

Starch oxidised

Ingredient synonym names

Bleached starch,
Oxidized corn starch

IDENTIFIER DETAILS

CAS Number	FEMA Number	Additive Number	Ingredient EC Number
65996-62-5	-	1404	-
CAS Additional Number	FL Number	CoE Number	
-	-	-	

Ingredient chemical structure

Under controlled conditions of temperature, pressure, and time, the oxidizing agent reacts with the starch to cleave the polymeric chains and to oxidize the end groups from aldehyde to carboxylic acid groups.

Chemical formula -

Ingredient CLP Classification

Ingredient REACH Registration Number

-

Acute Oral Toxicity	Eye Damage/Irritation	Carcinogenity
0	0	0
Acute Dermal Toxicity	Respiratory Sensitisation	Reproductive Toxicity
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) -

Starch oxidised

Acceptable Daily Intake (ADI) comments

The EFSA ANS Panel concluded that there is no need for a numerical ADI [EFSA ANS, 2017].

FDA Status

CFR21

175: Indirect food additives: adhesives and components of coatings [Starch]

176: Indirect food additives: paper and paperboard components [Starch]

182: Substances generally recognized as safe [Starch]

184: Direct food substances affirmed as generally recognized as safe [Starch]

CoE limits - Beverages
(mg/kg)

-

CoE limits -
Food (mg/kg)

-

CoE limits -
Exceptions (mg/kg)

-

HUMAN EXPOSURE

Ingredient Natural Occurrence (if applicable)

Although derived from natural compounds, oxidised starch is a purely synthetic compound.

References - Ingredient Natural Occurrence

No data identified

Ingredient Reported Uses

Modified starch is a food additive which is prepared by treating starch or starch granules, causing the starch to be partially degraded (GSFA, viewed 22/09/09). Modified starch is used as a thickening agent, stabilizer, or an emulsifier. Apart from food products, modified starch is also found in pharmaceuticals, paper and many other applications. Starches are modified for a number of reasons. Starches may be modified to increase their stability against excessive heat, acid, time and cooling or freezing; to change their texture; to decrease the viscosity or to lengthen or shorten gelatinization time. These materials have extensive use in the textile and paper industries.

References - Ingredient Reported Uses

General standard for food additives (GSFA), Codex alimentarius, FAO/WHO Food Standards, viewed 22/09/09 <http://www.codexalimentarius.net/gsfonline/additives/details.html?id=69>

TOXICITY DATA

In Vivo Data

Acute Toxicity Data

No data identified

In Vivo Carcinogenicity/Mutagenicity

In the micronucleus assay there was no significant increase in the incidence of micro nucleated erythrocytes in the bone marrow of mice exposed to glucose syrup and maltitol crystal, administered by oral gavage at doses between 3.75-30 g/kg [Takizawa et al., 1984].

Starch oxidised

The EFSA ANS Panel presented the following data [EFSA ANS, 2017].

A carcinogenicity study (89-week) in mice was available for hydroxypropyl distarch phosphate. There was no evidence of carcinogenicity. This chronic study in mice demonstrated some histopathological changes in the kidneys characterised by intratubular mineralisation, which according to the authors, was of no toxicological significance for the human health.

Carcinogenicity studies in rats were available for phosphated distarch phosphate, acetylated distarch phosphate, acetylated starch, acetylated distarch adipate, hydroxypropyl distarch phosphate and starch sodium octenyl succinate. There was no evidence of carcinogenicity. The long-term studies in rats did not reveal any significant effects, except for caecal enlargement. As this effect was observed without associated histopathological changes, it was considered to be of no toxicological significance for humans. Kidney lesions (pelvic and corticomedullary mineralisation) in rats fed high levels (up to 62%; 31,000 mg/kg bw/day) of phosphated distarch phosphate, acetylated distarch phosphate, acetylated starch, acetylated distarch adipate and hydroxypropyl distarch phosphate were observed. The lesions were considered to be associated with an imbalance of Ca/P and Mg in the diet. As the rat is a particularly sensitive species for PN, while the effect was not observed in the hamster and the pig, the effect was considered to be of no relevance for risk assessment in humans. These renal changes were not observed in rats fed starch sodium octenyl succinate at 30% in the diet for up to 120 weeks.

References - In Vivo Carcinogenicity/Mutagenicity

Takizawa et al., (1984) Bacterial reversion of the assay and micronucleus test carried out on hydrogenated glucose syrups 'Malti towa' [powder] and maltitol crystal. *Mutat Res* 137(2-3): 133-137.

EFSA., Mortensen, A., Aguilar, F., Crebelli, R., Di Domenico, A., Dusemund, B., Frutos, M. J., & Leblanc, J. C. (2017). Re-evaluation of oxidised starch (E 1404), monostarch phosphate (E 1410), distarch phosphate (E 1412), phosphated distarch phosphate (E 1413), acetylated distarch phosphate (E 1414), acetylated starch (E 1420), acetylated distarch adipate (E 1422), hydroxypropyl starch (E 1440), hydroxypropyl distarch phosphate (E 1442), starch sodium octenyl succinate (E 1450), acetylated oxidised starch (E 1451) and starch aluminium octenyl succinate (E 1452) as food additives. *EFSA Journal*, 15(10).

Dermal Toxicity

No data identified

References - Dermal Toxicity

No data identified

Reproductive/ Developmental Toxicity

Maize starch pasteurized by irradiation was fed to OFA rats (Sprague Dawley derived) in an uncooked form (irradiated at 300 krad.) and in a cooked form (irradiated at 300 and 600 krad.) at dietary level of 62%. A six month toxicity trial was performed with uncooked starch and a 24 month trial with cooked starch. At the same time a reproduction study was conducted, over 3 generations with 2 litters per generation, using both uncooked and cooked irradiated starch. The parent generation (Fo) was randomly selected from animals in the feeding study and after the production of the F1a and F1b generations they were returned to that study. Control groups corresponding to each form of starch were established. They were fed the same diet except that the starch was not irradiated. The results of the various investigations conducted during the study (behaviour, growth, mortality, haematology, serum biochemistry, histopathology) did not reveal any toxicological effect due to treatment, nor any effect on reproduction. No significant differences were shown between treated and control group, [Truhaut et al., 1976].

Starch oxidised

The EFSA ANS Panel presented the following data [EFSA ANS, 2017].

Dietary reproductive toxicity studies in rats were available for phosphate distarch phosphate, acetylated distarch phosphate, acetylated starch and acetylated distarch adipate. No effects on reproductive performance or maternal and developmental toxicity were observed in the three-generation reproductive toxicity studies at dietary levels of up to 62% (equivalent to 31,000 mg/kg bw per day). No prenatal developmental toxicity studies were available.

References - Reproductive/ Developmental Toxicity

Truhaut et al., (1976). Toxicologic evaluation of irradiated corn starch by long term experimentation with rats. Eur J Toxicol Environ Hyg. 9(6): 347-56.

EFSA., Mortensen, A., Aguilar, F., Crebelli, R., Di Domenico, A., Dusemund, B., Frutos, M. J., & Leblanc, J. C. (2017). Re-evaluation of oxidised starch (E 1404), monostarch phosphate (E 1410), distarch phosphate (E 1412), phosphated distarch phosphate (E 1413), acetylated distarch phosphate (E 1414), acetylated starch (E 1420), acetylated distarch adipate (E 1422), hydroxypropyl starch (E 1440), hydroxypropyl distarch phosphate (E 1442), starch sodium octenyl succinate (E 1450), acetylated oxidised starch (E 1451) and starch aluminium octenyl succinate (E 1452) as food additives. EFSA Journal, 15(10).

Inhalation Toxicity

Gudziol et al. (2009) studied 36 healthy subjects that were nasally exposed to wheaten flour or corn starch dust whilst sitting in an exposure chamber. The constant flow rate was 3.111 per minute for a period of 15mins. The subjects breathed orally over a breathing tube clean air. The time interval between both exposures was seven days excluding cross over effects. The deposition efficiency of both types of food powder was particle size dependent. Highest it was with the particle sizes between 5-100 microm. Here it lay between 92% and 99%. The small particles of wheaten flour respective corn starch with an aerodynamic diameter between 1-4 microm deposited nasally 31% respectively 74%. The new relatively simple method of measurement of nasal deposition efficiency does not load the deeper respiratory tract. The results confirm the good filtering capability of the healthy nose for large dust particles. The nasal deposition of particles smaller than 5 microm is reduced but not absent. The small dust particles of wheaten flour and corn starch are very different nasally deposited [Gudziol et al., 2009].

References - Inhalation Toxicity

Gudziol et al. (2009). Investigations of nasal deposition efficiency of wheaten flour and corn starch. Laryngorhinootologie; 88:398-404. Article in German.

Cardiac Toxicity

No data identified

References - Cardiac Toxicity

No data identified

Addictive Data

No data identified

References - Addictive Data

No data identified

Starch oxidised

Behavioral data

Berlin et al., (2004) reported that 'glucose attenuates tobacco craving and withdrawal symptoms in temporarily abstinent smokers'. The researcher concluded that 'further studies assessing the direct effect of glucose on brain serotonin are needed to ascertain whether a glucose-induced reduction in craving is associated with an increase in brain serotonin, [Berlin et al., 2004].

Glucose and caffeine are reported to improve cognition and mood, (caffeine). The effects on both substances in combination were studied in a double-blind study, (20-participants). The researchers concluded that 'the data suggested that there is some degree of synergy between the cognition-modulating effects of glucose and caffeine which merits further investigation, [Scholey and Kennedy, 2004].

A paper reviewing advances in pharmacotherapy for treatment of tobacco dependence reported the rapid increase in non-nicotine pharmacotherapies, (including glucose), [Foulds et al., 2004].

Messier, (2004) reported the memory enhancing properties of glucose have been studied for a period of almost 20-years and glucose memory improvement occurs at two optimal doses in animals (100 mg/kg and 2 g/kg), which is thought to correspond to two physiological mechanisms underlying glucose effects in memory. However, dose-response studies in humans are reported to be rare with glucose being reported to facilitate tasks where are difficult to master or involve divided attention rather than easier tasks. There is reported evidence that impaired glucose regulation is associated with impaired cognition (episodic memory in particular). Results of a few studies have revealed that treatments that improve glucose regulation also improve cognition, (in diabetic patients), [Messier, 2004].

References - Behavioral data

Berlin et al., (2005) Effect of glucose on tobacco craving. Is it mediated by tryptophan and serotonin? *Psychopharmacology (Berl)*. 178(1):27-34.

Foulds et al., 2004. Advances in pharmacotherapy for tobacco dependence. *Expert. Opin. Emerg. Drugs*. 9(1):39-53.

Messier C, (2004). Glucose improvement of memory: a review. *Eur J Pharmacol*. Apr 19;490(1-3):33-57.

Scholey and Kennedy, (2004). Cognitive and physiological effects of an "energy drink": an evaluation of the whole drink and of glucose, caffeine and herbal flavouring fractions. *Psychopharmacology (Berl)*. 176(3-4):320-30.

In Vivo - Other Relevant Studies

Corn starch that had been oxidised (by adding 5.5% sodium hypochlorite to it), was orally administered to groups of male (n=15) and female (n=15) rats for 90 days at dietary levels of 0, 5, 10 or 25% [WHO, 1973]. No changes in the growth, food consumption, and blood and urine biochemistry were observed. Quantities of faeces dry matter/unit food was elevated to 25% in both sexes. This group of animals were also observed to have a raised relative weight of the caecum, particularly in females. No other statistically significant dose related changes were observed. Examination of the gross pathology of the test animals revealed no significant changes. Histopathological examination was not performed in this study [WHO, 1973].

The EFSA ANS Panel presented the following data [EFSA ANS, 2017].

Short-term and/or subchronic (90-day) studies in rats are available for all modified starches, except monostarch phosphate (E 1410), but occasionally also studies in dogs, pigs or hamsters were available. The modified starches were given at dietary levels up to 70%. The test duration was up to 90 days. Effects on body weight and feed

Starch oxidised

consumption were not observed up to dietary levels of 25%. Caeca weights of treated animals were not different from those of controls. Caeca weights were increased at exposure levels of 30% and higher, but without histopathological changes. The only significant histopathological change was the presence of pelvic and/or corticomedullary mineralisation in the kidneys, which was observed with modified as well as unmodified starches, and occurred more pronounced in females than in males. In a 90-day study with acetylated oxidised starch in rats, a NOAEL of 10% in the diet, equal to 5,900 mg/kg bw per day, was identified based on microscopic changes in the kidneys and urinary bladder epithelium, which were observed at 18,000 mg/kg bw per day, the following dose in this study.

Two chronic studies (52-week) were available, one with acetylated distarch phosphate and one with acetylated distarch adipate. At necropsy, relative organ weights showed no differences between the groups, except for caecal enlargement. Histopathological examination of kidney sections demonstrated the presence of treatment-related pelvic nephrocalcinosis. An apparent correlation was observed between the increased incidence of pelvic nephrocalcinosis, increased accumulation of calcium in the kidney and increased urinary excretion of calcium.

Maltose and higher saccharides present in corn syrup are not absorbed as such but are converted to glucose in the digestive process and then absorbed. The major pathways of glucose metabolism are well known in humans, glucose from the plasma is reported to be metabolised by glycogen formation. Anaerobic glycolysis leads to the formation of fat via the Krebs cycle. Glucose excretion in the urine is reported to occur after the rapid absorption of glucose leads to excessive blood glucose concentrations or during various disease states such as diabetes mellitus, with faecal absorption occurring only after either excessive ingestion of glucose or malabsorption [FDA, 1976].

The safety evaluation of maltitol and hydrogenated glucose syrups were evaluated by JECFA at the 27th, 29th, 41st and 49th meetings. A number of papers were reviewed examining the metabolic fate of maltitol and other higher polyols. In vitro and in vivo studies indicate that the available glycosidic linkages of higher order polyols in hydrogenated starch hydrolysates in a range of different polyols compounds are readily hydrolysed by digestive enzymes to maltitol and glucose. The absorption of glucose was reported to be predominantly in the upper intestine with absorption of maltitol occurring in the jejunum ileum and duodenum. In humans metabolism of maltitol occurred via intestinal microflora with some evidence that maltitol was absorbed. However, it was reported to be rapidly excreted in the urine unchanged [Modderman, 1993].

A sub chronic toxicity study for 13 weeks was conducted on hydrogenated polysaccharide fraction fed to rats at 0, 1.25, 2.5 or 5% in the diet. There was reported to be no treatment related effects at the end of the 13-week period [SCF 1999].

A study of male and female OFA rats [derived from Sprague-Dawley rats] were exposed to hydrogenated dextrin diets containing 0, 1.25, 2.5, 5% hydrogenated dextrin [equivalent to 0, 1, 2, or 4 g/kg/day per males and 0, 1.4, 2.8, or 5.2 g/kg/day for females] for 13 weeks, was carried out. There was found to be no toxicologically significant findings for any of the parameters investigated [JECFA, 1998].

In a study described by the WHO (1973) the digestibility of oxidized wheat starch (conditions not stated) was examined in rats by using the modified starch as the sole source of carbohydrate at a level of 63.7% (dry basis) of the diet. The degree of assimilation by and the general effects on groups of six rats over a feeding period of 28 days were assessed from consideration of body weight changes, faecal residues, digestibility coefficients for starch and post-mortem appearance of the animals and their gastrointestinal tracts. Body weight gain and digestibility coefficients were practically indistinguishable from those obtained for wheat starch or corn starch. Nothing abnormal was noted on post-mortem examination [WHO, 1973].

An additional study detailed by the WHO (1973) studied three groups of rats each receiving corn starch oxidized with 3.9%, 4.5% or 5.5% hypochlorite calculated as chlorine respectively. This corresponds to the introduction of

Starch oxidised

0.57% (2.04 COOH groups per 100 glucopyranose units), 0.8% (2.86 COOH groups per 100 glucopyranose units) and 0.9% (3.57 COOH groups per 100 glucopyranose units) carboxyl groups. To 5 g basal diet were added 1, 2 or 4g modified or control starch and this diet was fed to rats for 10 days. No tissue damage was associated with the diarrhoea and caecal enlargements observed in groups receiving 2 g or 4 g starch in their feed. Liver, kidney, heart and spleen weights were normal. Diarrhoea and caecal enlargement are known to occur in rats fed starches of poor digestibility or other carbohydrates [WHO, 1973].

The digestibility of oxidized starches at levels of 2.5%, 6% and 43.2% calculated as chlorine, equivalent to a carboxyl content of 0.32% (1.15 COOH per 100 glucopyranose units), 0.9% (3.81 COOH per 100 glucopyranose units) or 1.46% (5.23 COOH per 100 glucopyranose units) was studied in groups of six male and six female rats [WHO, 1973]. The animals were kept for seven days on 5 g basal diet and then given either 1 g or 2 g starch supplements for 21 days. Poor weight gain with diarrhoea were noted only with the highly oxidized material at both dietary levels. One rat from each of the high dietary level groups was examined. Marked caecal dilation was seen only in animals fed the heavily oxidized starch [WHO, 1973].

In a study by Caderni et al., (1996) female Sprague Dawley rats were fed diets containing sucrose, glucose, fructose, corn starch or Hylon 7 [a starch with a high amylose content]. After one month colon proliferation was assessed by measuring the uptake of [3H] thymidine in vitro. Glucose and fructose and cornstarch lowered mucosal proliferation compared to sucrose, which was considered to be a protective factor in colon carcinogenesis [Caderni et al., 1996].

Liu et al., (2005), have previously reported studies that indicated that dietary glucose (15% in drinking water) could markedly exacerbate the toxicity of parathion in adult rats. Liu et al., (2005) evaluated the effect of consumption of the commonly used sweetener, high fructose corn syrup (HFCS), on parathion toxicity in adult and juvenile rats. Animals were given free access to either water or 15% HFCS in drinking water for a total of 10 days and challenged with parathion (6 or 18 mg/kg, s.c., for juveniles or adults, respectively) on the 4th day. Signs of cholinergic toxicity, body weight and chow/fluid intake were recorded daily. Acetyl cholinesterase (AChE) activity and immunoreactivity (AChE-IR) in frontal cortex and diaphragm were measured on days 2, 4, and 7 days after parathion exposure. As HFCS was associated with a significant reduction in chow intake, adult rats were also pair-fed to evaluate the effect of reduced chow intake alone on parathion toxicity. The results indicated that the cholinergic toxicity of parathion was significantly increased by HFCS feeding in both juvenile and adult rats. The excess sugar consumption, however, did not significantly affect parathion-induced AChE inhibition in either tissue or either age group. Enzyme immunoreactivity in frontal cortex was generally not affected in either age group while diaphragm AChE-IR was significantly reduced by parathion and HFCS alone in adult animals at 2 and 4 days time points, and more so by the combination of sugar feeding and parathion exposure in both age groups. Food restriction alone did not exacerbate parathion toxicity. While the mechanism(s) remains unclear, Liu et al., (2005) concluded that voluntary consumption of the common sweetener HFCS can markedly amplify parathion acute toxicity in both juvenile and adult rats [Liu et al., 2005].

References - In Vivo - Other Relevant Studies

Caderni et al., (1996) Dietary sucrose, glucose, fructose, and starches affect colonic functions in rats. *Nutr Cancer* 25(2): 179-186. the Food and Drug Administration.

JECFA (1983) The evaluation of certain food additives and contaminants. Twenty seventh report of the Joint FAO/WHO Expert Committee on Food Additives. WHO Technical Report Series No 696

JECFA (1986). The evaluation of certain food additives and contaminants. Twenty ninth report of the Joint FAO/WHO Expert Committee on Food Additives. WHO Technical Report Series No 733.

JECFA (1993). The evaluation of certain food additives and contaminants. Fourty first report of the Joint FAO/WHO Expert Committee on Food Additives. WHO Food Additives Series No 32.

Starch oxidised

JECFA (1998). Safety evaluation of certain food additives and contaminants. The forty-ninth meeting of the Joint FAO/WHO Expert Committee on Food Additives (JECFA). WHO Additives Series No 40

Liu J et al., (2005) Dietary modulation of parathion-induced neurotoxicity in adult and juvenile rats. *Toxicology*. 210(2-3):135-145

Modderman (1993) Safety assessment of hydrogenated starch hydrolysates. *Regulatory Toxicology and Pharmacology* 18: 80-114.

SCF (1999) Scientific Committee on Food: Opinion on a maltitol syrup not covered by the current specifications [given on 2nd December 1999].

WHO FOOD ADDITIVES SERIES NO. 5, Toxicological evaluation of some food additives including anticaking agents, antimicrobials, antioxidants, emulsifiers and thickening agents, 1973
<http://www.inchem.org/documents/jecfa/jecmono/v05je74.htm>

In Vitro Data

In Vitro Carcinogenicity/Mutagenicity

Two preparations of maltitol, hydrogenated glucose syrup and maltitol crystal were tested for mutagenicity using the Ames test. There was found to be no detectable activity in any of the following Ames *Salmonella typhimurium* strains TA98, TA100, TA1535, TA1537 or TA1538 at doses of 0.5-50 mg/plate both with and without metabolic activation. There was also no effect when tested using *Escherichia coli* WP2/pKM101 both with and without metabolic activation at 0.5-50 mg/plate [Takizawa et al., 1984].

Negative results were also obtained for hydrogenated dextrin when tested in the Ames strains TA98, TA100, TA1535 and TA1537 at doses of 5-5000 µg/plate both with and without metabolic activation [JECFA, 1993].

References - In Vitro Carcinogenicity/Mutagenicity

JECFA (1993). The evaluation of certain food additives and contaminants. Fourty first report of the Joint FAO/WHO Expert Committee on Food Additives. WHO Food Additives Series No 32.

Takizawa et al., (1984) Bacterial reversion of the assay and micronucleus test carried out on hydrogenated glucose syrups 'Malti towa' [powder] and maltitol crystal. *Mutat Res* 137(2-3): 133-137.

In Vitro - Other Relevant Studies

A study in which the ability of Soya infant formulas were assessed for there cariogenic properties (as they contain glucose syrup or maltodextrins instead of lactose) revealed that Soya infant formula is more acidogenic than infant formulae (with lactose) and bovine milk. The researchers highlighted the high caries inducing potential of Soya infant milk, [based on the glucose syrup and maltodextrin content], [Bhat and Dubey, 2003].

An increased consumption of high-fructose corn syrup (HFCS) was reported to have a 'temporal relation to the epidemic of obesity, and the over-consumption of HFCS in calorically sweetened beverages may play a role in the epidemic of obesity', [Bray et al., 2004].

References - In Vitro - Other Relevant Studies

Bhat and Dubey, (2003). Acidogenic potential of Soya infant formula in comparison with regular infant formula and bovine milk: a plaque pH study. *J. Indian Soc Pedod Prev Dent*. 21(1): 30-4.

Starch oxidised

Bray et al., (2004). Consumption of high-fructose corn syrup in beverages may play a role in the epidemic of obesity. *Am J Clin Nutr.* 79(4): 537-43.

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 starch at 19,000 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 starch at 19,000 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 starch 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].

Roemer et al., (2002) reported on a study in which cigarettes containing various additives in three different combinations were produced. Smoke condensates prepared from these cigarettes were then tested in two different in vitro assays. The mutagenicity of the smoke condensate was assayed in the Salmonella plate incorporation [Ames] assay with tester strains TA98, TA100, TA102, TA1535 and TA1537 in the presence and absence of an S9 metabolic activation system. The cytotoxicity of the gas/vapour phase and the particulate phase was determined in the neutral red uptake assay with mouse embryo BALB/c 3T3 cells. The authors concluded that the in vitro mutagenicity and cytotoxicity of the cigarette smoke was not increased by the addition of the ingredients which included sugar [corn syrup] at levels up to 62507 ppm [a multiple of its typical use in a US cigarette] [Roemer et al., 2002].

A recent mouse skin painting study investigated the carcinogenicity of condensate prepared from cigarettes containing a number of additives in combination, including starch at 97 ppm. The authors concluded that the study “did not indicate any substantive effect of these ingredients on the tumorigenicity of cigarette smoke condensate” [It should be noted that the cigarettes contained a typical American blend humectant and sugar component (i.e. glycerine $\approx$ 20,000 ppm, propylene glycol at $\approx$ 24,000 ppm, and brown invert sugar at $\approx$ 24,000 ppm)] [Gaworski et al., 1999].

When tested at 97 ppm in cigarettes, in a 13-week inhalation study, the presence of starch had no discernible effect on the character of extent of the biologic responses normally associated with inhalation of mainstream cigarette smoke in rats.”[Gaworski et al., 1998] [however, it should be noted that the cigarettes had been spiked with a number of flavour ingredients in combination prior to smoking, and they contained a typical American blend humectant and sugar component (i.e. glycerine $\approx$ 20,000 ppm, propylene glycol at $\approx$ 24,000 ppm, and brown invert sugar at $\approx$ 24,000 ppm)] [Gaworski et al., 1998].

The mutagenicity of the smoke condensate was assayed in the Salmonella plate incorporation [Ames] assay with the tester strain TA98 in the presence of an S9 metabolic activation system. The cytotoxicity of the cigarette condensate was determined in the neutral red uptake assay and the (3-(4,5-dimethylthiazol-2-yl)-5-(3-carboxymethoxyphenyl)-2-(4-sulfophenyl)-2H tetrazolium, inner salt assay (MTS assay) with the human hepatocellular liver carcinoma cell line, HEP-G2. It was concluded that the in vitro mutagenicity and cytotoxicity of the cigarette smoke was not increased by the addition of the ingredients, which included oxidised starch at levels up to 5,000 ppm.

Starch oxidised

References - Emissions and Associated Toxicity Data

Baker RR, et al., (2004). An overview of the effects of tobacco ingredients on smoke chemistry and toxicity. Food Chem Toxicol. 42 Suppl: S53-83.

Gaworski et al., (1998). Toxicologic evaluation of flavor ingredients added to cigarette tobacco: 13-week inhalation exposure in rats. Inhalation Toxicol., 10, 357-381.

Gaworski et al., (1999). Toxicologic evaluation of flavor ingredients added to cigarette tobacco: skin painting bioassay of cigarette smoke condensate in SENCAR mice. Toxicology, 139, 1-17.

In vitro toxicity testing of tobacco ingredients in burnt form (Internal document R-59).

Roemer et al., (2002). Evaluation of the potential effects of ingredients added to cigarettes. Part 3: In vitro genotoxicity and cytotoxicity. Food and Chemical Toxicology 40, 105-111.