Final Report of the Cosmetic Ingredient Review Expert Panel

Maltitol and Maltitol Laurate

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Final Report on the Safety Assessment of Maltitol and Maltitol Laurate

ABSTRACT: Maltitol, a disaccharide alcohol derived from maltose, is used in 33 cosmetic products at concentrations up to 15% and functions as a flavoring agent, humectant, and a skin-conditioning agent. The particle sizes produced in Maltitol products intended as aerosolized cosmetics are large compared to respirable particle sizes. Maltitol Laurate, the ester of Maltitol and lauric acid, functions in cosmetics as an emulsion stabilizer and skin-conditioning agent, but is not in current use. Maltitol is hydrolyzed less readily by endogenous enzymes and a considerable amount undergoes fermentation in the lower gastrointestinal tract. Small absorbed amounts are excreted unchanged in urine. Maltitol was not toxic in acute and subchronic oral animal studies with mice, rats, and dogs. At 69.09%, Maltitol was not an ocular irritant and a non- to weak irritant in rabbits. As with sorbitol, mannitol, xylitol, lactitol, and lactose, Maltitol has been demonstrated to be nonmutagenic and nongenotoxic in a variety of *in vitro* test systems, including the Ames test, with and without the presence of metabolic activation. In a chronic oral toxicity/carcinogenicity study, both benign and malignant phaeochromocytomas occurred in male and female rats treated with 4.5 g/kg/d. No increases in mammary gland adenomas or fibroadenomas were observed. Maltitol reduced the tumor incidence in rats treated with 1,2-dimethylhydrazine. Maltitol administered by gavage to rabbits at 5 g/kg/d did produce an increase in the number of early resorptions and post-implantation losses, but resulted in no malformations. Maltitol at 2.5 g/kg/d was not a reproductive or developmental toxin. In acute oral toxicity, primary skin irritation, eye irritation and human patch testing studies using 69.09% Maltitol, no irritation were observed. In human patch tests, Maltitol was not irritating at levels up to 69.09%. The CIR Expert Panel noted that sugar alcohols are highly water soluble and not likely to be absorbed from the skin. Based on the structure of Maltitol, it will not absorb UV light. While no safety test data were available for Maltitol Laurate, its safety may be inferred based on the available data for Maltitol and for Lauric Acid, the two hydrolysis products of Maltitol Laurate. A previous safety assessment of Lauric Acid by the CIR Expert Panel found it safe for use in cosmetics. Although not in current use, were Maltitol Laurate to be used, the CIR Expert Panel would expect use in product types and at concentrations similar to Maltitol. Accordingly, the CIR Expert Panel assessment found Maltitol and Maltitol Laurate safe as cosmetic ingredients in the practices of use and concentrations described.

INTRODUCTION

This report presents available information pertinent to the safety of Maltitol, a sugar alcohol, that functions as a flavoring agent, humectant, and skin-conditioning agent (humectant) and is used in a wide variety of cosmetic product types, and Maltitol Laurate, a sugar alcohol ester, which functions as an emulsion stabilizer and skin-conditioning agent, but is not currently in use.

Lauric Acid, the fatty acid esterified to Maltitol to form Maltitol Laurate, was itself the subject of a safety assessment of a group of fatty acids by the Cosmetic Ingredient Review (CIR) Expert Panel (Elder 1987). Little acute toxicity was reported in rats at oral doses up to 19 g/kg. Clinical tests of products containing Lauric Acid at concentrations up to 13% were not irritating, sensitizing, or photosensitizing. These fatty acids were re-reviewed to update the practices of use in cosmetics and to consider any newly available safety test data and the conclusion was reaffirmed (Andersen 2005). Overall, Lauric Acid and other fatty acids in the 12 to 18 carbon chain length group were considered safe in the practices of use and concentration reviewed (up to 25% for Lauric Acid).

CHEMISTRY

Maltitol

As listed in the *International Cosmetic Ingredient Dictionary and Handbook* (Gottschalck and Bailey 2008), Maltitol (CAS No. 585-88-6; D-form) is a disaccharide polyol obtained by hydrogenation of maltose. It conforms to the structure shown in Figure 1.

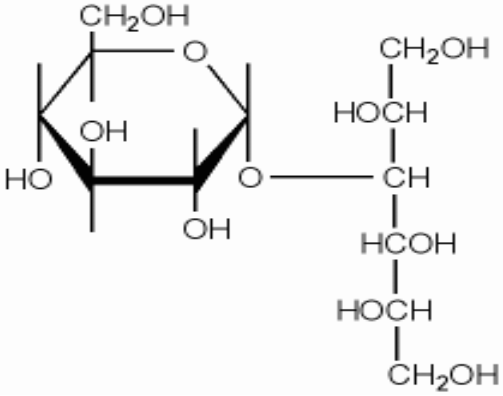

Figure 1. Structure for Maltitol (Gottschalck and Bailey 2008).

D-Glucitol, 4-O-á-D-glucopyranosyl- and Maltitol Solution are technical names; Crystalline Maltisorb is a trade name; and Lafrin-AM and Nikkol Aquasome LAV are trade name mixtures for/with Maltitol (Gottschalck and Bailey 2008).

According to the Registry for Toxic Effects of Chemical Substances (RTECS 1995), synonyms for Maltitol include:

Amalti Syrup;
Amalti MR 100;
D-Glucitol, 4-O-alpha-D-glopyranosyl- (9CI);
D-4-O-alpha-D-Glucopyranosylglucitol;
4-O=alpha-D-Glucopyranosyl-D-Glucitol;
Malbit;
Malti Mr;
Maltisorb;
Maltit;
Maltitol (6CI, 7CI); and
D-Maltitol.

Table 1 presents the chemical and physical properties of Maltitol.

Maltitol Laurate

According to the *International Cosmetic Ingredient Dictionary and Handbook* (Gottschalck and Bailey 2008), Maltitol Laurate (CAS No. 75765-49-0) is the ester of Maltitol (q.v.) and lauric acid that conforms to the structure shown in Figure 2.

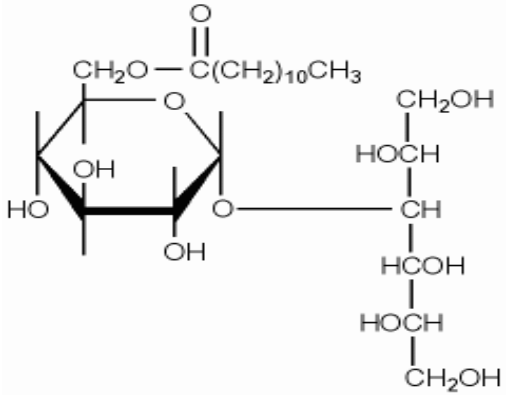

Figure 2. Chemical Structure for Maltitol Laurate (Gottschalck and Bailey 2008).

D-Glucitol; 4-O-á-D-Glucopyranosyl-; and MonododecanoateMaltitol Monolaurate are technical names and Maltel SML is a trade name for Maltitol Laurate (Gottschalck and Bailey 2008).

Physical and chemical properties of Maltitol Laurate were not available.

Maltitol Syrup

According to the Food Chemicals Codex (1996), Maltitol syrup is a water solution of a hydrogenated, partially hydrolyzed starch containing Maltitol, sorbitol, and hydrogenated oligo- and polysaccharides and that is a clear, colorless, syrupy liquid having a sweet taste. It is very soluble in water and slightly soluble in alcohol.

According to Lynch et al. (1996), material that contains 50 - 90% Maltitol has been considered as hydrogenated glucose syrup, now referred to as Maltitol syrup.

An opinion by the European Commission's Scientific Committee on Foods (SCF) on Maltitol syrup concluded that: the use of this new material does not raise any additional safety concerns in relation to existing Maltitol syrups. Its use is therefore considered acceptable (SCF 1999).

The World Health Organization (WHO) reported the specifications that maltitol syrup has a Maltitol content of no less than 50%, a sorbitol content of no more than 8%, a maltotritol content of no more than 25% and a content of hydrogenated polysaccharides containing more than 3 glucose or glucitol units of no more than 30% (WHO 1999).

The United States Pharmacopeia (USP 2004) stated that Maltitol solution is a water solution of a hydrogenated, partially hydrolyzed starch. It contains, on the anhydrous basis, not less than 50.0% of D-Maltitol (C12H24O11) (w/w), and not more than 16.0% of D-sorbitol (C6H14O6) (w/w).

According to the European Food Safety Authority (EFSA), Maltiol syrup is authorized in Europe as a sweetener (food additive) in food. It is a mixture of Maltitol, sorbitol, and hydrogenated glucose syrup blended to achieve the final Maltitol syrup (EFSA 2006).

Method of Manufacture

According to Fukahori (1998), Maltitol is a sugar alcohol produced by the hydrogenation of maltose.

Analytical Methods

No analytical methods specific for the detection of Maltitol or Maltitol Laurate were available.

Impurities

No impurities data were available for Maltitol or Maltitol Laurate.

Table 1. Physical and Chemical Properties of Maltitol.

Property	Value	Reference
Molecular Weight	344.36	RTECS (1995)
Appearance	solid; white to off-white	Fisher Scientific (2007)
Melting Point	149°C - 152°C	Fisher Scientific (2007)
Stability	stable under normal temperatures and pressures.	Fisher Scientific (2007)
Reactivity	incompatible with oxidizing agents; decomposition products are carbon dioxide and carbon monoxide.	Fisher Scientific (2007)

USE

Cosmetic

According to the *International Cosmetic Ingredient Dictionary and Handbook*, Maltitol functions in cosmetics include use as a flavoring agent, a humectant, and skin-conditioning agent. Maltitol Laurate functions as an emulsion stabilizer and miscellaneous skin-conditioning agent in cosmetics (Gottschalck and Bailey 2008).

Ingredient uses as a function of cosmetic product type are provided by industry to the U.S. Food and Drug Administration (FDA) under the Voluntary Product Registration Program (VCRP). Concentrations of use are provided by industry to the Cosmetic, Toiletry, and Fragrance Association (CTFA), now the Personal Care Products Council (Council).

As provided to the VCRP in 2006, Maltitol was used in 33 cosmetic products (FDA 2006). Concentration of use data from the inductry survey ranged from 0.0009% to 15% (CTFA 2007). The highest concentration of 15% was reported in skin cleansing creams, lotions, liquids, and pads. The available usage and use concentration data are given in Table 3, along with the total number of products in each product type. For example, 1 of 32 eye lotions contains Maltitol at a concentration of 2%. In some cases, no uses were reported under the VCRP, but industry reported a use concentration; e.g., no uses of Maltitol in baby shampoos were reported under the VCRP, but an industry use concentration at 4% was reported, indicating use in at least 1 product.

No uses of Maltitol Laurate were reported under the VCRP (FDA 2006) and there were no use concentrations reported in the industry survey (CTFA 2007).

Jensen and O'Brien (1993) reviewed the potential adverse effects of inhaled aerosols, which depend on the specific chemical species, the concentration, the duration of the exposure, and the site of deposition within the respiratory system.

The aerosol properties associated with the location of deposition in the respiratory system are particle size and density. The parameter most closely associated with this regional deposition is the aerodynamic diameter, $\mathbf{d_a}$, defined as the diameter of a sphere of unit density possessing the same terminal setting velocity as the particle in question. These authors reported a mean aerodynamic diameter of 4.25 ± 1.5 μm for respirable particles that could result in lung exposure (Jensen and O'Brien, 1993).

Bower (1999), reported diameters of anhydrous hair spray particles of 60 - 80 μm and pump hair sprays with particle diameters of ≥80 μm.

Johnsen (2004) reported that the mean particle diameter is around 38 μm in a typical aerosol spray. In practice, he stated that aerosols should have at least 99% of particle diameters in the 10 - 110 μm range.

Non-cosmetic

The Scientific Committee for Food of the European Union (SCF 1985) assessed the safety of sweeteners, concluding that Maltitol is acceptable for use, also without setting a limit on its use. Like other polyols, Maltitol may produce a laxative effect when consumed at very high levels.

Table 3. Current uses and concentrations of Maltitol in cosmetics.

Product Category (Total # of formulations)	2005 uses (FDA 2006)	2007 concentrations (CTFA 2007)
Baby Products		
Shampoos (38)	-	4%
Bath Preparations		
Soaps and detergents (594)	-	0.1% - 8%
Eye Makeup Preparations		
Eye lotions (32)	1	2%
Non-Coloring Hair Preparations		
Hair conditioners (715)	-	0.8%
Hair sprays/aerosol fixatives (294)	-	0.8%
Hair tonics, dressings, etc. (623)	-	0.8%
Wave sets (59)	-	0.8%
Other non-coloring hair preparations (464)	-	0.8%
Hair Coloring Preparations		
Hair dyes and colors (1600)	-	0.7%
Tints (56)	-	0.7%
Rinses (46)	-	0.7%
Color sprays (4)	-	0.7%
Lighteners with color (14)	-	0.7%
Bleaches (103)	-	0.7%
Other hair coloring preparations (73)	-	0.7%

Table 3 (continued). Current uses and concentrations of Maltitol in cosmetics.

Product Category (Total # of formulations)	2005 uses (FDA 2006)	2007 concentrations (CTFA 2007)
Makeup Preparations		
Blushers (459)	-	7%
Face powders (447)	-	7%
Foundations (530)	1	7%
Leg and body paints (10)	-	4%
Lipsticks (1681)	-	4%
Makeup bases (273)	1	7%
Rouges (115)	-	4%
Makeup fixatives (37)	-	7%
Other makeup preparations (304)	1	7%
Oral Hygiene Products		
Dentifrices (54)	-	0.3%
Other oral hygiene products (10)	-	3%
Personal Hygiene Products		
Underarm deodorants (281)	-	8%
Douches (8)	-	8%
Feminine hygiene deodorants (7)	-	8%
Other personal hygiene products (390)	-	8%
Skin Care Preparations		
Skin cleansing creams, lotions, liquids, and pads (1009)	4	7% - 15%
Depilatories (49)	-	7%
Face and neck skin care preparations (546)	1	0.5% - 7%[a]
Body and hand skin care preparations (992)	5	0.6% - 7%[a]
Foot powders and sprays (43)	-	7%
Moisturizers (1200)	8	0.0009% - 7%[a]
Night skin care preparations (229)	1	7%[a]
Paste masks (mud packs) (312)	1	0.5% - 7%
Skin fresheners (212)	1	7%
Other skin care preparations (915)	8	7%
Suntan Preparations		
Suntan gels, creams and liquids (138)	-	4%
Indoor tanning preparations (74)	-	4%
Other suntan preparations (41)	-	4%
Total uses/ranges for Maltitol:	**33**	**0.0009% - 15%**

[a] includes a spray formulation at 7%.

Volgarev (1989) reported that Maltitol is used as a dietary sweetener and is derived from mono- and disaccharides in the food industry. The relative sweetness of Maltitol is 90.

The Food Chemicals Codex (1996) reported that Maltitol syrup is used in foods as a humectant, texturizing agent, stabilizer, and sweetener.

According to WHO (1997), sugar alcohols are primarily used as bulk sweetening agents or as sugar eplacements. They include the monosaccharide-derived polyols mannitol, sorbitol, xylitol, and the disaccharide-derived forms Maltitol, isomalt and lactitol, as well as polyol mixtures such as Maltitol syrup and sorbitol syrup, which contain hydrogenated polysaccharides.

According to the Federal Register (FDA 2007), when used in food, Maltitol is among the noncariogenic carbohydrate sweeteners for FDA permits claims for reducing dental caries.

Fukahori et al. (1998) reported that Maltitol has been used as a filler in solid pharmaceuticals and as a sweetener in many foods.

The Joint Food and Agriculture Organization/World Health Organization Expert Committee on Food Additives (JECFA 2006)

has reviewed the safety data and concluded that Maltitol is safe. JECFA has also established an acceptable daily intake (ADI) for Maltitol of "not specified", meaning no limits are placed on its use. An ADI "not specified" is the safest category in which JECFA can place a food ingredient.

Calorie Control Council (2007) submitted a notification of the GRAS status of Maltitol to the U.S. Food and Drug Administration (FDA). The notification described the use of Maltitol as a flavoring agent, formulation aid, humectant, nutritive sweetener, processing aid, sequestrant, stabilizer and thickener, surface finishing agent and texturizer. The petition also addressed the use of Maltitol at levels of up to 99.5% in hard candy and cough drops, 99% in sugar substitutes, 85% in soft candies, 75% in chewing gum, 55% in non-standardized jams and jellies and 30% in cookies and sponge cake. The Calorie Control Council also noted that the safety of Maltitol as a food ingredient is substantiated by numerous studies in both humans and animals.

GENERAL BIOLOGY

Absorption, Distribution, Metabolism, Excretion

Felber et al. (1987) studied the metabolism of Maltitol compared to that of sucrose in a group of 8 normal human subjects (ages 24 ± 2 yr; weight: 96 ± 3% of their ideal body weight). Each subject ingested 30 g of Maltitol or 30 g of sucrose with a 1 week interval between the 2 studies. Blood samples were taken and after a 45 min rest-period, the subjects were given 30 g of either Maltitol or sucrose in a randomized order. Each sugar was dissolved in 200 ml of lemon-flavored water. Blood samples were collected in the fasting state and every 30 min over the 6 hr experimental period. Gas exchange measurement was performed during 30 min in the post-absorptive state and over 6 h following ingestion. Urine was collected at the end of the study to determine urinary nitrogen.

The protein oxidation was assumed by the study authors to remain constant throughout the study and to be equivalent to 6.235 x N, where N is the number of grams of nitrogen excreted in the urine per minute.

In the 2 experiments, the mean fasting plasma glucose levels (89 ± 2 mg/dl), insulin levels (11.4 ± 1.0 µU/ml), mean basal glucose levels (1.41 ± 0.17 mg/kg· min), and the lipid oxidation rates (1.10 ± 0.09 mg/kg · min) did not differ. Thirty minutes after ingestion, plasma glucose increase was greater after sucrose (38 ± 4) than after Maltitol (21 ± 4 mg/dl, $p < 0.02$), as was plasma insulin level increase (25.5 ± 5.0 after sucrose vs. 9.3 ± 2.7 µU/ml after Maltitol, $p < 0.001$).

The peak of the stimulation of glucose oxidation occurred 60 min after the ingestion of sucrose and 90 min after the ingestion of Maltitol. The change in glucose oxidation was significantly decreased with Maltitol than with sucrose during the first 90 min after ingestion. It was slightly increased with Maltitol than with sucrose beginning from the 210th min. Maltitol resulted in a cumulated suprabasal glucose oxidation which amounted to 40% that obtained with sucrose after 180 min (Felber et al. 1987).

Beaugerie et al. (1991) studied the clinical tolerance, intestinal absorption, and energy value of isomalt, sorbitol, Maltitol, and lactitol when taken on an empty stomach. Six healthy volunteers were tested in 5 periods during which they ingested 10 g lactulose and then, in random order, an iso-osmotic solution of the 4 sugar alcohols. The fraction of sugar alcohols absorbed in the small intestine was determined by comparing the amounts of hydrogen excreted in the breath for 8 h after lactulose and the sugar alcohols. The energy value was determined knowing the amounts absorbed in the small intestine and digested in the colon.

All volunteers exhibited good tolerance to sugar alcohols. The mean percentage of malabsorption in the small intestine was significantly increased for lactitol (84 ± 14%, m ± SEM) than for Maltitol and isomalt (44 ± 7 and 40 ± 7%). The authors suggested that under the conditions of this experiment, bacterial digestion of the sugar alcohols reaching the colon was complete and did not affect their clinical tolerance (Beaugerie et al. 1991).

According to WHO (1999), in humans, Maltitol was hydrolyzed less readily by endogenous enzymes and a considerable amount underwent fermentation in the lower gastrointestinal tract. The small amount that was absorbed was excreted unchanged in the urine.

Gastrointestinal Effects

Ellis & Krantz (1941) and Patil et al. (1987) reported that large intake (~2 - 70 g/day) of non-digestable saccharides and sugar alcohol causes diarrhea in animals.

As reported by Koizumi et al. (1983), the maximum non-effective laxative dose of Maltitol is approximately twice that of sorbitol.

Glycemic Response

According to Vavasour (1999), ingestion of polyglycitol and Maltitol syrups in diabetic and non-diabetic subjects resulted in a lower glycemic response than with glucose in the following order: Maltitol syrup < polyglycitol syrup < glucose, which reflects the relative proportion of glucose released by hydrolysis of each material.

Effect on Absorption of Other Chemicals

As reported by Koizumi et al. (1983), Maltitol is a potentially useful agent as an enhancer of intestinal calcium absorption.

Niwa et al. (1980) examined the effects of Maltitol on gastrointestinal absorption of acetaminophen, sulfisoxazole and riboflavin in mice. When drugs were orally administered with a 9.6% or 12.0% Maltitol solution, the blood levels of drugs became lower than that in the control, and drug absorption was inhibited. According to the authors, these results were not caused by molecular interaction between drugs and sugar alcohols, but by the action of Maltitol which accelerated small intestine motility, secretion and vascular permeability.

Goda et al. (1992) demonstrated that the consumption of 10% Maltitol diet by rats resulted in increased calcium absorption.

According to Goda et al. (1993) and Kishi et al. (1996), in vitro experiments using everted ileal segments of rats suggested that Maltitol accelerated passive diffusion of calcium in the lower part of the small intestine.

Fukahori et al. (1998) investigated the relationship between the gastrointestinal transit and the plasma concentration of orally administered ^{45}Ca using various segments of the rat gastrointestinal tract and the plasma to clarify the putative factors in the Maltitol-induced enhancement of calcium absorption in the gastrointestinal tract in vivo. Seven week old male Wistar rats, 190 - 210 g, were used. The experimental solution contained 175 mM $CaCl_2$, 5 kBq/mL ^{45}Ca and 20% (w/v) Maltitol.

After a 24-h fast, rats were administered 1 mL of the $[^{45}Ca]CaCl_2$ solutions (7 mg calcium equivalent) into the stomach via a gastric tube. Rats were anesthetized with ether 20, 40, 60, 90 or 120 min following administration of the test material, with a separate group of 5 rats being used at each time-point. An incision was made to the abdomen and a blood sample (~ 8 - 12 mL) was withdrawn from the aorta with a syringe. The sample was then centrifuged for 15 min.

After collection of the blood sample, the gastrointestinal tract was separated into stomach, 5 cm of the duodenum, 4 small intestine segments of equal length (upper and lower jejunum, upper and lower ileum), cecum and colon. The collected luminal contents were mixed with HCl (20 mL), shaken, and centrifuged for 15 min. ^{45}Ca radioactivity in the supernatant was determined and the amount of exogenously administered calcium remaining in the luminal contents was determined.

After intragastric administration of $[^{45}Ca]CaCl_2$ solution with Maltitol, plasma ^{45}Ca concentration sharply declined after the peak. Determination of ^{45}Ca radioactivity remaining in the various segments of the gastrointestinal tract revealed that administration of Maltitol elicited slower gastric emptying and slower intestinal transit, which resulted in extensive ^{45}Ca distribution along the small intestine throughout the course of the experiment. The luminal contents of the small intestine were significantly greater in rats given Maltitol than in the control group.

According to the authors, these results suggest that the enhancing action of Maltitol on intestinal calcium absorption could be attributed to reduced gastrointestinal calcium transit and increased luminal fluid content because of the osmotic activity of Maltitol. This would not only accelerate the dissolution of calcium, but also enable a larger area of the small intestine to absorb calcium for a longer period of time (Fukahori et al. 1998).

ANIMAL TOXICOLOGY

Acute Oral Toxicity

Shiseido Research Center (2008a) evaluated the acute oral toxicity of Maltitol (25.3 mL/kg body weight) in 5 male DD strain mice (22.5 g - 28.0 g). The animals were weighed and examined for 8 days and necropsy was performed at the end of the study. There were no observed clinical signs or necropsy findings related to the test material. The acute oral LD_{50} was >25.3 mL/kg body weight in mice accoring to the test conditions.

Subchronic Oral Toxicity

According to WHO (1999), the toxic potential of a materials that contained more than 49% of hydrogenated polysaccharides, consisting of 10% sorbitol, 8% Maltitol and 82% higher-order polyols was evaluated. No treatment-related toxicity was seen in any rats or dogs when the material containing 10% sorbitol, 8% Maltitol and 82% higher-order polyols was administered in the diet at dosages of up to 18 and 43 g/kg of body weight per day, respectively, for 90 days.

Chronic Oral Toxicity

Herrman (1993) summarized a combined long-term chronic toxicity/carcinogenicity study in rats using a commercial preparation (details not provided) containing approximately 87% Maltitol, which was fed to Crl:CD(SD)BR male and female rats at doses of 0, 0.5, 1.5, or 4.5 g/kg bw/day. Note, the highest dose corresponded to an average of about 10% of the commercial product in the diet. Rats were maintained on this diet for 52 weeks (20 animals/sex/group) after which they were killed.

Animals were examined daily in both experiments for signs of ill health or behavioral changes. Feed intake and body weights were recorded prior to administration of the test substance, at weekly intervals for the first 12 weeks, and then every 4 weeks until the completion of the study. Animals were monitored twice daily for mortality. Those found dead or killed moriband, as well as those killed at the end of the study underwent complete necropsies; organs were removed, weighed, and histologically examined. In the long-term study, cecum and colon diameters were measured. Ten animals/sex/group were subjected to ophthalmoscopic examination prior to the start of treatment and at weeks 13, 26, and 52 in the chronic study. Hematological examinations, blood chemistry tests, and urinalyses were performed on 10 animals/sex/group at weeks 14, 26, and 51 of the study.

No animals in the mid- or high-dose groups died. Three animals in the control group and 4 in the low-dose group died due to accidents; none of these deaths were related to treatment. Also, no treatment-related clinical signs were noted, nor did treatment have an effect on body weight. In males, sporadic food consumption was noted, but no clear trend was observed. In females of the high-dose group, mean feed consumption was significantly less at 12 and 52 weeks than the other groups. No ophthalmological changes were observed, neither were there significant differences noted in blood chemistry or urinalysis. Some differences were observed in hematological parameters; however, except for a decrease in leukocytes in the mid-dose females, none of these findings were present at all observation points. Gross or histopathological changes were not observed. There was an increase in cecum diameter in males of the high-dose group, which the authors concluded to be due to higher values in 3 out of 20 rats; the opposite was observed for females of the low- and high-dose groups. In this study the no adverse effect level (NOEL) was the highest dose tested - 4.5 g commercial product/kg bw/day (Herrman 1993).

Ocular and/or Mucosal Irritation

Shiseido Research Center (2008a) studied the eye irritation of Maltitol (69.09%) in 3 rabbits. The test material was instilled into one eye of each animal without irrigation. The other eye remained untreated and served as the control. The eye reactions were evaluated according to the Draize scoring method. The eye

irritation index of test sample was 2.0 at 4 h following instillation of test sample. It was therefore concluded that Maltitol is a non-irritant under the test conditions.

Dermal Irritation

Shiseido Research Center (2008a) studied the primary dermal irritation of Maltitol (69.09%) in 8 rabbits. The dorsal skin of the animals were clipped. Four of the rabbits were used for the intact skin procedure and the remaining 4 animals were used for the abraded procedure. The abraded skin was scratched in a criss-cross pattern by a needle. The test material (0.3 mL) was applied under occlusion to the dorsal skin of each animal. After 24 h and 72 h of exposure, the patches were removed, and skin reactions were evaluated according to the Draize scoring method. The Primary Irritation Index (PII) was 0.1 - none to weak irritant under the test conditions.

In a cumulative skin irritation study by Shiseido Research Center (2008a), Maltitol (69.09%) was evaluated in 3 guinea pigs (330 g - 390 g body weight). The flank of the animals was clipped and shaved free of hair. The test sample was applied onto the flank once daily for 3 days. The skin reactions were evaluated at 24 h following each application. It was concluded that Maltitol was a non- to weak irritant under the test conditions.

Dermal Sensitization

No dermal sensitization data were available.

Phototoxicity

No phototoxicity data were available.

REPRODUCTIVE AND DEVELOPMENTAL TOXICITY

Bussi et al. (1985) reported on the effects of Maltitol in gravid female New Zealand White rabbits. Maltitol was administered by gavage to the animals from day 6 through day 18 of pregnancy at doses of 1.25, 2.5, or 5 g/kg/day. The number of rabbits used in the study was not provided. At 5 g/kg/day there was an increase in the number of early resorptions and increased post-implantation losses. No effects were observed in any treated group on maternal body weight increase, number of viable and dead fetuses, or on fetal body weights. In addition, no malformed fetuses were found at any of the doses administered.

GENOTOXICITY

Takizawa et al. (1984) reported on a bacterial reversion assay and micronucleus test carried out on hydrogenated glucose syrups 'Malti-Towa' (powder) and maltitol crystal. 'Malti-Towa' is a reduced maltose syrup. Two preparations of Maltitol, hydrogenated glucose syrups and maltitol crystal were examined for genotoxic potential in a series of short-term tests. In the bacterial reversion assay, Maltitol induced no detectable revertants in any of the tester strains, *Salmonella typhimurium* TA98, TA100, TA1535, TA1537, TA1538 or *Escherichia coli* WP2/pKM101at doses of 0.5 - 50 mg per plate with and without rat liver S9 mix. In the micronucleus test, no significant increase in the frequency of micronucleated erythrocytes was observed in bone marrow of mice after administration of the 2 preparations at 3.75 - 30 g/kg by gastric intubation.

According to Lynch et al. (1996), sorbitol, mannitol, xylitol, lactitol, Maltitol, maltitol syrup, isomalt, and lactose have been demonstrated to be nonmutagenic and nongenotoxic in a variety of in vitro test systems, including the Ames test, both with and without the presence of metabolic activation. The authors concluded that these polyols are nonmutagenic and nongenotoxic.

Canimoglu and Rencuzogullari (2006) reported on the cytogenic effects on Maltitol in human peripheral lymphocytes. Maltitol did not induce sister chromatid exchanges at any of the doses (1.25, 2.5, and 5 mg/mL) and treatment periods (24 and 48 hrs). Maltitol induced chromosome aberrations and the frequency of micronucleus formation at 24 and 48 hrs in a non dose-dependent manner. Maltitol did not decrease the replication index or the mitotic index at all doses and treatment periods, nor did it alter the pH or osmolality of the medium. The authors concluded that Maltitol has a weak genotoxic potential and appears to be non-cytotoxic to human peripheral lymphocytes in vitro.

CARCINOGENICITY

Herrman (1993) summarized a combined long-term chronic toxicity/carcinogenicity study in rats using a commercial preparation (details not provided) containing approximately 87% Maltitol, which was fed to Crl:CD(SD)BR male and female rats at doses of 0, 0.5, 1.5, or 4.5 g/kg bw/day. The highest dose corresponded to an average of about 10% of the commercial product in the diet. Rats were maintained on this diet for 52 weeks in the long-term toxicity study (20 animals/sex/group) or for 106 weeks in the carcinogenicity study (50 animals/sex/group), after which they were killed.

Animals were examined daily in both experiments for signs of ill health or behavioral changes. Feed intake and body weights were recorded prior to administration of the test substance, at weekly intervals for the first 12 weeks, and then every 4 weeks until the completion of the study. Animals were monitored twice daily for mortality. Those found dead or killed moriband, as well as those killed at the end of the study underwent complete necropsies; organs were removed, weighed, and histologically examined. In the long-term study, cecum and colon diameters were measured. Ten animals/sex/group were subjected to ophthalmoscopic examination prior to the start of treatment and at weeks 13, 26, and 52 in the chronic study. Hematological examinations, blood chemistry tests, and urinalyses were performed on 10 animals/sex/group at weeks 14, 26, and 51 of the study.

In the carcinogenicity study, mortality was not affected by treatment and no treatment-related clinical signs were observed. Body weights of all treated males and high-dose group females were comparable to those of animals in their respective control groups, whereas mean body weights of low- and high-dose females were slightly lower than those of controls. Feed consumption was not affected by treatment and no gross pathological treatment-related changes were observed in any organs, including the intestine and cecum. There were occasional masses or nodules of the adrenal glands noted, but they were not dose-related.

Table 4 summarizes the histopathological changes related to treatment, which were observed in the adrenal gland. Both benign and malignant phaeochromocytomas occurred with higher incidences in male and female rats of the high-dose group when compared to the control group. Additionally, there was slight to moderate medullary hyperplasia occurring at increased frequency in all treated groups.

In females, there was an increased incidence of mammary gland adenocarcinomas: 4/50 (8%), 2/43 (4.6%), 8/50 (18.6%, P = 0.054), and 10/50 (20%, P = 0.044) in the controls and low-, mid- and high-dose animals, respectively. There were no observable increases of mammary gland adenomas or fibroadenomas observed (Herrman 1993).

In a study by Modderman (1993), the authors determined that long-term consumption of hydrogenated starch hydrolysates (HSH) in drinking water at a concentration of 18% (w/v) did not indicate any potential of carcinogenicity.

Co-carcinogenicity

Tumor Inhibition

Tsukamura et al. (1998) examined the effects of Maltitol on the incidence of 1,2-dimethylhydrazine (DMH)-induced colon cancer in rats. The authors noted that Maltitol is fermented in the colon due to partial hydrolysis in the small intestine. Eighty-nine male F344 rats (4 wks old) were used for the first experiment. The animals were fed a fiber-free diet supplemented with 1 or 5 g/100 g Maltitol for 27 weeks. The composition of the experimental diets are summarized in Table 5. Each group of rats were injected with DMH or vehicle alone for the first 14 weeks of the study. Maltitol supplementation at 1 g/100 g of the diet significantly reduced tumor incidence in the cecum and the 5% supplement reduced tumor incidence in both the cecum and proximal colon in DMH-treated rats.

In the second experiment, the effect of the 1 g Maltitol diet on the short chain fatty acid dose in cecal contents of placebo and DMH-treated rats was investigated. Forty-three male F344 rats (4 wks old) were used.

Rats were randomly divided into 2 diet groups: control (fiber-free) diet and 1% Maltitol diet group. Intake of the 1 g Maltitol diet doubled ($P < 0.05$) the dose of butyrate, but did not affect acetate or propionate in the cecal contents.

According to the authors, the results suggest that dietary Maltitol has a protective effect against DMH-induced tumors in rat cecum and proximal colon and that butyrate produced by bacterial fermentation of Maltitol in the cecum may be involved in the protection (Tsukamura et al. 1998.

CLINICAL ASSESSMENT OF SAFETY

Shiseido Research Center (2008a) performed a primary skin irritation test of Maltitol (69.09%) in humans in a 24 h closed patch test. Fifty-four healthy female volunteers were used in the study. The test material was applied to an adhesive patch and placed on the intact forearm of the subjects for 24 h. The plaster was removed and skin responses were scored. Positive skin reactions were not observed in 54 volunteers at 24 h after application of the test material. It was therefore concluded by the uthors that Maltitol does not possess a skin irritation potential under the test conditions.

Shiseido Research Center (2008b) performed a 24 h closed patch test to evaluate the primary skin irritation potential of Maltitol (64.5%) in 51 healthy female volunteers. The test material was applied to an adhesive path and placed on the intact forearm of the subjects for 24 h. The plaster was removed and skin responses were scored. Positive skin reactions were not observed in 51 volunteers at 24 h after application of the test material. It was concluded that Maltitol does not possess a skin irritation potential under the test conditions.

Shiseido Research Center (2008c) performed a 24 h closed patch test to evaluate Maltitol (53.2%) in 55 healthy female volunteers. The test material was applied to an adhesive patch, and was placed on the intact forearm of the subjects for 24 h. The plaster was removed and skin responses were scored. Positive skin reactions were not observed in any of the 55 volunteers at 24 h following application of the test material. It was concluded that the test material does not posses a skin irritation potential under the conditions of this test.

Table 4. Numbers of animals with specific histopathological changes observed in the adrenal gland in rats given a commercial preparation containing Maltitol (Herrman 1993).

Endpoint[a]	Male rats				Female rats			
	Dose Level[b]							
	0	0.5	1.5	4.5	0	0.5	1.5	4.5
Medullary hyperplasia	24	32	38	32	14	22	24	34
Phaeochromocytoma								
Benign	8	4	10	20	2	2	4	10
Malignant	6	12	4	10	2	2	2	4
Total Phaeochromocytoma	14	16	14	30	4	4	6	14

[a] 50 adrenal glands/sex/group were examined except for mid-dose males, in which 49 were examined.
[b] g/kg body weight per day

Table 5. Composition (g/100 g dry matter) of Experimental Diets (Tsukamara et al. 1998).

Ingredient	Diet		
	Control	1% Maltitol	5% Maltitol
Casein, milk	20	20	20
Soybean oil	5	5	5
$_{DL}$ -Methionine	0.3	0.3	0.3
Mineral mix (AIN-76)	3.5	3.5	3.5
Vitamin mix (AIN-76)	1.5	1.5	1.5
Choline bitartrate	0.2	0.2	0.2
Inositol	0.01	0.01	0.01
Cornstarch	50	50	50
Maltitol	0	1	5
Sucrose	to make 100 g		

Case Report

Azami (2000) reported on paralytic ileus accompanied by pneumatosis cystoides intestinalis (PCI). An 87-yr old woman, who was diagnosed with diabetes at the age of 73, began to experience abdominal distention and appetite loss. She had received acarbose, as well as 5 mg/day of gibenclamide, and had habitually used about 100 g Maltitol daily for about a year. Her symptoms subsided quickly with discontinuation of diet or cessation of acarbose and Maltitol usage. The patient's condition appeared to be attributable to gas levels produced by fermentation of disaccharides and Maltitol.

Dietary Effects

Abraham et al. (1981) reported on a non-calorigenic sweetener containing 58% Maltitol (by weight). The sweetener, according to the authors, has no influence on hematological and biochemical parameters. The dose that could be tolerated without effects was between 20 - 30 g/day. Above this dose, flatus production with abdominal discomfort was observed.

Koizumi et al. (1983) examined the laxative effects of sorbitol and Maltitol. Maltitol or sorbitol was administered at doses of 0.8 g/kg to 20 healthy subjects (10 males, 10 females) and 6 diabetic patients (3 males, 3 females). The average age was 35 ± 7.6 yrs for males and 39 ± 6.1 for females. Maltitol and sorbitol caused diarrhea in 75% and 95% of the patients, respectively. Stool was watery in most of the subjects. The serum concentration of each sweetening agent was as low as 0.3 mg/dl 2 hrs after administration. The serum concentrations of sodium, potassium, chlorine, urea nitrogen (BUN), glucose and insulin did not change 2 hrs after administration.

Elias and Homburger (1986) stated that Maltitol consumption should be limited due to its laxative effect when ingested in excessive quantities.

Storey et al. (1998) investigated the gastrointestinal effects of ingesting Maltitol in chocolate and whether any gastrointestinal effects were dose-related. In a double-blind, crossover study, 20 healthy volunteers (ages 18 - 24 yrs) ingested 100g chocolate containing 40g sucrose, 10g sucrose plus 30g Maltitol after fasting and not fasting. The was no difference in the effects between fasting and non-fasting periods, and order of consumption had no effect on symptomology. Relative to ingestion of sucrose, 30 g Maltitol caused no significant difference in symptoms, but 40g resulted in mild borborygmi ($P < 0.05$) and mild flatulence ($P < 0.01$), but not moderate or severe symptoms. Neither 30g Maltitol nor 40g Maltitol/sucrose caused significantly greater laxation than sucrose ingestion ($P > 0.05$).

In a separate study, 10 healthy volunteers (ages 18 - 24 yrs) ate the same test materials before breath molecular hydrogen (H_2) testing. Forty g Maltitol in chocolate caused a greater total breath H_2 excretion compared with 30g Maltitol compared with sucrose ($P < 0.05$). This dose-related response was consistent with the lower symptomology after ingestion of 30 vs. 40g Maltitol. The authors suggested that 30g Maltitol in chocolate causes no significant symptomology in young adults; however, 40g Maltitol caused borborygmi and flatus but no increased laxation. An increased H_2 response indicated colonic fermentation of Maltitol (Storey et al.1998).

Ruskoné-Fourmestraux et al. (2003) evaluated the gastrointestinal tolerance to an indigestible bulking sweetener containing sugar alcohol using a double-blind random cross-over study. Twelve healthy volunteers ingested Maltitol or sucrose throughout the day, either occasionally (once a week for each sugar, first period) or regularly (every day for two 9 day periods, second period). In both patterns of consumption, daily sugar doses were increased until diarrhea and/or a severe digestive symptom occurred, at which the dose level was defined as the threshold dose (TD).

In the first period (occasional consumption), the mean TD was 92 ± 6 g with Maltitol and 106±4 g with sucrose (P=0.059). The mean intensity of digestive symptoms was 1.1 and 1.3, respectively (P=NS). Diarrhea appeared in 6 and 1 subjects respectively (P=0.035). In the second period (regular consumption), the mean TD was 93±9 g with Maltitol and 113±7

g with sucrose (*P*=0.008). The mean intensity of digestive symptoms was 1.7 and 1.2, respectively (*P*=NS). However, diarrhea appeared in 8 and 3 subjects, respectively (*P*=0.04). Maltitol and sucrose TDs between the 2 periods were not statistically different.

According to the authors, under these experimental conditions and in comparison to sucrose: (a) occasional or regular consumption of Maltitol is not associated with severe digestive symptoms; (b) in both patterns of Maltitol consumption, diarrhea frequency is higher, but it appeared only for very high doses of Maltitol, much greater than those currently used; and (c) Maltitol does not lead to intestinal flora adaptation after a 9 day period of consumption (Ruskoné-Fourmestraux et al. 2003).

SUMMARY

Maltitol functions in cosmetics as a flavoring agent, humectant, and a skin-conditioning humectant. Maltitol Laurate functions as as an emulsion stabilizer and skin-conditioning agent in cosmetics. Maltitol was reported to be used in 33 cosmetic formulations with a concentration range of 0.0009% to 15%. No uses of Maltitol Laurate were reported.

Maltitol is a sugar alcohol produced by the hydrogenation of maltose. Material that contains 50 - 90% Maltitol has been considered as hydrogenated glucose syrup, now referred to as Maltitol syrup.

Maltitol syrup is a water solution of a hydrogenated, partially hydrolyzed starch containing Maltitol, sorbitol, and hydrogenated oligo- and polysaccharides.

Maltitol is stable under normal temperatures and pressures. It is incompatible with oxidizing agents. Hazardous decomposition products of Maltitol are carbon dioxide and carbon monoxide.

The metabolism of Maltitol was compared to that of sucrose in a group of 8 normal human subjects (ages 24 ± 2 yr; weight: 96 ± 3% of their ideal body weight). Each subject ingested 30 g of Maltitol or 30 g of sucrose with a 1 week interval between the 2 studies. The protein oxidation was assumed by the study authors to remain constant throughout the study and to be equivalent to 6.235 x N, where N is the number of grams of nitrogen excreted in the urine per minute.

In the 2 experiments, the mean fasting plasma glucose levels (89 ± 2 mg/dl), insulin levels (11.4 ± 1.0 µU/ml) levels, mean basal glucose levels (1.41 ± 0.17 mg/kg· min), and the lipid oxidation rates (1.10 ± 0.09 mg/kg · min) did not differ. The change in glucose oxidation was significantly lower with Maltitol than with sucrose during the first 90 min after ingestion. It was slightly higher with Maltitol than with sucrose beginning from the 210th min. Maltitol resulted in a cumulated suprabasal glucose oxidation which amounted to 40% that obtained with sucrose after 180 min.

Maltitol was hydrolyzed less readily by endogenous enzymes and a considerable amount undergoes fermentation in the lower gastrointestinal tract. The small amount that is absorbed is excreted unchanged in the urine.

Large intake (~2 - 70 g/day) of non-digestable saccharides and sugar alcohol causes diarrhea in animals.

The maximum non-effective dose of Maltitol is approximately twice that of sorbitol. The authors noted that Maltitol is a potentially useful agent as an enhancer of the intestinal calcium absorption. The consumption of 10% Maltitol diet by rats resulted in increased calcium absorption.

Ingestion of polyglycitol and Maltitol syrups in diabetic and non-diabetic subjects resulted in a lower glycemic response than with glucose in the following order: Maltitol syrup < polyglycitol syrup < glucose, which reflects the relative proportion of glucose released by hydrolysis of each material.

Two studies were performed using (A) 10, 15, and 20% sucrose in feeding and (B) 20% HSH compared to 20% sorbitol, both in feed. It was determined by the author that 20% in feed may result in effects due to nutrient imbalance, therefore the concentration of 18% in water was used as the highest dose in the study, without exceeding the maximum tolerated dose. Drinking bottles were replaced 3 time per week.

At the end of the first experiment, 40 animals (10 males and 10 females from both the control and HSH-treated groups) were sacrificed. The remaining animals were sacrificed in 2 stages: 48 animals were sacrificed after 20.5 months and the rest at 24 months. The total number of spontaneous deaths in the control group, 21, was higher than for the HSH-treated group, 16. No mortality was observed for the first 8 months of the study. After 12 months, the cumulative mortality rate rose to 2% in both groups, and at 16 months was approximately 5% in the control group and 6% in the treated group. Thereafter, the mortality rate of the control group exceeded that of the HSH-treated animals. During the first week of the study, diarrhea was observed among HSH-treated animals, which all disappeared by the fourth week. The author determined that long-term consumption of HSH in drinking water at a concentration of 18% (w/v) did not induce signs of toxicity in rats.

A combined long-term chronic toxicity/carcinogenicity study was reported in rats using a commercial preparation (details not provided) containing approximately 87% Maltitol, which was fed to Crl:CD(SD)BR male and female rats at doses of 0, 0.5, 1.5, or 4.5 g/kg bw/day. Note, the highest dose corresponded to an average of about 10% of the commercial product in the diet. Rats were maintained on this diet for 52 weeks (20 animals/sex/group) after which they were killed. The rats were treated for 106 weeks.

Animals were examined daily in both experiments for signs of ill health or behavioral changes. Food intake and body weights were recorded prior to administration of the test substance, at weekly intervals for the first 12 weeks, and then every 4 weeks until the completion of the study. Animals were monitored twice daily for mortality. Those found dead or killed moriband, as well as those killed at the end of the study underwent complete necropsies; organs were removed, weighed, and histologically examined. In the long-term study, cecum and colon diameters were measured. Ten animals/sex/group were subjected to ophthalmoscopic examination prior to the start of treatment and at weeks 13, 26, and 52 in the chronic study. Hematological examinations, blood chemistry tests, and urinalyses were performed on 10 animals/sex/group at weeks 14, 26, and 51 of the study.

No animals in the mid- or high-dose groups died. Three animals in the control group and 4 in the low-dose group died due to accidents; none of these deaths were related to treatment. Also, no treatment-related clinical signs were noted, nor did treatment have an effect on body weight. In males, sporadic food consumption was noted, but no clear trend was observed. In females of the high-dose group, mean feed consumption was significantly less at 12 and 52 weeks than the other groups. No eye abnormalities were observed, nor were there significant differences noted in blood chemistry or urinalysis. Some differences were observed in hematological parameters, however, except for a decrease in leukocytes in the mid-dose females, none of these signs were shown at all observation times. After gross or histopathological examination, no treatment-related effects were observed. There was an increase in cecum diameter in males of the high-dose group, which the authors concluded to be due to higher values in 3 out of 20 rats; the opposite was observed for females of the low- and high-dose groups. In this study the no adverse effect level (NOEL) was the highest dose tested - 4.5 g commercial product/kg bw/day.Sorbitol, mannitol, xylitol, lactitol, Maltitol, maltitol syrup, isomalt, and lactose have been demonstrated to be nonmutagenic and nongenotoxic in a variety of in vitro test systems, including the Ames test, both with and without the presence of metabolic activation. The authors concluded that these polyols are nonmutagenic and nongenotoxic.

Maltitol was administered by gavage to the animals from day 6 through day 18 of pregnancy at doses of 1.25, 2.5, or 5 g/kg/day. At 5 g/kg/day only there was an increase in the number of early resorptions, and increased post-implantation losses. No effects were observed in any treated group on maternal body weight increase, number of viable and dead fetuses, or on fetal body weights. No malformed fetuses were found at any of the doses administered.

The cytogenic effects on Maltitol was evaluated in human peripheral lymphocytes. Maltitol did not induce sister chromatid exchanges at all concentrations (1.25, 2.5, and 5 mg/mL) and treatment periods (24 and 48 hrs). Maltitol induced chromosome aberrations and the frequency of micronucleus formation at 24 and 48 hrs in a non dose-dependent manner. Maltitol did not decrease the replication index or the mitotic index at all doses and treatment periods, nor did it alter the pH or osmolality of the medium. Maltitol has a weak genotoxic potential and appears to be non-cytotoxic to human peripheral lymphocytes in vitro.

In acute oral toxicity, primary skin irritation, eye irritation and human patch testing studies using 69.09% Maltitol, no irritation was observed.

In a human patch tests using 65.45% and 53.2% Maltitol, respectively, no irritation were observed in 51 and 55 healthy female volunteers after 24 h.

DISCUSSION

The CIR Expert Panel considered that this class of chemicals, sugar alcohols, generally have a high water solubility. Chemicals with such high water solubility are not readily absorbed from the skin. Also based on basic chemistry, the Panel noted that sugar alcohols will not absorb significant amounts of UV light. Therefore, photosensitization or photoirritation are not safety concerns.

Overall, the Panel noted that Maltitol was not toxic in acute, subchronic, and chronic animal toxicity studies. In human patch tests, Maltitol was not irritating at levels up to 69.09%. While no safety test data were available for Maltitol Laurate, the Panel concluded that the safety Maltitol Laurate may be inferred based on the available data for Maltitol and for Lauric Acid, the two hydrolysis products of Maltitol Laurate (a previous safety assessment of Lauric Acid completed by the CIR Expert Panel found Lauric Acid safe as then used in cosmetics at levels up to 25%).

In the absence of inhalation toxicity data, the Panel determined that Maltitol can be used safely in hair sprays, because the ingredient particle size is not respirable. The Panel reasoned that the particle size of aerosol hair sprays (~38 μm) and pump hair sprays (>80 μm) is large compared to respirable particulate sizes (≤10 μm). The Panel did note that the available inhalation toxicity data for Lauric Acid demonstrated an absence of toxicity as expected.

The CIR Expert Panel recognized that there are data gaps regarding use and concentration of these ingredients. However, the overall information available on the types of products in which these ingredients are used and at what concentrations indicate a pattern of use, which was considered by the Expert Panel in assessing safety. Although Maltitol Laurate is not in current use, it is expected that, were it to be used, it would be used in product types and at concentrations similar to Maltitol. Accordingly, the CIR Expert Panel concluded that the available data are sufficient to support the safety of Maltitol and Maltitol Laurate as cosmetic ingredients in the practices of use and concentration as described in the safety assessment.

CONCLUSION

The CIR Expert Panel concluded that Maltitol and Maltitol Laurate are safe as cosmetic ingredients in the practices of use and concentration as described in this safety assessment.[1]

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