

EMISSIONS FROM HIGH-TEMPERATURE FRYING (Group 2A)

For definition of Groups, see Preamble Evaluation.

VOL.: 95

5. Summary of Data Reported

[The text of these Summaries and Evaluations may be edited for language and clarity during the checking of the main text of the Monographs.]

5.1 Exposure data

Cooking with gas or electric stoves can elevate concentrations of fine and ultrafine particles, organic compounds, such as polycyclic aromatic hydrocarbons and heterocyclic amines adsorbed to those particles, and certain organic gases such as carbonyls (aldehydes and ketones). Even gas burners or electric heating elements alone (with no food) can create 10^{11} ultrafine particles per minute. Gas burners create extremely small ultrafine particles that peak at 10 nm, whereas electric toasters create particles that peak at 30 nm and an empty gas oven creates particles that peak at 45 nm. The addition of foods and multiple cooking processes increases the median diameter of fine and ultrafine particles and may increase the concentration by an order of magnitude. Cooking may be the single largest source of exposure to ultrafine particles.

The 24-h mass concentrations of fine particles due to cooking appear to be of the order of 2–8 $\mu\text{g}/\text{m}^3$, or about 25% of typical indoor mass concentrations in the USA, which may not be typical for developing countries. Short-term fine particle mass concentrations due to cooking are in the region of 50 $\mu\text{g}/\text{m}^3$. Frying, sautéing and grilling appear to produce a greater fine particle mass than other forms of cooking. Overall, cooking is the second largest source of indoor fine particle mass after tobacco smoking. The type of stove appears to make little difference in the particle concentrations produced.

Concentrations of polycyclic aromatic hydrocarbons due to cooking are also elevated, but are less well characterized. Although they have been found in cooking oils, the relative quantities produced by the oils compared with the quantities produced by both the cooking fuel and the food itself have not been determined. The contribution of commercial cooking operations to outdoor levels of polycyclic aromatic hydrocarbons has been found to be substantial. Carbonyls such as formaldehyde, acetaldehyde and acrolein are also produced by both residential and commercial cooking.

5.2 Human carcinogenicity data

To examine the potential association of 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 disentangled 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 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 increasing frequency or cumulative exposure (frequency and duration) from high-temperature frying and increasing 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

In two studies, inhalation of high concentrations of emissions from high-temperature unrefined rapeseed oil caused an increase in the incidence of lung carcinomas (mainly adenocarcinomas) in male and female mice combined and male and female rats combined. The exposure-response was significant in male and female mice and female rats but not in male rats.

5.4 Mechanistic and other relevant data

See also Section 5.4 in the monograph on combustion of coal and biomass.

The available information on the genotoxic and mutagenic activity of cooking oil fumes includes data in cooks and homemakers that show the induction of 8-oxoguanine DNA glycosylase 1, which is a DNA repair enzyme that removes 8-hydroxydeoxyguanine. *In vitro*, 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. *In vivo*, 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.

Extracts or condensates of emissions from cooking oil fumes are mutagenic in *Salmonella*. In strain TA98 in the presence of a metabolic activation system, the geometric mean potency in terms of revertants per milligram of particle is 115 for cooking oils. The mutagenic potencies of wood smoke emissions are five times higher than those of cooking oil fumes and those from smoky coal are five times higher than those from wood smoke emissions. Because of the different number of particles emitted from oil, wood and coal, the mutagenic potencies of these emissions expressed as revertants per cubic metre of air are 70 for cooking oil fumes compared with 1300 for emissions from the combustion of wood and 15 325 for emissions from the combustion of smoky coal.

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.

6. Evaluation and Rationale

There is *limited evidence* in humans that emissions from high-temperature frying are carcinogenic.

There is *sufficient evidence* in experimental animals that emissions from high-temperature unrefined rapeseed oil are carcinogenic.

Overall evaluation

Emissions from high-temperature frying are *probably carcinogenic to humans (Group 2A)*.

Rationale

Among the studies in humans, four were considered most informative because they allowed the effects of cooking-oil emissions to be distinguished from those of the fuels used for heating the stove. These studies, in four different populations, consistently showed an increased risk for lung cancer and showed an exposure–response relationship between increasing frequency or duration of high-temperature frying and increasing risk for lung cancer. Confounding by the fuel used to heat the stove could be ruled out with reasonable confidence in only one of these studies.

These epidemiological results are supported by the evidence from studies in experimental animals. Although positive results in experimental animals were observed only for unrefined rapeseed oil heated to high temperatures, positive results for mutagenicity were observed in virtually every category of in-vivo test. These mutagenicity data would have been enough to support an evaluation of Group 2A if the evidence of carcinogenicity in experimental animals had been less than *sufficient* or the evidence of carcinogenicity in humans had been less than *limited*. The mechanistic data also show that lipid peroxidation is an important mechanism that leads to carcinogenesis by these mixtures, although there may also be a small contribution from the mechanisms by which polycyclic aromatic hydrocarbons induce cancer (see Volume 92).

The evaluation was made for ‘emissions from high-temperature frying’. This wording was determined after considering several aspects of the available data.

The available studies involved frying at high temperatures. Emissions from low-temperature cooking methods can be considerably different from those studied. Data indicate that there is little mutagenic potential for cooking oil heated below 100 °C and high mutagenic potential above 230 °C.

No differences were apparent between stir-frying, deep-frying and pan-frying when these methods were investigated separately. Other high-temperature cooking methods (e.g. baking) were not included

because the Working Group reasoned that their emissions could be considerably different from those of frying.

The epidemiological data are not detailed enough to distinguish among different cooking oils and fats and experimental animal data were available for unrefined rapeseed oil only, although data are available that indicate a higher mutagenic potency for unsaturated fats.

The epidemiological data do not permit the risk to be attributed to a specific chemical compound or to the cooking oil alone. Some risk could be attributable to the food being cooked, to emissions from the heated stove or cooking vessel itself or to the fuel used to heat the stove. Nevertheless, it might be reasonable to attribute some risk to cooking oils, because in-vivo and in-vitro data indicate that emissions from some oils heated to high temperatures are mutagenic.