Guar gum

Ingredient synonym names

Burtonite V-7-E
Cluster Bean (Cyamopsis spp.)
Cyamopsis psoraloides
Cyanopsis tetragonoloba L
Guar Flour
Guar Powder
Guar gum depolymerised
Gum Cyamopsis
Gum Guar
Jaguar
Regonol

IDENTIFIER DETAILS

Ingredient chemical structure

CAS Number	FEMA Number	Additive Number	Ingredient EC Number
9000-30-0	2537	E412	232-536-8

CAS Additional Number	FL Number	CoE Number
- 57406-68-5 57406-71-0 63799-54-2 85510-16-3 9008-17-7 9010-50-8 9049-33-6 9066-07-3 68411949 78615-64-2	-	166

Chemical formula: galactomannan composed of a straight chain of D-mannose with a D-galactose side chain at approximately every other mannose unit

Ingredient CLP Classification

Ingredient REACH Registration Number

-

Acute Oral Toxicity	Eye Damage/Irritation	Carcinogenity
0	0	0

Acute Dermal Toxicity	Respiratory Sensitisation	Reproductive Toxicity

Guar gum

0	0	0
Acute Inhalation Toxicity	Skin Sensitisation	Aspiration Toxicity
0	0	0
Skin Corrosive/Irritant	Mutagenicity/ Genotoxicity	Specific Target Organ Toxicity
0	0	0

SPECIFICATIONS

Melting Point	-	Boiling Point	-

STATUS IN FOOD AND DRUG LAWS

Acceptable Daily Intake (ADI, mg/kg)	No data provided
Acceptable Daily Intake (ADI) comments	No data provided
FDA Status	

CoE limits - Beverages (mg/kg)	No data provided	CoE limits - Food (mg/kg)	No data provided	CoE limits - Exceptions (mg/kg)	No data provided

HUMAN EXPOSURE

Ingredient Natural Occurence (if applicable)

Guar gum is derived from the seed of the guar plant, Cyamopsis tetragonolobus, a leguminous plant which resembles the soybean plant. India and Pakistan are the major sources of supply, although the U.S. is also a producer. The guar is a hardy and drought-resistant plant, which grows 3 to 6 feet high with vertical stalks. The guar pods, which grow in clusters along vertical stems, are about 6 inches long and contain 6 to 9 seeds. The endosperm, which comprises 35 to 42% of the seed, is the source of the gum [Fenaroli, 1995]

References - Ingredient Natural Occurence

Fenaroli (1995) Fenaroli's Handbook of Flavor Ingredients, Volume I 3rd Edition, 1995.

Ingredient Reported Uses

Guar gum is reportedly used in foods e.g. baked goods at 1.96 ppm, breakfast cereals at 11.26 ppm, cheese at 7.83 ppm and processed vegetables at 10.75 ppm [Fenaroli, 1995]. Guar gum is widely used as a food stabiliser. [NTP, 2002]

References - Ingredient Reported Uses

Fenaroli (1995) Fenaroli's Handbook of Flavor Ingredients, Volume I 3rd Edition, 1995.

NTP (1982). Carcinogensis bioassay of guar gum in F344 rats and B6C3F1 mice (feed study). National Toxicology Program Technical Report Series Vol: 229 (1982) 114

TOXICITY DATA

In Vivo Data

Acute Toxicity Data

6770mg/kg, Oral - Rat
9400 mg/kg, Oral -Rat
8100 mg/kg, Oral - Mouse
7000 mg/kg, Oral - Rabbit
6000 mg/kg, Oral - Hamster
Sax (1984). Dangerous Properties of Industrial Materials, 6th Edition. Ed. N.I.Sax. Van Nostrand Reinhold Company, New York

In Vivo Carcinogenicity/Mutagenicity

In a tumour inhibition study, male Sprague-Dawley rats received an oral dose of 10% Guar gum in a fibre-free diet for 24 weeks, followed by 12mg/kg bw 1/wk 1,2-dimethylhydrazine [a known carcinogen] for 8 weeks [the study was a total of 32 weeks). The authors concluded that there were no significant differences between groups for bodyweight and that Guar gum did not significantly reduce the incidence of rat colon adenocarcinoma. [Heitman et al., 1992].

A summary of the chronic toxicity and carcinogenicity data, as described by the EFSA ANS Panel, is presented below (EFSA, 2017). This data was presented and analysed by the National Toxicology Program (NTP, 1982):

Groups of B6C3F1 mice (50 animals/sex per day) were fed diets containing 25,000 and 50,000 mg guar gum/kg diet (equivalent to 3750 and 7500 mg/kg bw/day), for 103 weeks (NTP, 1982, Melnick et al., 1983). After week 20 of treatment, the mean body weights of females from the highest dose group were statistically significant lower than those of untreated controls. In treated mice of either sex, feed consumption was statistically significant lower than controls. Male mice from both treated groups showed a statistically significant lower incidence of hepatocellular carcinomas than controls. However, no statistically significant differences in hepatocellular adenomas incidences were reported in male mice. The combined incidence of hepatocellular adenomas or carcinomas in male mice remained statistically significantly lower in the highest dose group compared to controls. In this study, female mice did not show any statistically significant increases in neoplasms. Guar gum was considered by the authors as not carcinogenic in mice under the conditions of this bioassay (NTP, 1982).

Groups of F344 rats (50 animals/sex per dose) were fed diets containing 25,000 and 50,000 mg guar gum/kg diet, which is equivalent 1250 and 2500 mg/kg bw/day for 103 weeks (NTP, 1982; Melnick et al., 1983). No substance-related effects on survival or clinical signs were reported. After week 40 of treatment, mean body weights of females from the highest dose group were statistically significant lower than those of untreated controls. In female rats receiving 50,000 mg/kg diet, the weight gain was depressed by 13 % compared to the controls. Male rats did not show a marked depressed weight gain at the end of the study. In treated rats of either sex, feed consumption was statistical significantly less than controls. Upon histopathological examination, the only major finding in male rats was statistically significant increased incidences of adenomas of the pituitary gland as compared to control animals from the study. The increased incidences showed an apparent dose-dependency. However, the same male rats showed decreased incidences of carcinomas of the pituitary gland in both treated groups as compared to control animals from the study. The decreased incidence of carcinomas in the pituitary gland was statistically significant in the intermediate dose animals. When both incidences of adenomas and carcinomas of the pituitary

gland were considered together, no statistically significant differences were observed compared to controls animals from the study. Other neoplasms, non-neoplastic degenerative and inflammatory lesions were observed in male rats but they were reported as common of the type, incidence and distribution observed in aging rats of this strain and in control animals. Female rats did not show statistically significant increases in adenoma or in carcinoma incidences of the pituitary gland. The only major finding in female rats from both treated groups was an increased incidence of benign phaeochromocytomas as compared to control animals of the study. However, no changes in the incidences of malignant phaeochromocytomas were reported and the combined incidences of benign and malignant phaeochromocytomas in these animals were not statistically significant. Male rats did not show any statistically significant incidence differences on phaeochromocytomas of the adrenal gland. Guar gum was considered by the authors as not carcinogenic in rats under the conditions of this bioassay (NTP, 1982).

Overall, the EFSA ANS Panel considered guar gum as not carcinogenic (EFSA, 2017). The EFSA ANS Panel derived a NOAEL of 2500 mg/kg bw/day, the highest dose tested from the rat study and a NOAEL of 7500 mg/kg bw/day, the highest dose tested, from the mice study.

References - In Vivo Carcinogenicity/Mutagenicity

Heitman et al. (1992). Dietary supplementation with pectin and Guar gum on 1,2-dimethylhydrazine-induced colon carcinogenesis in rats. Carcinogenesis 13(5), 815-818.

NTP (1982). Carcinogensis bioassay of guar gum in F344 rats and B6C3F1 mice (feed study). National Toxicology Program Technical Report Series Vol: 229 (1982) 114.

EFSA (2017). Mortensen, A., Aguilar, F., Crebelli, R., Di Domenico, A., Frutos, M. J., Galtier, P., Gott, D., Gundert-Remy, U., Lambre, C., Leblanc, J.C., (2017). Re-evaluation of guar gum (E412) as a food additive. EFSA journal, 15(2).

Melnick, R. L., Huff, J., Haseman, J. K., Dieter, M. P., Grieshaber, C. K., Wyand, D. S., Russfield, A. B., Murthy, A.S.K., Fleischman, R.W., Lilja, H. S., (1983). Chronic effects of agar, guar gum, gum arabic, locust-bean gum, or tara gum in F344 rats and B6C3F1 mice. Food and Chemical Toxicology, 21(3), 305-311.

Dermal Toxicity

No data identified.

References - Dermal Toxicity

No data identified.

Reproductive/ Developmental Toxicity

Olejeme et al., (1992) studied the effect of cellulose, wheat bran and Guar gum on the development of rat foetuses and weaned offspring. The rats were separated into nine groups of six rats. Three groups received a diet supplemented with Guar gum at 5%, 10% and 15% respectively. On day 16 of the study, a sample of each group was sacrificed and the foetuses and placentas were removed, weighed and examined. The remainder of the dams were allowed to deliver and nurse their offspring until weaned. Of those rats receiving Guar gum at 10% or more, the weight gain of the dams was significantly lower than other groups. The total number of ossification centres in the foetal skeleton of those pups from the two higher Guar gum groups was significantly lower than the other groups. However, few pups from those 2 groups survived until weaning compared with groups receiving different dietary supplements. [Olejeme et al., 1992].

A summary of reproductive and developmental data, as described by the EFSA ANS Panel, is presented below (EFSA, 2017):

Pregnant CD-1 mice (18 to 21 animals/group) were treated by oral gavage once daily from gestation day (GD) 6 to 15 with doses of 8, 37, 170 and 800 mg/kg bw/day of guar gum in corn oil (FDRL, 1972). Six out of 19 mice dosed with 800 mg/kg bw/day died and three litters were lost. At necropsy on GD 17, the dams appeared to be completely normal and the number of implantations and live foetuses was comparable to the control group. The surviving dams of the 800 mg/kg bw/day group appeared to be completely normal. Doses up to 170 mg/kg bw/day had no noticeable effects on implantation or on maternal and foetal survival. The numbers of live or dead foetuses, the average implantation sites and foetal weights did not differ among the groups. The sex distribution of foetuses was not affected by the treatment. In the two highest dose groups, the percentages of litters with resorptions were increased. The number of abnormalities observed in the skeletons at foetal pathological examination of the guar gum treated groups did not differ from the number in the control dams. Moreover, no abnormalities were observed in the soft tissues of the foetuses examined from the treated groups. The EFSA ANS Panel noted that the effects related to developmental toxicity, mainly resorptions, foetal death and retardation, were due to the administration of guar gum at the maternal lethal level, being the highest dose tested (800 mg/kg bw/day) (EFSA, 2017).

Pregnant Wistar rats (20 to 24 animals/group) were treated by oral gavage once daily from GD 6 to 15 with doses of 9, 42, 200 and 900 mg/kg bw/day of guar gum in corn oil (FDRL, 1972). At necropsy on GD 20, animals receiving doses of up to 900 mg/kg bw/day appeared to be completely normal and there were no noticeable effects on implantation or on maternal and foetal survival. The numbers of live or dead foetuses, resorptions, average implant sites and foetal weights did not differ among the groups. Furthermore, the sex distribution of foetuses was not affected by treatment. The number and type of variants and abnormalities seen at foetal pathological examination of the guar gum groups in either soft tissues or skeletons did not differ from the number in the control group.

Guar gum was added in the diet of male and female Osborne–Mendel rats at 1 %, 2 %, 4 %, 7.5 % or 15 % for 13 weeks before mating (Collins et al., 1987). This is equivalent to 700, 1400, 2700, 5200 and 11,800 mg/kg bw/day of guar gum. The treatment was continued during mating and throughout gestation. After mating was confirmed, pregnant females were separated in groups of 38 to 42 animals per treatment group. The dams were sacrificed on day 20 of gestation. There were no treatment related effects upon female mating. No changes in behaviour and mortalities were reported, and the clinical findings were unremarkable. During gestation, feed consumption was reduced in all treated groups compared to control animals. The reduction in feed consumption was only statistically significant in the 2700 and 5200 mg/kg bw/day dose groups and was not dose-related. Maternal body weights were significantly decreased in the 2700, 5200 and 11,800 mg/kg bw/day groups after 13 weeks of treatment, however the EFSA ANS Panel considered that only the effects in the two highest doses were biologically relevant. No effect on the number of pregnant females or sex ratio was observed in the treatment groups when compared to the controls. The dams fed 11,800 mg/kg bw/day guar gum had a slightly, but statistically significant lower number of corpora lutea and implantations than in the control group, however no effect on implantation was observed. Furthermore, there were no effects on the soft or hard tissues at foetal pathological examination. The EFSA Panel identified a NOAEL for general toxicity of 2700 mg/kg bw/day, a NOAEL of 5200 mg/kg bw/day for fertility effects based on a decreased number of corpora lutea and a NOAEL for developmental toxicity of 11,800 mg/kg bw/day, the highest dose tested (EFSA, 2017).

Pregnant golden hamsters (20 to 21 animals/group) were treated by oral gavage once daily from GD 6 to 10 with doses of 6, 28, 130 and 600 mg/kg bw per day of guar gum in corn oil (FDRL, 1972). At necropsy on GD 14, animals receiving doses of up to 600 mg/kg bw/day appeared to be completely normal and showed no noticeable effects on implantation nor on maternal and foetal survival. The numbers of live or dead foetuses, resorptions, average implant sites or foetal weights did not differ among the groups. The sex distribution of foetuses was not affected by the treatment. The number of abnormalities seen in either soft tissues or skeletons at foetal pathological examination of the guar gum-treated groups did not differ from the number in the control group.

From a dietary combined fertility and developmental 13 weeks toxicity study in female and male Osborne–Mendel rats (Collins et al., 1987), the EFSA ANS Panel identified a NOAEL for general toxicity of 2700 mg/kg bw/day, a

NOAEL of 5200 mg/kg bw/day for fertility effects and a NOAEL for developmental toxicity of 11,800 mg/kg bw/day (EFSA, 2017).

In an FDA sponsored study, Guar gum showed no clear evidence of teratogenic potential on gross examination of chicken embryos. However, a LD50 of 0.05 mg Guar gum dissolved in water per egg was established when it was injected into the yolk sac at 0 hours [Verrett et al., 1980]. The authors do not discuss the significance of these findings.

References - Reproductive/ Developmental Toxicity

Collins et al., (1987). Study of the teratogenic potential of Guar gum. Food and Chemical Toxicology. 25 (11), p807-814.

Olejeme et al. (1992). Effects of different types and levels of dietary fibre on foetal development of rats. FASEB J. 6 (5), A1941.

Verrett et al. (1980). Toxicity and teratogenicity of food additive chemicals in the developing chicken embryo. Toxic. Appl. Pharmac., 56, 265.

EFSA (2017). Mortensen, A., Aguilar, F., Crebelli, R., Di Domenico, A., Frutos, M. J., Galtier, P., Gott, D., Gundert-Remy, U., Lambre, C., Leblanc, J.C., (2017). Re-evaluation of guar gum (E412) as a food additive. EFSA journal, 15(2).

FDRL (Food and Drug Research Laboratories), (1972). Teratologic evaluation of FDA 71-16 (guar gum) in mice, rats, hamsters. Unpublished report, final report prepared under dhew contract no. FDA 71-260. Maspeth, NY. Pb-221 800.

Inhalation Toxicity

A standard textbook also states that occupational asthma has been reported in people working in the industrial production of Guar gum [Leung et al., 1996].

Bush et al., (1990) cite a paper by Malo, which describes Guar gum as having a high molecular weight and therefore being associated with occupational rhinitis and asthma, with 5-8% of carpet factory workers sensitive to Guar gum. Bush et al. therefore question the importance of Guar gum as an occupational hazard in the food industry [Bush et al., 1990].

References - Inhalation Toxicity

Bush et al. (1990). Occupational asthma from vegetable gums. J. Allergy Clin. Immun., 86, 562.

Leung et al., (1996). S.Encyclopedia of Common Natural Ingredients Used in Food, Drugs and Cosmetics. New York, NY. John Wiley & Sons, Inc. 1996. 290.

Cardiac Toxicity

No data identified.

References - Cardiac Toxicity

No data identified.

Addictive Data

References - Addictive Data

No data identified.

Behavioral data

No data identified.

References - Behavioral data

No data identified.

In Vivo - Other Relevant Studies

Sprague Dawley rats fed a diet of 5 % guar gum partially hydrolysed guar gum and cellulose and other dietary fibres for a period of three weeks revealed guar gum to cause a significant decease in food intake, weight gain and increase in liver weight. Additionally epidydimal adipose tissue weight was significantly higher in guar gum fed rats than those fed cellulose. No significant effect was noted on serum lipids however, serum IgG was significantly lower than those animals fed cellulose. IgA and IgG productivity in mesenteric lymph node (MLN) lymphocytes were reported to be significantly higher in rats fed guar gum, glcomannan and pectin (in comparison with those fed cellulose) but Ig productivity in spleen lymphocytes was not reported to be marked. Guar gum was the only substance reported to significantly increase IgM productivity in MLN lyphocytes when compared with rats fed cellulose. These results were reported to suggest ‘ enhancement of the immune function by dietary fibre is mainly expressed in the gut immune system’, [Yamada et al., 2003].

In a petition made to EFSA, data from a 90-day toxicity study for two oxidised depolymerised guar gums prepared by alkaline hydrolysis was presented. Although these studies were performed using guar gum products produced under different conditions of manufacture than those of the present opinion the petitioner indicated that the very similar structure of the depolymerised guar gum products to native guar gum suggested that toxicological evaluations of the products manufactured much earlier were still relevant for the current product. Specifications on these two preparations were provided by the petitioner and included moisture (11.8 and 13.7%), crude protein (3.6 and 2.8%), crude fibers (2.2 and 1.7%), ash (1.7 and 4.7%) lipids (0.11 and 0.09%), gum content (91.4 and 89.5%) and negative for peroxide. These partially depolymerised guar gums prepared by alkaline hydrolysis were added to the daily diet of male and female weanling rats at levels of 0 (control), 20 or 50 g/kg foodestimated to amount to doses of respectively 0, 1000 or 2500 mg/kg bw/day for a period of 90 days. Growth, food consumption, chemical, clinical and histopathological examination of theexposed animals indicated that no adverse effects were observed that were attributable to the test substance (EFSA 2007).

In summary significant safety data on guar gum have been published. These include a 103-weekcarcinogenicity study in rats and mice up to 50 g/kg in the diet, which was preceded by a subchronic study at doses up to 100 g/kg diet, and a developmental toxicity study in rats up to150 g/kg diet (NTP 1982a; Melnick et al. 1983). Guar gum when given to F344/N rats or B6C3F1 mice at dietary levels of 25 or 50 g/kg (amounting to about 1250 or2500 mg/kg bw/day for rats and 3600 and 7200 mg/kg bw/day for mice) for 103 weeks did not induce cancer (NTP, 1982a; Melnick et al., 1983). The No Observed Adverse Effect levels for each of these studies was the highest dose tested (EFSA 2007).

Nicolosi et al (2001) discussed several dietary cardiovascular disease risk factors, in particular the association of increased cholesterol and an increased risk of coronary heart disease, and the importance of dietary intervention. They cite a paper by Anderson et al (1994) that demonstrates the significant cholesterol-lowering effect of dietary Guar gum in rats [Nicolosi et al, 2001].

Tobacman et al., (2000) Investigated the consumption of carrageenan and other water-soluble polymers commonly

used as food additives, and their association with the incidence of mammary carcinoma. The authors concluded that there was no significant positive correlation between the increased consumption of Guar gum and mammary carcinoma. [Tobacman et al 2000]

Guar gum taken as a supplement [as opposed to smaller amounts used as a food additive] is thought to increase the viscosity of the bowel content, resulting in the feeling of postprandial fullness, and therefore may reduce appetite. Clinical trials suggest that Guar gum may lower body weight and the medical literature suggests oral treatment for overweight patients. Guar gum containing preparations are freely available as slimming aids, both in the USA and Europe. However, a meta-analysis of randomised trials suggests a non-significant difference between patients receiving Guar gum and those receiving a placebo. Eleven trials were statistically pooled out of the 34 trials identified by the authors, and they point out that 3% of patients dropped out of the trials because of the adverse effects produced by Guar gum supplements. These include abdominal pain, flatulence, diarrhoea and cramps. No information is given regarding the dose level or frequency of the Guar gum supplements taken [Pittler & Ernst, 2001].

The ministry of Health, Labour and Welfare recommend Japanese people to intake a certain amount of dietary fiber, believing that incorporating more dietary fiber into the diet can reduce the risk of colorectal cancer. During two fourteen-day sessions in spring and autumn, 9 healthy female students took the same diets. During the first session, the students were provided a strict dietary formula, while during the second session, they were administered an 12.5 g/day of partially hydrolysed guar gum (PHGG, purity 80%, equivalent to 10 g of dietary fibre) dissolved in adequate amount of water at the end of each meal. There were no adverse reactions to the treatment reported. PHGG intake resulted in increase of the fecal bulk for 4 subjects and fecal moisture for 5 out of 9 subjects, but decreased fecal hardness in 3 subjects; the benefit of bowel movements provided by the PHGG intake, however, varied greatly among the subjects [Sakata et al., 2006] .

High-fibre diets have been associated with effecting the caloric intake and bioavailability of essential minerals and trace elements. Guar gum is often used as a thickener in infant formula milk. Using a dialysis machine calibrated to reflect the digestive capabilities of infants, Guar gum has been shown to decrease the availability of calcium, iron and zinc [Bosscher et al. 2001].

A brief metabolic study demonstrates the above affect on iron absorption in rats. The absorption of radiolabelled iron by jejunal segments of both normal and iron deficient rats was inhibited by an oral dose of Guar gum. The highest dose administered, 30 mg, inhibited absorption by 25% [Woelbling et al, 1980].

Grizard et al (2001) studied insulin and corticosterone level changes produced by Guar gum. Three groups of 9 male Wistar rats were fed different experimental diets for 3 weeks. These diets consisted of AIN-76 formulations in which 10 % [w/w] wheat starch was replaced with either sucrose, Guar gum or LMM-pectin at the same concentration. For those rats receiving the Guar gum diet, a significant growth rate reduction was noted [by 24%] compared with controls. This is likely to have been brought about by the effects of dietary Guar gum on lipid absorption. Blood triacylglycerine was strongly reduced [by 76% compared with controls] and total cholesterol was reduced by 39%. In addition, postprandial insulin concentrations were significantly reduced without altering postprandial glucose levels and glucagonemia was up-regulated. The authors discuss the mechanisms of action of Guar gum to produce these effects demonstrating that they are all linked to the bulk & high viscosity of Guar gum modifying the diffusion of nutrients. They also cite Morgan et al (1979) who shows that humans fed Guar gum also have a resulting decrease in insulin secretion and production of gastric inhibitory polypeptide [Grizard et al., 2001].

A similar study to that of Grizard et al., (2001) demonstrated that a diet including Guar gum decreases the absorption of trichloroethylene [TCE] in rats. In this instance by 12 % compared with groups fed similar meals. The rats were fed a meal of Guar gum – docosahexaenoic acid for 3 weeks and then given a single oral dose of 100 mg TCE. Analysis was then carried out 10 hours later [Nakashima et al. 2001].

Another study investigating the dietary effects of Guar gum in human volunteers demonstrates that no significant effect on either systemic appearance of dietary amino acids, or plasma and urinary dietary urea kinetics, occurred following the consumption of 15N-labelled protein meal [30 g soy protein] containing 1% high-viscosity Guar gum. However, there was an early effect on endogenous urea kinetics [Mariotti et al., 2001].

Following the inclusion of up to 59.6 g/day Guar gum into the diet of 6 insulin dependent diabetic volunteers, a small reduction in cholesterol levels occurred. Guar gum was taken daily for 4-6 weeks and the results compared against the previous 2 weeks as a control [Carroll et al., 1981].

A standard text book states that in women, ingestion of Guar gum resulted in permanent weight loss without effecting serum lipids in hypercholesterolemia, and that similar observations occurred in both male & elderly patients. However, in diabetics, positive results are reported in use of Guar gum as a long-term dietary supplement, in control of hypercholesterolemia. The text also reports that long-term administration [21g/day] of Guar gum produced a sustained improvement of Type 2 diabetes, significantly lowering serum total and LDL cholesterol concentrations. On average, total cholesterol was reduced by 14 % immediately before meals as well as by 16% in postprandial glucose levels. The text also refers to a human study on patients with diabetes mellitus, where Guar gum consumption did not adversely affect mineral balance. However, flatulence has been commonly reported as a Guar gum side effect [Leung et al., 1996].

A study to develop a colon-specific delivery system for mesalazine (5-ASA) using a guar gum carrier using X-ray imaging revealed that the matrix tablets reached the colon and was not subjected to disintegration in the upper region of the GI system in all of the 8-subjects involved, [Tugcu-Demiroz et al., 2004].

A study in which the effects of partially hydrolysed guar gum, (PHGG) in yogurt, (containing 6g PHGG) on the elevation of postprandial serum lipid levels, (11-male subjects) suggested that their was ‘potential for PHGG to reduce the risk of hyperlipemia’, [Kondo et al., 2004].

An in vivo pharmacolkinetic study (in humans) revealed that a guar gum-based colon-targeted formulation did not release the drug in the stomach and small intestine, but delivered it to the colon which resulted in the slow absorption of the drug therefore making it available for local action in the colon, [Krishnaiah et al., 2003].

All depolymerised guar gum preparations have average molecular weights that match the criteria set for the molecular weight of food grade guar gum specified to be between 50 000 g/mol and 8 000 000 g/mol by the EU and FAO/WHO/JECFA. The galactose to mannose (G/M) ratio of all preparations is typically between 0.5 and 0.66 and there was no difference in G/M ratio between the native and the depolymerised gums. The chromatograms of the sugars after complete hydrolysis of the guar gum indicated that the sugar profile is identical in native and depolymerised guar gum .(EFSA 2007).

In the petition made to EFSA, it states that the partially depolymerised guar gum products are very similar to native guar gum with respect to structure and composition, and that the chemical processes used to obtain the reduction in molecular weight are sufficiently mild as not to introduce residual contaminants and/or to result in a significant difference in the quality and quantity of residual contaminants that are found in native guar gum Given the processing conditions of the depolymerised guar gum, certain amounts of salts are formed as a result of the neutralization step in the process. The use of sodium hydroxide to neutralise citric acid used for the acid hydrolysis step results in formation of sodium citrate. The use of phosphoric acid to neutralise sodium hydroxide used for the oxidative alkaline hydrolysis step results in the formation of sodium phosphate. These salts are permitted for use as food additives in various foods. (EFSA 2007).

Partially hydrolyzed guar gum (PHGG) is produced from guar gum and has the same chemical structure but less than one-tenth the original molecular length making it good as a film former, foam stabilizer and swelling agent. Its properties make it a beneficial dietary fibre without altering the rheology, taste, texture and colour of guar gum.

PHGG is known as Benefiber(R) in the USA and has GRAS status (EFSA 2007).

A summary of repeat dose administration of guar gum was presented by the EFSA ANS Panel and are described below (EFSA, 2017). Many of these studies were presented by JECFA or the National Toxicology Program:

In a dose-range finding study, groups of B6C3F1 mice (10 animals/sex per group) were fed diets containing guar gum at 6300, 12,500, 25,000, 50,000 or 100,000 mg/kg (equivalent to 945, 1875, 3750, 7500 and 15,000 mg/kg bw/day) for 13 weeks (NTP, 1982). One female mouse of the highest dose group died. In female mice receiving 50,000 and 100,000 mg/kg guar gum weight gain was depressed. In treated mice of either sex, the feed consumption was comparable with or higher than that of control mice. No compound-related clinical signs or histopathological effects were observed. On the basis of this data, a NOAEL for mice of 15,000 mg/kg bw/day, the highest dose tested was derived by the EFSA ANS Panel.

Weanling albino rats (5 males/group) received a basal diet or basal diet plus 6 % (equivalent to 7200 mg/kg bw/day) guar gum for 91 days (Booth et al., 1963). There were no differences in differences in weight gain or feed intake observed, between the two groups. No significant alterations in haemoglobin, in erythrocyte and leucocyte counts or in organ weights were observed (Booth et al., 1963).

A total of 15 rats were fed a diet containing 0.5 % guar gum which is equivalent to 600 mg/kg bw/day, for 21 days (Keane et al., 1962 cited by JECFA, 1975b). Increased weight gain and protein efficiency with higher water content were reported.

Ten male newly weaned Sprague–Dawley rats were fed a diet containing 2 % guar gum for 36 days, which is equivalent to 2400 mg/kg bw/day. Guar gum was reported to not influence the growth of rats or the digestibility of the diet (Vohra et al., 1979).

Wistar rats (10 young and 15 old males) were fed 8% guar gum, which was equivalent to 9,600 mg/kg bw/day in the diet, for 6 weeks. Young rats responded to guar gum consumption with reduced body weight gain and improved carbohydrate tolerance; these responses were not seen in older rats (Track et al., 1985).

Five growing male Wistar rats were fed a diet containing 5 % guar gum for 3 weeks. In comparison to the control group, feed consumption slightly decreased, resulting in a lower increase in body weight. The utilisation efficiency declined but protein utilisation remained unchanged (Hidehisa et al., 1994).

Six groups of Osborne–Mendel rats were fed guar gum at 1 %, 2 %, 4 %, 7.5 %, and 15 % dietary levels for 13 weeks. This is equivalent to approximately 1200, 2400, 4800, 9000 and 18,000 mg/kg bw/day (Graham et al., 1981). At the two highest doses, male rats had significantly reduced weight than controls at week 12. Since weight gain was significantly reduced in males at the two highest doses and in females in all dose groups, without ‘drastic’ reductions in feed consumption, it was suggested by the authors that ‘utilisation’ of guar gum by the rats was probably poor. Only glucose values in males showed a reduced linear trend with doses. Liver and kidney weights of males showed significant changes, however, only kidney weights showed a dose-related decreasing trend. Only bone marrow showed atrophy characterised by proportional reductions of all cellular elements in the high dose group. The authors concluded that the lack of more consistent linear trends in some parameters may suggest that the duration of the feeding was too short for patterns to be established in this study. Based on the lack of histopathological effects for most organs and a lack of a dose-related effect on bone marrow, the EFSA ANS Panel identified a NOAEL of 18,000 mg/kg bw/day from this study (EFSA, 2017).

In a dose-finding study groups of F344 rats (10 animals/sex per group) were fed diets containing guar gum at 6300, 12,500, 25,000, 50,000 or 100,000 mg/kg (equivalent to 315, 625, 1250, 2500, 5000 mg/kg bw/day), for 13 weeks (NTP, 1982). Two female rats died in the top two doses. A dose-related slight decrease in feed consumption was reported for rats in both sexes. Feed consumption by rats in the 100,000 mg/kg diet group was 80 % that of the controls and the weight gain of male rats in this group was decreased by 16 % as compared to

controls. In female rats, weight gain was not affected by treatment. No compound-related clinical signs or histopathological effects were reported. On the basis of this data, a NOAEL of 5000 mg/kg bw/day was derived by the EFSA ANS Panel (EFSA, 2017).

The JECFA evaluation of guar gum refers to a study on four groups of five male and five female Beagle dogs fed 1 %, 5 % or 10 % of a precooked mixture of gum blend (equivalent to approximately 750, 3,750 and 7500 mg/kg bw/day), for 30 weeks (Cox et al., 1974, unpublished report as cited by JECFA, 1975b). Gut hypermotility, soft bulky faeces and reduced digestibility were observed at the highest dose and were considered of no toxicological significance. No adverse effects were noted for haematological and urinary parameters, or gross and histopathological and ophthalmological examinations.

Two monkeys were given 1 g guar flour daily in their diet. Growth, wellbeing, haematology parameters and urea were reported to remain normal. One animal died after 16 months and the other was sacrificed after 24 months. Gross and histopathology showed no abnormal effects (Krantz, 1947; unpublished, cited in JECFA, 1975b).

The EFSA ANS Panel concluded that overall, in short-term and sub-chronic studies in mice, rats, dogs and monkeys, no adverse effects were observed at the highest dose tested (EFSA, 2017).

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In Vitro Data

In Vitro Carcinogenicity/Mutagenicity

Guar gum has been shown to be negative in the Ames Salmonella typhimurium assay with and without S9 activation systems [both rat and hamster liver fractions used]. The strains tested were TA97, TA98, TA100, TA1535 and TA1537 [Zeiger et al., 1988].

A paper investigating the inhibitory activity of heat treated vegetables on mutagenicity using Salmonella typhimurium (TA98 & TA100), suggests that the presence of water soluble indigestible polysaccharides when heated greatly increased the inhibitory action against specific mutagens. [Yamaguchi, 1992]

Guar gum failed to induce gene mutations in Drosophila melanogaster in a sex-linked recessive lethal mutagenesis test [Lee, 1983]

References - In Vitro Carcinogenicity/Mutagenicity

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In Vitro - Other Relevant Studies

No data identified.

References - In Vitro - Other Relevant Studies

No data identified.

Emissions and Associated Toxicity Data

Baker et al., [2004]; examined the effects of the addition of 482 tobacco ingredients upon the biological activity and chemistry of mainstream smoke. The ingredients, essentially different groups of flavourings and casings, were added in different combinations to reference cigarettes. The addition of guar gum at 100 ppm was determined not to have affected the mutagenicity of the total particulate matter (TPM) of the smoke in either the Ames, in vitro micronucleus assay or the neutral red assay when compared with that of the control cigarettes [Baker et al., 2004].

The addition of guar gum at 100 ppm to reference cigarettes, used in a 90 day-sub-chronic inhalation exposure in

rats, led to a series of pathological changes to smoke exposure that were indistinguishable from those changes caused by the control cigarettes. This indicated that addition of guar gum to a reference cigarette had no discernable effect upon the type or severity of the treatment related pathological changes associated with tobacco smoke exposure [Baker et al., 2004]

A total of 95 ingredients were tested individually through addition at different concentrations to the tobacco of experimental cigarettes. Mainstream cigarette smoke chemistry analysis, bacterial mutagenicity testing, and cytotoxicity testing were conducted. The authors concluded that these ingredients, which included guar gum produced minimal changes in the overall toxicity profile of mainstream cigarette smoke, and in some cases, the addition of high levels of an ingredient caused a small reduction in toxicity findings, probably due to displacement of burning tobacco [Gaworski et al., 2011].

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