking-related effects were separated from fuel-related effects and when the studies reported results on the exposure–response relationships between high-temperature frying (i.e. stir-frying, deep-frying and pan-frying) and lung cancer. Studies that only collected information on cooking habits (e.g. age at starting to cook, years of cooking), ventilation in the kitchen or frequency of eye irritation due to cooking or smokiness in the kitchen were considered to be less informative because they did not allow the effects of emissions from cooking oil to be distinguished from those of combustion products of cooking fuels.

On this basis, four case–control studies were considered to be the most informative. The study conducted in Hong Kong Special Administrative Region used a composite index that accounted for both the frequency and the duration of all three types of high-temperature frying; it found a significant threefold increased risk for lung cancer associated with moderate to high categories of exposure (>150 total dish–years) and an eightfold increased risk associated with the highest category (>200 total dish–years).

In the other three informative studies in Shanghai (two studies) and Gansu, China, the risk for lung cancer increased generally with increasing frequency of stir-frying, deep-frying and pan-frying and a nearly twofold increased risk was associated with the highest
frequency of high-temperature frying. In the study conducted in Gansu, however, the risk for lung cancer increased significantly with increasing frequency of stir-frying but not of deep-frying. However, potential confounding by solid cooking fuel could not be ruled out with reasonable confidence in these three studies. In the study from Hong Kong that compared risk (per 10 dish–years) for the three types of high-temperature frying, the magnitude of risk was highest for deep-frying, intermediate for pan-frying and lowest for stir-frying, but all were associated with a significantly elevated risk for lung cancer. In the studies in Shanghai and Gansu, the effects of the different types of frying were not mutually adjusted for and, because of the substantial differences in the frequency of stir-frying and deep-frying, a direct comparison of the risk estimates associated with an individual type of frying could not be made.

These four studies also provided information on the specific type of cooking oil. There was no significant difference in risk estimates for lung cancer with use of any particular type of cooking oil (peanut oil, corn oil or canola oil — a type of rapeseed oil) in the study in Hong Kong. In the three other studies, risk was higher for women who cooked with canola oil most frequently. Some increased risk was associated with cooking with linseed oil in the population-based case-control study conducted in Gansu and with cooking with soya bean oil in the study in Shanghai.

In summary, results from the four most informative studies demonstrate an exposure–response relationship between increased frequency of or cumulative exposure (frequency and duration) to high-temperature frying and increased risk for lung cancer. These four studies were conducted in different populations in Hong Kong, urban Shanghai (two studies) and rural Gansu where study characteristics differed, and where cooking practices and other co-factors may also have differed. However, confounding by cooking fuel could not be ruled out with reasonable confidence in the latter three studies. Furthermore, all epidemiological evidence was based on case–control studies and recall bias may have contributed to the positive findings in some of these studies.

5.3 **Animal carcinogenicity data**

Inhalation of high concentrations of emissions from high-temperature frying of unrefined rapeseed oil caused an increase in the incidence of lung carcinomas (mainly adenocarcinomas) in male and female mice in one study and female rats in another study.

5.4 **Mechanistic and other relevant data**

See also Section 5.4 in the monograph on household use of solid fuels.

The available information on the genotoxic and mutagenic activity of cooking oil fumes includes data from professional and home cooks that show the induction of 8-oxoguanine DNA glycosylase 1, which is a DNA repair enzyme that removes 8-hydroxydeoxyguanosine. In experimental animals, cooking oil-fume condensates from rapeseed and soya bean oils induced micronuclei in the bone marrow of both mice and
rats, oxidative DNA damage, enhanced transformation of tracheal epithelia and accumulation of TP53 protein. Cooking oil-fume condensate also induced chromosomal aberrations in the diploid male germ cells of mice. In cultured human or animal cells, cooking oil fumes from a variety of oils induced DNA adducts, DNA damage (comet assay), oxidative damage, sister chromatid exchange, chromosomal aberrations, unscheduled DNA synthesis and DNA cross-links. Cooking oil fumes induced DNA damage in naked calf thymus DNA.

Extracts or condensates of emissions from cooking oil fumes are mutagenic in Salmonella. In strain TA98, in the presence or absence of a metabolic activation system, the mutagenic potency in terms of revertants per milligram of particle reached several thousands or in terms of revertants per cubic metre of air reached several hundreds.

Several studies showed that the mutagenicity of cooking fumes in Salmonella was positively correlated with heating temperature, the extent of unsaturation and the concentration of unsaturated fatty acids. Polycyclic aromatic hydrocarbons and lipid peroxidation products also contribute to the mutagenic activity of cooking oil fumes.