

Letter to the Editor



Sir: Recently a paper by Gabriel et al. (1) was published on "The Biochemical and Histological Effects of Feeding Thermally Oxidized Rapeseed Oil and Lard to Rats." From their study the authors concluded that seborrhea, diarrhea, polyuria, excessive hair loss, liver and heart lipid changes, and injury (histological) to hearts, livers, and kidneys resulted from the feeding of the oxidized fats.

Over the years a number of studies have been published on the toxicity of oxidized fats and oils (2-7). The results of these studies depend largely on the methods used to prepare the fats. Some of the studies have been carried out on fats that have been heated under exaggerated conditions of temperature and aeration that bear little if any resemblance to even extreme home or commercial use conditions (2-4). Other studies have been conducted on concentrates of reaction products that actually occur in the used oils at low levels (5,6). In either case, the extrapolation of such studies as they relate to practical use conditions is unwarranted.

In contrast to the preceding studies, fats that were prepared under prolonged commercial frying conditions were shown to be without harmful effects when fed for 2 yr (7). The end point of the frying in this study (except for the one silicone-containing sample which was used for 216 hr) was a predetermined foam height: a typical indicator used in commercial frying to determine maximum useful oil life. Thus, the oils represented the extreme of practical commercial conditions. A slight decrease in absorption of the fat was the only effect attributed to the heating. The animals grew well, and histopathology conducted at the termination of the study showed no differences between the test and control animals. In another study, Lanteaume et al. (8) fed oil that had been heated in air at 200 C for 2-4 days or that had been used 60 times for frying potatoes. The oil was fed to rats for a period of 1 yr and the only effect was, again, a slight decrease in the absorption of the fat. Finally, fats heated to the extreme of commercial frying conditions have been shown to be without teratogenic or harmful reproductive potential (9,10).

In the study carried out by Gabriel et al (1) the authors claim that the heating conditions were selected in order to produce a fat whose toxicity was similar to that of used fat from commercial frying operations. It is noteworthy, however, that the fatty acid composition of the fats fed showed almost complete destruction of linoleic and linolenic acids with corresponding increases in the levels of oleic and stearic acids. In previous studies, lightly hydrogenated soybean oil that was subjected to actual frying conditions in air for 60 hr showed linoleic acid levels reduced from an initial level of 31% to a final value of 30%. Even after 126 hr of frying, the linoleic acid content of the oil was 30.5% (7). Similarly, the linoleic acid content of lard was reduced from 10% to 6% by 116 hr of frying. In a separate study, frying onion rings, scallops, and potatoes in hydrogenated soybean oil for 56 hr resulted in reducing the linoleic acid content of the fat from 32.1% to 29.6% (9). Thus, the fat prepared by Gabriel et al. showed extensive fatty acid degradation far beyond that of fat which was actually used for frying for many hours.

An additional question concerning the study carried out by Gabriel et al. (1) is raised by an apparent essential fatty acid (EFA) deficiency in the diets containing the oxidized fats (but not the fresh fats). The current NRC requirement

for essential fatty acids for male rats is 0.6% of the diet (expressed as linoleate) (11). Based on the analytical data obtained by Gabriel, the linoleate content of their low-erucic rapeseed diet was calculated to be 0.17% while the lard diet was 0.21% linoleate. The effect of this apparent EFA deficiency must particularly be questioned in view of the fact that several of the symptoms of such a deficiency are similar to those observed by the authors. Specifically, hair and skin changes as well as heart and kidney damage are both symptomatic of EFA deficiency and were observed in the study. The scaly skin on the feet and tail that was observed by the authors is particularly characteristic of EFA deficiency.

In view of the gross differences in the fatty acid compositions of fats prepared under prolonged commercial frying conditions and those prepared by Gabriel et al., it is apparent that the latter is not representative of used commercial frying fats. The apparent deficiency in essential fatty acids in the diets containing the oxidized fats raises further questions about the validity of the study.

In light of these considerations we must question the significance of this work as it relates to the condition of frying fats consumed in the home or in commercial establishments. It is our conclusion that the authors have reported on observations made on a laboratory-produced phenomenon bearing little or no relationship to the condition of fats and oils consumed by the public at large. We, therefore, urge that investigators performing comparable studies in the future examine carefully the composition of the lipids that are produced under laboratory conditions before making any extrapolations relating the observations of the investigation to the quality of frying fats that are used for actual frying of food.

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