

POLYGLYCEROL POLYRICINOLEATE**E number:** E 476**Recommendation:**

A re-evaluation of polyglycerol polyricinoleate is not necessary.

Chemical name/synonyms:

Glycerol esters of condensed castor oil fatty acids, polyglycerol esters of polycondensed fatty acids from castor oil, polyglycerol esters of interesterified ricinoleic acid, PGPR.

Chemical formula: -**EINECS number:** -**CAS number:** -**Functional Class:** Emulsifier.

Specification: The EU specification for these substances apply to the additive free of sodium, potassium and calcium salts of fatty acids, however these substances may be present up to a maximum level of 6% (expressed as sodium oleate).

Manufacture: Polyglycerol polyricinoleate is obtained by esterification of polyglycerol with condensed castor oil fatty acids.

Definition: Polyglycerol polyricinoleate consists of polyglycerol esters of interesterified fatty acids present in castor oil. It is insoluble in water and ethanol and soluble in ether.

EC specifications: E 476 Polyglycerol polyricinoleate [5].

Assay: -

Polyglycerols: The polyglycerol moiety shall be composed of not less than 75% of di-, tri- and tetraglycerol and shall contain not more than 10% of polyglycerols equal to or higher than heptaglycerol.

In addition the specification includes purity criteria on Hydroxyl value, Acid value, Arsenic, Lead, Mercury, Cadmium and Heavy metals.

JECFA specifications: Polyglycerol esters of interesterified ricinoleic acid [4].

Assay: -

Polyglycerols: The polyglycerol moiety shall be composed of not less than 75% of di-, tri- and tetraglycerol and shall contain not more than 10% of polyglycerols equal to or higher than heptaglycerol.

In addition the specification includes purity criteria on Arsenic and Heavy metals.

Exposure: Polyglycerol polyricinoleate is permitted only in cocoa-based confectionary, including chocolate up to 5 g/kg and in some fat-products up to 4 g/kg. I takes 90 chocolate with 5 g and 112,5 g fat product with 4 g additive to reach the ADI of 7.5 mg/ kg bw.

In the EU monitoring system polyglycerol polyricinoleate was examined at tier 2 level and the calculated intake by adults and the whole population is reported in the range of 4 - 33% of ADI, hile the calculated intake by young children is reported by one member state as 49 - 53%. It was concluded that no further examination was needed at this stage.

SCF/JECFA evaluation:

SCF status: An ADI 0-7.5 mg/kg bw was established in 1978 [3]. The basis was not specified but it may be that the 1973 JECFA ADI was endorsed. The Committee noted that rats fed the substance at dietary levels of 18% developed a hepatomegaly, which was reversible and not associated with any significant abnormalities in the enlarged livers. Otherwise no toxicological data were specified.

JECFA status: An ADI 0-7.5 mg/kg bw was established in 1973 [1]. The basis was a study in rats with a level causing no toxicological effects on 15000 ppm (1.5%, highest dose) equivalent to 750 mg/kg bw in a long-term study in rats. The safety factor was 100. In other long-term studies liver and kidney enlargement was noticed at higher doses, 5 and 10%.

Background data:

Subacute/subchronic toxicity: No obvious adverse effects. The liver hypertrophy can be regarded as a normal functional response to an increased hepatic work load.

Short-term studies are available in rat and chicken. Liver enlargement was noticed in two (same author) of eight studies in rats. Enlarged kidney and liver was reported in the only study conducted in chicken. These effects were not accompanied by effects as revealed by histopathology. In some special studies on the liver enlargement the effects were reversible in mice and not proportional to the feeding level. The hypertrophy was regarded as a normal functional response to an increased hepatic workload. No hyperplasia was observed. No other effects were demonstrated [2].

Genotoxicity: -

Chronic toxicity/Carcinogenicity: No evidence of carcinogenicity. The liver hypertrophy can be regarded as a normal functional response to an increased hepatic workload. Long-term studies are available in mice and rats. The long-term studies in rats and mice did not show carcinogenic potential. The enlargement of liver and kidneys observed in long-term tests was not accompanied by any lesions detectable by histopathology. Only the rat study showed a noeffects level for liver enlargement, which was 1,5%, in the diet. The kidney lesions were not followed by histopathological findings [2].

The significance of the findings in kidneys is not possible to predict. Carcinogenicity studies in rats and mice showed no carcinogenicity at 5% in the diet (highest dose). No kidney or liver effects were reported [8].

Reproduction toxicity: A three-generation study was carried out in rats. No adverse effect were noted even at the highest dose i.e. 1.5% [2]. One recent communication deals with teratology by suggesting that this additive may increase the sex ratio (proportion male). This may be caused by glycerol that may be involved in the process of sex selection [7]. However, this hypothesis is not supported by recent metabolism studies [6]. A three-generation reproduction study in rats showed no effects on breeding performance at 1.5% in diet (highest dose) [9].

Effect in humans: A study is available in human volunteers eating up to 10 g/day for 2 weeks. This study showed no adverse effects [2]. A recent study in human volunteers showed no adverse effects at intake of 10g/day for 2 weeks (highest dose) [10].

Other: A study where rats were fed diet containing 18% of the substance (no dosing period specified) is available. It revealed the development of reversible hepatomegaly. No histopathological abnormalities of the enlarged livers were found. The hepatomegaly was not associated with hyperplasia [3].

Biochemical aspects: *In vitro* and *in vivo* studies are available. Radiolabel from orally administered substance was found in faeces, urine, and CO₂. It was suggested that the label in faeces was present as free polyglycerols, indicating hydrolysis in the gastrointestinal tract [2]. A thorough study on the metabolism in rats is published recently. Ingested polyglycerol polyricinoleate is extensively digested in the intestinal tract to its two major polymeric components: the polyglycerols, which are quantitatively excreted unchanged, and polyricinoleic acid that is degraded to ricinoleic acid that is absorbed and readily metabolised. Data show no evidence of tissue storage of its two major components [6].

Conclusion:

The toxicological data available to JECFA in 1973 or to SCF in 1977 did not include all the data, which are normally required for an ADI to be set for a food additive. However, later data confirm the safety of the substance within the ADI, and taken together with the limited exposure, there seems to be no need for an re-evaluation. Polyglycerol polyricinoleate as defined by the specifications seems to be covered by the toxicological evaluation.