

6. Evaluation and Rationale

There is *limited evidence* in humans for the carcinogenicity of emissions from high-temperature frying.

There is *sufficient evidence* in experimental animals for the carcinogenicity of emissions from high-temperature unrefined rapeseed oil.

Overall evaluation

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

Rationale

Among the studies of cancer 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 increased frequency or duration of high-temperature frying and increased 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 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 cooking oil has little mutagenic potential when heated below 100°C and high mutagenic potential when heated 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 between 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.