Amended Safety Assessment of Lard and Lard-Derived Ingredients as Used in Cosmetics

Status: Re-Review for Panel Review
Release Date: March 17, 2017
Panel Meeting Date: April 10-11, 2017

The 2017 Cosmetic Ingredient Review Expert Panel members are: Chair, Wilma F. Bergfeld, M.D., F.A.C.P.; Donald V. Belsito, M.D.; Ronald A. Hill, Ph.D.; Curtis D. Klaassen, Ph.D.; Daniel C. Liebler, Ph.D.; James G. Marks, Jr., M.D.; Ronald C. Shank, Ph.D.; Thomas J. Slaga, Ph.D.; and Paul W. Snyder, D.V.M., Ph.D. The CIR Director is Lillian J. Gill, D.P.A. This report was prepared by Lillian C. Becker, Scientific Analyst/Writer.

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Cosmetic Ingredient Review

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MEMORANDUM

To: CIR Expert Panel and Liaisons

From: Lillian C. Becker, M.S.
Scientific Analyst and Writer

Date: March 17, 2017

Subject: Re-review of Lard and Lard-Derived Ingredients

Attached is the re-review of Lard and Lard-Derived Ingredients. [*Lard042017rep*] According to CIR procedures, re-reviews are to be conducted 15 years after a safety assessment is published. In the safety assessment published in 2001, the Cosmetic Ingredient Review (CIR) Expert Panel stated that these ingredients are safe as used in cosmetic products, provided that established limitations imposed on heavy metal and pesticide concentrations are not exceeded. These limits are: lead, not more than 0.1 ppm; arsenic (as As), ≤3 ppm; mercury (as Hg), ≤1 ppm; and total PCB/pesticide contamination, not more than 40 ppm, with not more than 10 ppm for any specific residue. [*Lard042017Prev*]

After an exhaustive search, no new toxicity data were discovered. However, impurity data were discovered and included in this re-review.

Updated concentration of use data were submitted and incorporated into the re-review. [*Lard042017Data_1,2*] The maximum concentration of use of Lard Glyceride in the original report was ≤10%; it is now reported to be 1.6%. There were no concentrations of use reported in a 2016 industry survey for any of the other ingredients in this report. In 1984, Lard was reported to be used at ≤10% and Hydrogenated Lard Glyceride was reported to be used at ≤1%. The number of reported uses of Lard increased from 3 to 4. [*Lard042017FDA*] Hydrogenated Lard Glyceride decreased in number of uses from 6 to 1. Lard Glyceride remained at 2 uses. No other data were submitted.

The Panel is now being asked to consider whether there is a reason to re-open the review, or should the original conclusion be reaffirmed, in which case the review would not be re-opened.

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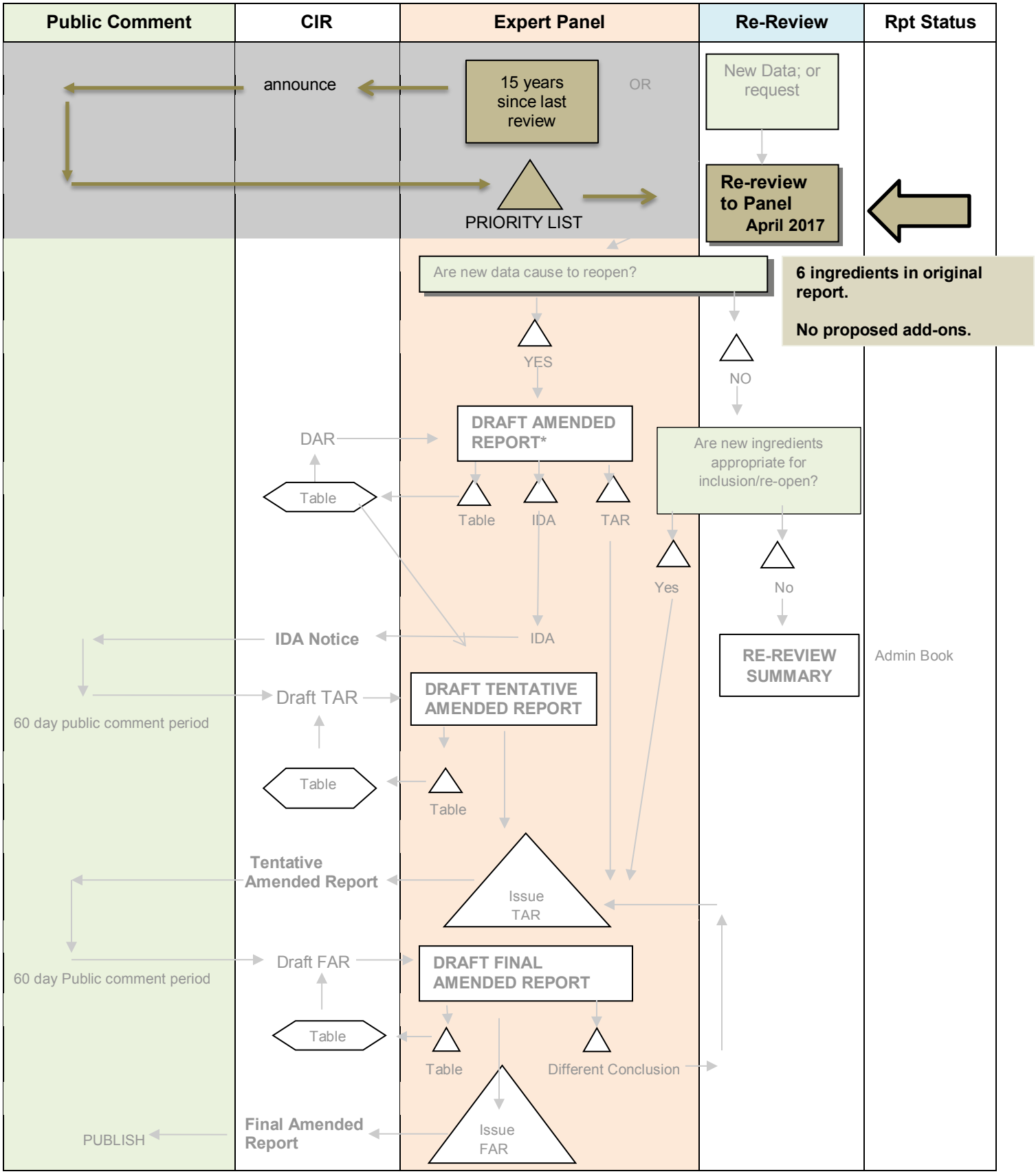
RE-REVIEW FLOW CHART

INGREDIENT/FAMILY Lard and Lard-Derived Ingredients

MEETING April 2017



*If Draft Amended Report (DAR) is available, the Panel may choose to review; if not, CIR staff prepares DAR for Panel Review.

History of Lard and Lard-Derived Ingredients

2001 - The safety assessment was published with the conclusion that these ingredients are safe as used in cosmetic products, provided that established limitations imposed on heavy metal and pesticide concentrations are not exceeded.

2016 – This report is due to be re-reviewed

April, 2017 – The Panel examines the re-review packet. There is no new toxicity data. There is new use and impurity data.

Lard-Derived Ingredients Data Profile for ***April*, 2017**. Writer – Lill Becker																					
	ADME			Acute toxicity			Repeated dose toxicity			Irritation					Sensitization						
	Dermal Penetration	Log K_{ow}	Use 2016	Oral	Dermal	Inhale	Oral	Dermal	Inhale	Ocular Animal	Ocular In Vitro	Dermal Animal	Dermal Human	Dermal In Vitro	Animal	Human	In Vitro	Repro/Devel	Genotoxicity	Carcinogenicity	Phototoxicity
Lard							O											O	O	O	
Hydrogenated Lard							O														
Lard Glyceride			X																		
Hydrogenated Lard Glyceride			X																		
Lard Glycerides																					
Hydrogenated Lard Glycerides																					

O = Old Data

N = New Data (there isn't any)

Lard and Lard-Derived Ingredients

Ingredient	CAS #	InfoBase	SciFinder	PubMed	TOXNET	FDA	EU	ECHA	IUCLID	SIDS	HPVIS	NICNAS	NTIS	NTP	WHO	FAO	FEMA	Web
Lard Glyceride	61789-10-4	0	0	1	0	Y	No	N	3/0	N	N	N	N	N	N	N	N	N
Hydrogenated Lard Glyceride	8040-05-9 91744-55-7	0	0	1	0	N	No	N	N	N	N	N	N	N	N	N	N	N
Lard Glycerides	91744-46-6	0	0	1	0	Y	No	N	N	N	N	N	N	N	N	N	N	N
Hydrogenated Lard Glycerides	91744-48-8	Old data	0	1	0	N	No	N	N	N	N	N	N	N	N	N	N	N
Lard	61789-99-9	CFR	430/0	1759/0	0	Y	No	N	N	N	Y	N	N	N	N	N	N	N
Hydrogenated Lard	73138-67-7	CFR	0	2/0	Not useful	N	No	N	N	N	Y	N	N	N	N	N	N	N

Minutes of Lard and Lard-Derived Ingredients

March 1998
66th Meeting of the Expert Panel

Lard Glyceride, Hydrogenated Lard Glyceride,
Lard Glycerides, Hydrogenated Lard Glycerides,
Lard, and Hydrogenated Lard

Dr. Schroeter said that his Team agreed that the following data are needed to complete the Panel's safety assessment of this group of ingredients:

(1) Current concentration of use
(2) Impurities/contaminants of cosmetic grade ingredients (especially pesticides and halogenated compounds)*
(3) Dermal irritation and sensitization
(4) Ocular toxicity, if available

*Depending on this information, gross pathology and histopathology of skin and other major organ systems associated with repeated dermal exposures and genotoxicity data may be needed

Dr. Schroeter also noted that his Team determined that UV absorption data are not needed.

Dr. Belsito wanted to know why items 3 and 4 are included in the preceding list of data requests. He said that one would not expect Lard to cause dermal irritation and sensitization or ocular toxicity, and that only items 1 and 2 are needed.

Dr. Shank noted that on many occasions, data have not been submitted in response to the Panel's request for impurities data, and the Panel subsequently had to request skin irritation/sensitization data.

With Dr. Shank's comments in mind, Dr. Belsito wanted to know why UV absorption data should be deleted from the list of data requests. Dr. Belsito noted that it is possible that a photoabsorbing impurity or a sensitizing impurity may be present.

Dr. Schroeter agreed that UV absorption data should also be requested.

The Panel voted unanimously in favor of issuing an Insufficient Data Announcement with the following data requests:

(1) Current concentration of use
(2) Impurities/contaminants of cosmetic ingredients (especially pesticides and halogenated compounds); depending on this information, gross pathology and histopathology of the skin and other major organ systems associated with repeated dermal exposures and genotoxicity data may be needed
(3) UV absorption; if there is significant absorption, then a photosensitization study will be needed
(4) Skin irritation and sensitization
(5) Ocular toxicity, if available

September, 1998
68th Meeting of the Expert Panel

Lard Glyceride, Hydrogenated Lard Glyceride,
Lard Glycerides, Hydrogenated Lard Glycerides,
Lard, and Hydrogenated Lard

Dr. Schroeter recalled that an insufficient data announcement on this group of ingredients was issued at the March 19-20, 1998 Panel meeting. The data requests included in this announcement were as follows:

(1) Current concentration of use
(2) Impurities/contaminants of cosmetic ingredients (especially pesticides and halogenated compounds); depending on this information, gross pathology and histopathology of the skin and other major organ systems associated with repeated dermal exposures and genotoxicity data may be needed

(3) UV absorption; if there is significant absorption, then a photosensitization study will be needed
(4) Skin irritation and sensitization
(5) Ocular toxicity, if available

Dr. Schroeter noted that if a limiting statement (no pesticides or heavy metal residues) were included in the report discussion, his Team would be able to conclude that this ingredient family is safe as used. In other words, contaminants could be eliminated by using food grade Lard/Lard compounds.

Dr. Shank said that rather than mentioning the contaminants in the report discussion, it could be stated that food grade Lard/Lard compounds should be used in cosmetics.

Dr. McEwen said that some reference in which "food grade" is defined in terms of ingredient composition should be cited in the report discussion.

Dr. Belsito said that the Panel should cite its own specifications.

Dr. Andersen said that the Panel will probably be in a position to do both. He said that if limitations included in the Food Chemicals Codex are appropriate, then there is no reason why this information should not be mentioned.

Dr. Bergfeld wanted to know whether any limitations associated with the definition of "food grade" should be included in the report discussion or in the conclusion.

Dr. Bailey favored including this information in the report conclusion, because, in his opinion, most individuals will pay attention to the conclusion.

Referring to the Panel's review of Acid Violet 43, Dr. Andersen noted that the specifications were important to the Panel's determination of safety and were added to the report conclusion.

Dr. Bailey wanted to know which specifications would be used.

Dr. Andersen said that the Food Chemicals Codex specifications or the USP lanolin limits for pesticides, etc. would be used.

The Panel voted unanimously in favor of issuing a Tentative Report with a safe as used conclusion on Lard Glyceride, Hydrogenated Lard Glyceride, Lard Glycerides, Hydrogenated Lard Glycerides, Lard, and Hydrogenated Lard.

Dr. Bergfeld confirmed that the Panel unanimously approved the conclusion, discussion, and restrictions.

March 3-4, 1999
70th Meeting of the Expert Panel

Lard Glyceride, Hydrogenated Lard Glyceride,
Lard Glycerides, Hydrogenated Lard Glycerides,
Lard, and Hydrogenated Lard

Dr. Belsito recalled that the Panel issued a Tentative Report with the following conclusion at the September 10-11, 1998 Panel meeting: Based on the available data, the CIR Panel concludes that Lard Glyceride, Hydrogenated Lard Glyceride, Lard Glycerides, Hydrogenated Lard Glycerides, Lard, and Hydrogenated Lard are safe as used in cosmetic products, provided that established limitations imposed on heavy metal and pesticide concentrations are not exceeded. Dr. Belsito also noted that the limitations on lead (<0.1 mg/kg, or 0.1 ppm), arsenic (3 ppm), mercury (1 ppm), and total PCB/pesticide contamination (<40 ppm), with not more than 10 ppm for any specific residue, are included in the report discussion.

The Panel voted unanimously in favor of issuing a Final Report with the conclusion stated in the preceding paragraph.

Dr. Schroeter noted that limitations on heavy metal impurities in Acid Violet 43 (used in rinse-off products) are used as a benchmark in the report discussion, but that the ingredients being reviewed are used in leave-on products. With this in mind, he said that some other benchmark (i.e., an ingredient that is used in leave-on products) should have been used.

Dr. Belsito said that though Acid Violet 43 is a rinse-off hair dye, the impurities restrictions used in

the CIR report on this ingredient were actually those stated for a food colorant. This was done because all of the toxicity data available for the safety assessment of Acid Violet 43 were on the food dye.

Dr. McEwen recalled that because the Panel did not have sufficient information on Acid Violet 43 (non-certified) dye but did have sufficient information on the certified dye, the Panel agreed to adopt the specifications on the certified dye that are found in the Code of Federal Regulations, assuming that the certified dye would be safe for hair dye use.

Dr. Shank did not disagree with the restrictions on heavy metals, but with the fact that it appears that the Panel is using a hair dye as a reference to heavy metal contaminants for a lipid.

Dr. Belsito said that the Panel could consult the CIR report on Acid Violet 43 for the original reference for the restrictions on heavy metal impurities and use it in the current report on the Lard Glyceride ingredient family.

The Panel concurred with Dr. Belsito's proposal.

The Final Report conclusion unanimously approved by the Panel is stated in the first paragraph of this section.

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INTRODUCTION

In a safety assessment published in 2001 of Lard and Lard-derived ingredients, the Cosmetic Ingredient Review (CIR) Expert Panel (Panel) stated that these ingredients are safe as used in cosmetic products, provided that established limitations imposed on heavy metal and pesticide concentrations are not exceeded.[1] These limits are: lead, not more than 0.1 ppm; arsenic (as As), ≤3 ppm; mercury (as Hg), ≤1 ppm; and total PCB/pesticide contamination, not more than 40 ppm, with not more than 10 ppm for any specific residue. The six ingredients in this re-review are:

Lard
Hydrogenated Lard
Lard Glyceride
Hydrogenated Lard Glyceride
Lard Glycerides
Hydrogenated Lard Glycerides

Because it has been 15 years since the safety assessment on Lard and Lard-derived ingredients was published, the Panel is being asked to determine, based on data presented in this report, whether a re-review is warranted or the original conclusion can be reaffirmed.

The reported functions of these ingredients include emulsion stabilizer, skin-conditioning agent – emollient, and viscosity increasing agent (Table 1).[2]

An exhaustive search was conducted for additional safety test data that have entered the literature since the 2001 final report was published. No new safety data were discovered. Updated use and impurity data are presented.

Summaries of data on Lard and Lard-derived ingredients from the original report are included in the appropriate sections in *italics*. Please see the original report for details [http://www.cir-safety.org/ingredients].

CHEMISTRY

Definition and Structure

The definitions and functions of the Lard-derived ingredients in this report are presented in Table 1.

The European Union (EU) defines Lard as the purified internal fat of the hog.[3] It consists primarily of stearin, palmitin and olein (Figure 1).

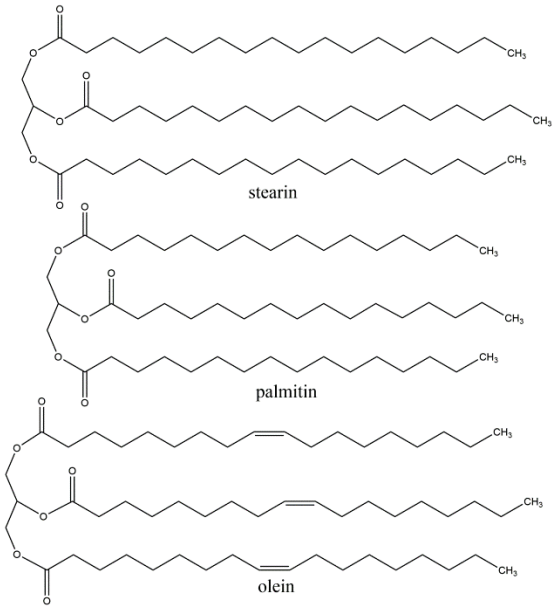


Figure 1. Primary composition of Lard.

Physical and Chemical Properties

New physical and chemical properties of the Lard-derived ingredients were not found in the published literature and no unpublished data were submitted.

Lard

*Lard is described as a soft, white unctuous substance with a bland taste and a characteristic odor (*Table 2*).*[1] *About 200 fatty acids are found in Lard. The chief constituents are stearic, palmitic, and oleic acids.*

Method of Manufacture

Lard is obtained by dry or wet rendering of fresh fatty porcine tissues (cuttings and trimmings) shortly after slaughter. Lard produced by wet-rendering processes is known as prime steam lard. Rendered Lard may be bleached, or bleached and deodorized.

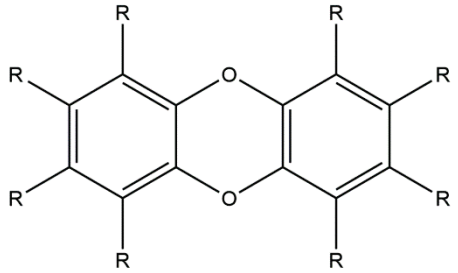
Impurities

From the Discussion: *Lead is limited to not more than 0.1 mg/kg (0.1 ppm). This value is adopted from the* Food Chemical Codex (FCC) *limit for unhydrogenated Lard (National Academy of Sciences 1996). Because the FCC did not address the presence of other heavy metals in Lard, the Panel adopted the limits found in 21 CFR 73&74. Those limits are ≤3 ppm arsenic (as As) and ≤ l ppm mercury (as Hg) (CIR 1998).*

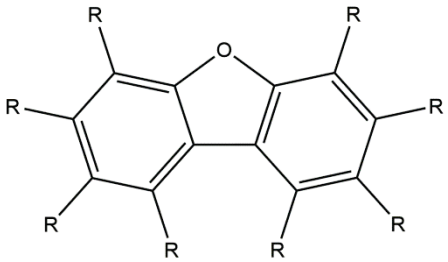
The Panel limited the total PCB/pesticide contamination to not more than 40 ppm with not more than 10 ppm for any specific residue. These limits are modeled after the USP standards for lanolin (Committee of Revision of the United States Pharmacopeial Convention 1995).

Dioxin and chemically-related compounds are a group of environmental contaminants found throughout the world.[4] Studies suggest that exposure to these chemicals may lead to a variety of adverse health effects including reproductive and developmental problems, cardiovascular disease, increased diabetes, and increased cancer. Because they tend to accumulate in the fat of food-producing animals, consumption of animal-derived foods (e.g., meat, poultry, eggs, fish, and dairy products) is considered to be the major route of human exposure to low levels of Dioxin and chemically-related compounds.

The FDA used toxic equivalents (TEQ) to calculate the toxicity-weighted concentrations of polychlorinated dibenzo-*p*-dioxin (PCDD) and polychlorinated dibenzofuran (PCDF) in 20 samples of food grade Lard (Figure 2).[4,5] TEQ expresses the toxicity-weighted concentrations of dioxins, furans and PCBs relative to the most toxic form of dioxin, 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD). In this analysis, the concentration of PCDDs and PCDFs in some of the samples were below the limit of detection; the average toxicity-weighted concentrations of these substances in the samples was calculated three ways, by assigning results below the limit of detection a value of zero, half the limit of detection, or equal to the limit of detection. By these methods, the average TEQ of PCDDs and PCDFs in the Lard samples was 0.12, 0.13, and 0.15 pg/g, respectively, depending on the value assigned to the non-detects.



polychlorinated dibenzo-*p*-dioxin (PCDD)
(wherein each R is Cl or H,
commonly 5 are Cl)



polychlorinated dibenzofuran (PCDF)
(wherein each R is Cl or H,
commonly 5 are Cl)

Figure 2. PCBs

USE

Cosmetic

The safety of the cosmetic ingredients included in this assessment is evaluated based on data received from the U.S. Food and Drug Administration (FDA) and the cosmetics industry on the expected use of these ingredients in cosmetics. Use frequencies of individual ingredients in cosmetics are collected from manufacturers and reported by cosmetic product category in FDA's Voluntary Cosmetic Registration Program (VCRP) database. Use concentration data are submitted by

Industry in response to surveys, conducted by the Personal Care Products Council (Council), of maximum reported use concentrations by product category.

According to VCRP survey data received in 2017, Lard was reported to be used in use in 4 bath soaps and detergents; this is an increase from the 3 formulations reported in 1998 (Table 3).[6] Lard Glyceride was reported to be used in 2 formulations (a face and neck formulation and a moisturizing formulation); in 1998, Lard Glyceride was reported to be used in 2 formulations (a body and hand care formulation and a moisturizing formulation). Hydrogenated Lard Glyceride was reported to be used in 1 formulation (eyebrow pencil), down from 6 formulations (4 eye formulations, a lipstick, and a makeup formulation) in 1998.

The results of the concentration of use survey conducted by the Council in 2016 indicate that Lard Glyceride was reported to be used at up to 1.6% in tonics, dressings and other hair grooming aids; Lard Glyceride was reported to be used at ≤10% in 1984 (types of formulations not specified).[7] There were no reported concentrations of use in the 2016 Council survey for any of the other ingredients in this report. In 1984, Lard was reported to be used at concentrations ≤10% and Hydrogenated Lard Glyceride was reported at ≤1%.

There were no current or historical uses reported for:
Hydrogenated Lard
Lard Glycerides
Hydrogenated Lard Glycerides

In some cases, reports of uses were received in the VCRP, but concentration of use data were not provided. For example, Lard was reported to be used in 4 cosmetic formulations, but no use concentration data were reported.

All of the Lard-derived ingredients named in this report are not restricted from use in any way under the rules governing cosmetic products in the European Union.[8]

Non-Cosmetic

Lard and lard oil are considered to be generally recognized as safe (GRAS) for the intended use when used in cotton and cotton fabric dry food packaging materials.[9] [21CFR182.70] Lard oil (which includes Lard Glyceride and Lard Glycerides) may be used as a defoaming agent for use in paper and paperboard for use in packaging, transporting, or holding food. [21CFR176.210]

Lard and Hydrogenated Lard are exempt from the processing and use information report requirements of the Chemical Data Reporting (CDR) Rule [40CRF711.15(b)(4)] issued under the Toxic Substances Control Act (TSCA).[10] Chemical substances are included on the CDR list only if the Environmental Protection Agency (EPA) has determined that there is low current interest in the processing and use information for the substances.

TOXICOKINETIC STUDIES

Dermal Penetration

Toxicokinetics studies on the Lard-derived ingredients were not found in the published literature and no unpublished data were submitted.

TOXICOLOGICAL STUDIES

Acute Toxicity

Acute toxicity data of the Lard-derived ingredients were not found in the published literature and no unpublished data were submitted.

Short-Term Toxicity

New short-term toxicity studies of the Lard-derived ingredients were not found in the published literature and no new unpublished data were submitted.

Lard

Thomasson (1955) conducted a 6-week feeding study in which Wistar rats were fed Lard at concentrations such that 10% to 73% of the total dietary calories were derived from Lard.[1] The food-efficiency of Lard was comparable to summer butterfat, and no adverse effect on mortality was observed.

Five 2-month-old rats from a single litter were used.[1] *Two of the rats (rats 1 and 2) were fed bread, milk, and water ad libitum. The experimental rats (rats 3-5) were given 4 to 5 g of Lard daily in addition to the control diet. An adult female rat (rat 6) was also fed the high-fat diet and a male rat was alternately fed the control and high-fat diet (each for a 1-week period). Growth of test rats was "somewhat stunted." Livers of rats fed high-fat diets were enlarged, were uniformly yellow, diminished in consistency and the cut surface was moist, shining, and oily. A "more or less accentuated state of adiposis" was noted upon microscopic examination of hepatic cells.*

Subchronic Toxicity

No subchronic toxicity studies of the Lard-derived ingredients were found in the published literature and no unpublished data were submitted.

Chronic Toxicity

New chronic toxicity studies of the Lard-derived ingredients were not found in the published literature and no new unpublished data were submitted.

Lard

Groups of male C57BL mice were fed diets containing 25% Lard from weaning though life, age 6 months through life, age 12 months through life, or for 5-month periods beginning at age 1, 7, or 12 months.[1] *Body weight gains were greater in Lard-fed mice compared to control mice: gains were the least striking in mice fed Lard for 5 months or from age 12 months onwards. The data were not analyzed for statistical significance, but the investigators concluded that lifetime feeding of a high-fat diet shortened the life span, with the most dramatic effects noted in mice that had been fed fat since weaning. The effect on survival of a 5-month high-fat diet depended on the treatment period; "given during the period of growth, the high fat ration did not alter the life span; in young adults it had a beneficial effect, while given to old animals it exerted a slightly injurious action on the life span".*

Hydrogenated Lard

Weanling rats were fed 50% Hydrogenated Lard (or other saturated fats) and a control group was fed 50% Lard through life.[1] *Rats were killed at 8-week intervals for necropsy. No lesions were found at necropsy: in a few rats fed the saturated fat, the adipose tissue had a mottled appearance. A foreign body-type granulomatous reaction was noted in the adipose tissue at microscopic examination of rats fed Hydrogenated Lard or the other saturated fats: the lesion consisted of multinucleated cells with nuclei arranged about the periphery or in clumps. Often, the fat material appeared partially dissolved, leaving radially oriented slitlike spaces that suggested fatty acid crystals. No acute inflammatory reaction, hemorrhage, or birefringent material were associated with the reaction. The reaction occurred within 16 weeks of feeding the Hydrogenated Lard diet and within 8 weeks of feeding the other saturated fats; it was not observed in control rats.*

DEVELOPMENTAL AND REPRODUCTIVE TOXICITY (DART) STUDIES

New DART studies of the Lard-derived ingredients were not found in the published literature and no new unpublished data were submitted.

Lard

[These are studies from the 1940s]

Five 2-month-old rats from a single litter were used.[1] *Two of the rats (rats 1 and 2) were fed bread, milk, and water ad libitum. The experimental rats (rats 3-5) were given 4 to 5 g of Lard daily in addition to the control diet. An adult female rat (rat 6) was also fed the high-fat diet and a male rat was alternately fed the control and high-fat diet (each for a 1-week period). Beginning a few days into the study, test rats 3 to 5 reduced their feed intake, became emaciated, and crouched in a corner of the cage. They had swollen snouts, reddened and edematous eyelids with thinner eyelashes, rumpled fur, and extensively reddened and swollen external genitalia. The male had similar lesions when fed the high-fat diet and had conspicuous swelling of the scrotum and testicles and priapism. These alterations disappeared after 9 to 10 days, reoccurred periodically, and occurred in a "progressively more attenuated form in female rats." Reproductive activity was not affected. No differences were noted in number of offspring between control and test rats. Offspring of test rats appeared smaller than control offspring and were weakened, sluggish, poorly nourished, and cyanotic with dry and wrinkled skin. Offspring from two litters whose dams had been fed the high-fat diet for 2 months were "in better condition" than those litters from dams that had been fed the high-fat diet for >3 months prior to delivery. Livers of pups from test group were small, yellow, and soft. "Adiposis" was noted upon microscopic examination. Hepatic cells were filled with granules and droplets of fat that were*

abundant at the periphery of the cell and formed vacuoles in some cells. The nuclei frequently were displaced to the periphery and the cytoplasm appeared reduced in amount.

In a follow-up study, the same investigators fed three test rats with 4 to 5 g of Lard/daily (in addition to laboratory feed) throughout pregnancy and lactation (2.5-3.5 months). The results were similar to those of the study reported above. In addition, pups of the test group had stunted growth, dry and wrinkled skin, cyanosis, and sluggish movements. Test group pups began to grow fine hair after 13 to 14 days and opened their eyes after 18 to 21 days, whereas the time frame for control rat pups was l0 to 11 days and 13 to 14 days, respectively. Some test pups died a few days after birth and the survivors (killed after 25-27 days of nursing) had "substantially" lower body weights compared to controls. The average weight was 23.3 g for test group pups and 34.6 g for control pups.

A four-generation study was conducted to determine nutritional requirements for reproduction and lactation. Wistar and Evans-Long rats were fed diets containing 10% Lard, 5% Lard (plus 10% Crisco, a commercial shortening), 2% Lard (plus 8% Crisco), and a combination of Crisco plus corn oil. Growth was comparable among treated and control rats. Dams fed the experimental diets lost weight during lactation, and weaned fewer young than those fed the control diet. Supplementary Lard was supplied ad libitum and as much as 8 g/day was eaten; no improvement in lactation was noted and dams fed the supplement killed their young during lactation.

GENOTOXICITY STUDIES

New genotoxicity studies of the Lard-derived ingredients were not found in the published literature and no new unpublished data were submitted.

Lard

No significant increase in mutation frequencies was observed in transgenic mice fed diets containing 31% Lard for 5 and 9 weeks when compared to basal diet fed controls.[1] The investigators concluded that uncooked fats were not mutagenic and were not initiators of carcinogenesis in the gut epithelium.

CARCINOGENICITY STUDIES

New carcinogenicity studies of the Lard-derived ingredients were not found in the published literature and no new unpublished data were submitted.

Lard

Lard did not increase the mutation frequency in the intestinal epithelium of orally dosed transgenic mice.[1] Two studies that examined cell proliferation as an indicator of carcinogenesis reported greater [3H]-thymidine incorporation in cells of the mammary gland and colorectum region in mice fed 15% Lard (compared to mice fed plant-source fats). In another study, no increase in cell hyperplasia and thymidine incorporation were found in the stomach, pancreas, or colon of rats fed 27% Lard (compared to controls fed 5% corn oil). Rats fed fiber-free diets containing 80 g/kg of Lard had crypt cell production rates (in the small intestine) comparable to that of rats fed corn oil. Lard dosed rats had significantly smaller rates in the distal colon, whereas corn oil-dosed rats had significantly smaller rates in the cecum. Cocarcinogenic effects were observed when high fat diets were fed with known carcinogens to mice, rats, and hamsters.

DERMAL IRRITATION AND SENSITIZATION STUDIES

No irritation or sensitization studies of the Lard-derived ingredients were found in the published literature and no unpublished data were submitted.

OCULAR IRRITATION STUDIES

No ocular irritation studies of the Lard-derived ingredients were found in the published literature and no unpublished data were submitted.

SUMMARY OF NEW DATA

This is a re-review of Lard and Lard-derived cosmetic ingredients. In a safety assessment published in 2001 of Lard and Lard-derived ingredients, the CIR Panel stated that these ingredients are safe as used in cosmetic products, provided that

established limitations imposed on heavy metal and pesticide concentrations are not exceeded. These limits are: lead, not more than 0.1 ppm; arsenic (as As), ≤3 ppm; mercury (as Hg), ≤1 ppm; and total PCB/pesticide contamination, not more than 40 ppm, with not more than 10 ppm for any specific residue.

An exhaustive search for additional safety test data that have entered the literature since this final report was published was conducted. No new safety data were discovered. Updated use and impurity data were presented.

According to the VCRP survey data received in 2017, Lard was reported to be used in use in 4 bath soaps and detergents; this is an increase from the 3 formulations reported in 1998. Lard Glyceride was reported to be used in 2 formulations (a face and neck formulation and a moisturizing formulation); in 1998, Lard Glyceride was reported to be used in 2 formulations (a body and hand care formulation and a moisturizing formulation). Hydrogenated Lard Glyceride was reported to be used in 1 formulation (eyebrow pencil), down from 6 formulations (4 eye formulations, a lipstick, and a makeup formulation) in 1998.

The results of the concentration of use survey conducted by the Council in 2016 indicate that Lard Glyceride was reported to be used at up to 1.6% in tonics, dressings and other hair grooming aids; Lard Glyceride was reported to be use at ≤10% in 1984 (types of formulations not provided). There were no reported concentrations of use in the 2016 Council survey for any of the other ingredients in this report. In 1984, Lard was reported to be used at ≤10% and Hydrogenated Lard Glyceride was reported to be used at ≤1%.

There were no current or historical uses reported for:
Hydrogenated Lard
Lard Glycerides
Hydrogenated Lard Glycerides

DISCUSSION FROM ORIGINAL SAFETY ASSESSMENT[1]

The Cosmetic Ingredient Review (CIR) Expert Panel was of the opinion that Lard Glyceride, Hydrogenated Lard Glyceride, Lard Glycerides, Hydrogenated Lard Glycerides, Lard, and Hydrogenated Lard can be used safely in cosmetic formulations. However, the Panel recognized the need to limit the presence of heavy metals and/or polychlorinated biphenyl (PCB) or other pesticide contamination.

Lead is limited to not more than 0.1 mg/kg (0.1 ppm). This value is adopted from the Food Chemical Codex (FCC) limit for unhydrogenated Lard (National Academy of Sciences 1996). Because the FCC did not address the presence of other heavy metals in Lard, the Panel adopted the limits found in 21 CFR 73&74. Those limits are ≤3 ppm arsenic (as As) and ≤1 ppm mercury (as Hg) (CIR 1998).

The Panel limited the total PCB/pesticide contamination to not more than 40 ppm with not more than 10 ppm for any specific residue. These limits are modeled after the USP standards for lanolin (Committee of Revision of the United States Pharmacopeial Convention 1995).

TABLES

Table 1. Definitions and functions of Lard and Lard-derived ingredients in this safety assessment.[2]

Ingredient	Definition	Function(s)
Lard 61789-99-9	Lard is the purified fat obtained from the abdomen of the hog.	Skin-conditioning agent - occlusive
Hydrogenated Lard 73138-67-7	Hydrogenated lard is the end product of controlled hydrogenation of Lard.	Skin-conditioning agent – occlusive; viscosity increasing agent - nonaqueous
Lard Glyceride 61789-10-4	Lard Glyceride is the monoglyceride derived from Lard.	Emulsion stabilizer; Skin-conditioning agent – emollient; viscosity increasing agent - nonaqueous
Hydrogenated Lard Glyceride 8040-05-9 91744-55-7	Hydrogenated Lard Glyceride is the end product of controlled hydrogenation of Lard Glyceride.	Skin-conditioning agent – emollient; surfactant – emulsifying agent; viscosity increasing agent - nonaqueous
Lard Glycerides 91744-46-6	Lard Glycerides is a mixture of mono, di, and triglycerides derived from Lard.	Skin-conditioning agent – emollient; viscosity increasing agent - nonaqueous
Hydrogenated Lard Glycerides 91744-48-8	Hydrogenated Lard Glycerides is the end product of controlled hydrogenation of Lard Glycerides.	Skin-conditioning agent – occlusive; viscosity increasing agent - nonaqueous

Table 2. Chemical and physical properties of Lard.

Property	Value	Reference
	Lard	
Physical Form	Solid, soft	1
Color	White	1
Odor	Characteristic	1
Density	0.917	1
Melting Point °C	36-42	1
Water Solubility	Insoluble	1
Other Solubility		
Alcohol	Slightly soluble	1
Benzene	Soluble	1
Chloroform	Soluble	1
Ether	Soluble	1
Carbon disulfide	Soluble	1
Petroleum ether	Soluble	1

Table 3. Current and historical frequency and concentration of use of Lard and Lard-derived ingredients according to duration and exposure.[1,6,7]

	# of Uses		*Max Conc of Use (%)*		*# of Uses*		*Max Conc of Use (%)*	
	2017	**1998**	**2016**	**1984****	**2017**	**1998**	**2016**	**1984**
	Lard				**Lard Glyceride**			
Totals*	**4**	**3**	**NR**	**≤10[#]**	**2**	**2**	**1.6**	**≤10[#]**
Duration of Use								
Leave-On	*NR*	*2*	*NR*	*DNR*	*2*	*2*	*1.6*	*DNR*
Rinse-Off	*4*	*1*	*NR*	*DNR*	*NR*	*NR*	*NR*	*DNR*
Diluted for (Bath) Use	*NR*	*NR*	*NR*	*DNR*	*NR*	*NR*	*NR*	*DNR*
Exposure Type								
Eye Area	NR	NR	NR	DNR	NR	NR	NR	DNR
Incidental Ingestion	NR	NR	NR	DNR	NR	NR	NR	DNR
Incidental Inhalation-Spray	NR	NR	NR	DNR	1[a];1[b]	1[a];1[b]	1.6[a]	DNR
Incidental Inhalation-Powder	NR	NR	NR	DNR	1[b]	1[b]	NR	DNR
Dermal Contact	4	2	NR	DNR	2	2	NR	DNR
Deodorant (underarm)	NR	NR	NR	DNR	NR	NR	NR	DNR
Hair - Non-Coloring	NR	NR	NR	DNR	NR	NR	1.6	DNR
Hair-Coloring	NR	1	NR	DNR	NR	NR	NR	DNR
Nail	NR	NR	NR	DNR	NR	NR	NR	DNR
Mucous Membrane	4	NR	NR	DNR	NR	NR	NR	DNR
Baby Products	NR	NR	NR	DNR	NR	NR	NR	DNR

	2017	**1998**	**2016**	**1984**
	Hydrogenated Lard Glyceride			
Totals*	**1**	**6**	**NR**	**≤1[#]**
Duration of Use				
Leave-On	*1*	*6*	*NR*	*DNR*
Rinse-Off	*NR*	*NR*	*NR*	*DNR*
Diluted for (Bath) Use	*NR*	*NR*	*NR*	*DNR*
Exposure Type				
Eye Area	1	4	NR	DNR
Incidental Ingestion	NR	1	NR	DNR
Incidental Inhalation-Spray	NR	NR	NR	DNR
Incidental Inhalation-Powder	NR	NR	NR	DNR
Dermal Contact	1	5	NR	DNR
Deodorant (underarm)	NR	NR	NR	DNR
Hair - Non-Coloring	NR	NR	NR	DNR
Hair-Coloring	NR	NR	NR	DNR
Nail	NR	NR	NR	DNR
Mucous Membrane	NR	1	NR	DNR
Baby Products	NR	NR	NR	DNR

*Because each ingredient may be used in cosmetics with multiple exposure types, the sum of all exposure types may not equal the sum of total uses.
**at the time of the 2001 safety assessment, concentration of use data were not reported by the FDA; 1984 data were presented.
[#] A summary of the concentration of use data was reported.
[a] It is possible these products are sprays, but it is not specified whether the reported uses are sprays.
[b] Not specified whether a spray or a powder, but it is possible the use can be as a spray or a powder, therefore the information is captured in both categories.
NR – no reported use
DNR – Details of use not reported.

REFERENCES

1. Andersen, FA. Final report on the safety assessment of Lard Glyceride, Hydrogenated Lard Glyceride, Lard Glycerides, Hydrogenated Lard Glycerides, Lard, and Hydrogenated Lard. *International Journal of Toxicology*. 2001;20(Suppl. 2):57-64.

2. Nikitakis, J and Breslawec HP. International Cosmetic Ingredient Dictionary and Handbook. 15 *ed*. Washington, DC: Personal Care Products Council, 2014.

3. European Chemicals Agency (ECHA). Substance information: Lard. https://echa.europa.eu/substance-information/-/substanceinfo/100.057.346. Last Updated 5-3-2016.

4. Food and Drug Administration (FDA). Dioxin analysis results/exposure estimates. http://www.fda.gov/Food/FoodborneIllnessContaminants/ChemicalContaminants/ucm077444.htm. Last Updated 12-9-2015. Date Accessed 2-8-2017.

5. Food and Drug Administration (FDA). PCDD/PCDF exposure estimates from non-TDS foods analyzed in 2001-2003. http://www.fda.gov/food/foodborneillnesscontaminants/chemicalcontaminants/ucm077478.htm. Last Updated 12-9-2015.

6. Food and Drug Administration (FDA). Frequency of use of cosmetic ingredients; *FDA Database*. Washington, DC, FDA. 2017.

7. Personal Care Products Council. 2-17-2016. Concentration of Use by FDA Product Category: Lard Ingredients.

8. European Commission. International Market, Industry, Entrepeneurship and SMEs; CosIng. http://ec.europa.eu/growth/tools-databases/cosing/. Last Updated 2016. Date Accessed 8-22-2016.

9. Select Committee on GRAS Substances (SCOGS). Select Committee on GRAS substances (SCOGS) opinion: Lard (packaging); lard oil (packaging). http://www.fda.gov/forconsumers/consumerupdates/ucm260450.htm. Last Updated 10-15-2015. Date Accessed 8-18-2016.

10. U.S. Environmental Protection Agency (EPA). List of Partially Exempt Chemicals under Chemical Data Reporting. https://www.epa.gov/chemical-data-reporting/list-partially-exempt-chemicals-under-chemical-data-reporting. Last Updated 10-9-2015. Date Accessed 8-23-2016.

Final Report on the Safety Assessment of Lard Glyceride, Hydrogenated Lard Glyceride, Lard Glycerides, Hydrogenated Lard Glycerides, Lard, and Hydrogenated Lard[1]

Lard obtained from the rendering of fatty porcine tissue is used in cosmetic products, as are several of its derivatives. These derivatives include Lard Glycerides (mono-, di-, and triglycerides derived from Lard), Lard Glyceride (the monoglycerides only), Hydrogenated Lard Glycerides, Hydrogenated Lard Glyceride, and Hydrogenated Lard. The latter three are produced by controlled hydrogenation of the described precursor. These ingredients function as skin-conditioning agents and, with the exception of Lard, as viscosity-increasing agents in several cosmetic products. No information was available regarding the fate during processing of impurities such as pesticides or heavy metals that may be found in animal tissue. Lard itself is established by the Food and Drug Administration (FDA) as a GRAS (generally recognized as safe) substance. Animal studies report adverse effects expected with the feeding of high fat diets, but other animal toxicity data were not available. Lard was not mutagenic in transgenic mice. Cell proliferation assays showed more proliferation in mice fed Lard compared to those fed plant-source fats, but another study showed no difference. Cocarcinogenic effects were observed when high-fat diets containing Lard were fed, with known carcinogens, to mice, rats, and hamsters. Consistent with the FDA GRAS determination, it was concluded that these ingredients may be used safely in cosmetic formulations. However, it was considered important to limit the presence of heavy metals and/or polychlorinated biphenyl (PCB) or other pesticide contamination. Accordingly, limits were established as follows: lead, not more than 0.1 ppm; arsenic (as As), $\leq$3 ppm; mercury (as Hg), $\leq$1 ppm; and total PCB/pesticide contamination, not more than 40 ppm, with not more than 10 ppm for any specific residue.

INTRODUCTION

This review summarizes data concerning the safety of Lard Glyceride (CAS No. 61789-10-4), Hydrogenated Lard Glyceride (CAS No. 8040-05-9), Lard Glycerides, Hydrogenated Lard Glycerides, Lard (CAS No. 61789-99-9), and Hydrogenated Lard (CAS No. 73138-67-7), as cosmetic ingredients. Most of the studies were conducted using Lard as the test agent.

CHEMISTRY

Definition and Structure

Lard Glyceride. Lard Glyceride (CAS No. 61789-10-4) is the monoglyceride derived from Lard (q.v.) (Wenninger and McEwen 1997).

Hydrogenated Lard Glyceride. Hydrogenated Lard Glyceride (CAS No. 8040-05-9) is the end product of controlled hydrogenation of Lard Glyceride (q.v.) (Wenninger and McEwen 1997).

Lard Glycerides. Lard Glycerides is a mixture of mono-, di- and triglycerides derived from Lard (q.v.) (Wenninger and McEwen 1997).

Hydrogenated Lard Glycerides. Hydrogenated Lard Glycerides is the end product of controlled hydrogenation of Lard Glycerides (q.v.) (Wenninger and McEwen 1997).

Lard. Lard (CAS No. 61789-99-9) is the purified fat obtained from the abdomen of the hog (Wenninger and McEwen 1997).

Hydrogenated Lard

Hydrogenated Lard (CAS No. 73138-67-7) is the end product of controlled hydrogenation of Lard (q.v.) (Wenninger and McEwen 1997).

Physical and Chemical Properties

Lard. Lard is described as a soft, white unctuous substance with a bland taste and a characteristic odor. Some properties of Lard are cited in Table 1.

About 200 fatty acids are found in Lard (Swern 1979). The chief constituents are stearic, palmitic, and oleic acids (Lewis 1993).

Method of Manufacture

Lard. Lard is obtained by dry or wet rendering of fresh fatty porcine tissues (cuttings and trimmings) shortly after slaughter.

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1091-5818/01 $12.00 + .00

TABLE 1
Properties of Lard

Property	Description	Reference
Density	0.917	Budavari 1989
Melting point	36°–42°C	Budavari 1989; Lewis 1993
Solubility	Insoluble in water, slightly soluble in alcohol, soluble in benzene, chloroform, ether, carbon disulfide, petroleum ether	Budavari 1989; Lewis 1993
Saponification no.	195–203	Budavari 1989

Lard produced by wet-rendering processes is known as prime steam lard. Rendered Lard may be bleached, or bleached and deodorized (National Academy of Sciences 1996; Swern 1979).

No information was found regarding potential impurities that may be found in animal tissue.

USE

Cosmetic

The chemical class and cosmetic function of Lard and lard-derived ingredients are cited in Table 2.

In 1998, Lard Glyceride was reportedly used in two formulations, Hydrogenated Lard Glyceride was used in six formulations, and Lard was used in three formulations (FDA 1998) (Table 3). Concentrations of use are no longer reported to the FDA (FDA 1992). However, data from 1984 indicated use of Lard Glyceride at concentrations of ≤10%, Hydrogenated Lard Glyceride at ≤1%, and Lard at ≤10% (FDA 1984).

International

Lard. Lard will be labeled as Adeps Suillus in the European Union when regulations for ingredient labeling under the 6th Amendment to the *EC Cosmetics Directive* go into effect (Wenninger and McEwen 1997).

Noncosmetic

Lard. Lard is a generally recognized as safe (GRAS) food ingredient (Informatics Inc. 1974).

GENERAL BIOLOGY

Serum Lipoproteins

Lard. In a 28-day study, hamsters fed 10% Lard and 0.12% dietary cholesterol had significantly increased ($p < .0001$) serum very-low-density lipoprotein (VLDL) and low-density lipoprotein (LDL) cholesterol and serum VLDL-triacylglycerol concentrations compared to both basal diet–fed controls and hamsters fed diets containing Lard but not additional cholesterol. No effect was noted on hepatic cholesterol ester concentrations (Sessions and Salter 1994).

Groups of miniature swine were fed diets containing 9% Lard (or sunflower or olive oil) for either 12 or 50 weeks. A control group was not included. Serum total cholesterol concentrations were significantly greater in the Lard-fed group at 12 weeks but not at 50 weeks. (A significant increase was observed in the sunflower-fed group at 50 weeks.) Lard-fed swine had a significant increase in LDL and significant decrease in high-density lipoprotein (HDL) between 12 and 50 weeks. The investigators considered that the atherogenic index was lower in the Lard-fed group in the long term compared to the polyunsaturated fat group (Seiquer et al. 1994).

Imaki et al. (1989) reported a significant increase ($p < .05$) in total cholesterol, a significant decrease ($p < .01$) in serum HDL-cholesterol, and significant increases ($p < .05$) in serum HDL-cholesterol and serum HDL-phospholipid fractions in four young men following 7 days of ingesting 30 g Lard/day.

Hypertension

Kaufman, Peterson, and Smith (1994) reported development of hypertension in male Sprague-Dawley rats following 10 weeks

TABLE 2
Cosmetic function of Lard-derived ingredients (Wenninger and McEwen 1997)

Ingredient	Chemical class	Function in cosmetics
Lard Glyceride	Glyceryl esters and derivatives	Emulsion stabilizer Skin-conditioning agent—emollient Viscosity-increasing agent—nonaqueous
Hydrogenated Lard Glyceride	Glyceryl esters and derivatives	Skin-conditioning agent—emollient Viscosity-increasing agent—nonaqueous Surfactant—emulsifying agent
Hydrogenated Lard Glycerides	Glyceryl esters and derivatives	Skin-conditioning agent—emollient Viscosity-increasing agent—nonaqueous
Lard	Fats and oils	Skin-conditioning agent—occlusive
Hydrogenated Lard	Fats and oils	Skin-conditioning agent—occlusive Viscosity-increasing agent—nonaqueous

TABLE 3
Frequency of use (FDA 1998)

Product category	No. of formulations in category	No. containing ingredient
Lard Glyceride		
Body and hand skin care (excluding shaving)	796	1
Moisturizing skin care	769	1
1998 Total for Lard Glyceride		2
Hydrogenated Lard Glyceride		
Eyebrow pencil	91	2
Eyeliner	514	1
Other eye makeup preparations	120	1
Lipstick	790	1
Other makeup preparations	135	1
1998 Total for Hydrogenated Lard Glyceride		6
Lard		
Hair straighteners	63	1
Other skin care preparations	692	2
1998 Total for Lard		**3**

of feeding diets having large amounts of either Lard or corn oil. The Lard diet provided 66.5% of energy as fat. Rats fed Lard became obese (weighing significantly more than low-fat fed controls) and their fasting insulin concentrations were significantly increased ($p < .05$) compared to rats of both the corn oil and control groups.

ANIMAL TOXICOLOGY

Oral Toxicity

Short-Term

Lard. Thomasson (1955) conducted a 6-week feeding study in which Wistar rats were fed Lard at concentrations such that 10% to 73% of the total dietary calories were derived from Lard. The food-efficiency of Lard was comparable to summer butterfat, and no adverse effect on mortality was observed.

Chronic

Lard. Groups of male C57BL mice were fed diets containing 25% Lard from weaning though life, age 6 months through life, age 12 months through life, or for 5-month periods beginning at age 1, 7, or 12 months. A control group was fed laboratory chow. Mice were killed at various times and examined for skeletal changes. Body weight gains were greater in Lard fed mice compared to control mice; gains were the least striking in mice fed Lard for 5 months or from age 12 months onwards. The data were not analyzed for statistical significance, but the investigators concluded that lifetime feeding of a high-fat diet shortened the life span, with the most dramatic effects noted in mice that had been fed fat since weaning. The effect on survival of a 5-month high-fat diet depended on the treatment period; "given during the period of growth, the high fat ration did not alter the life span; in young adults it had a beneficial effect, while given to old animals it exerted a slightly injurious action on the life span" (Silberberg and Silberberg 1954, 1955).

Hydrogenated Lard. Weanling rats were fed 50% Hydrogenated Lard (or other saturated fats) and a control group was fed 50% Lard. Rats were killed at 8-week intervals for necropsy. Samples of the perigastric, perirenal, periadrenal, perigonadal, and subcutaneous fat were removed, fixed, and stained for microscopic examination. No lesions were found at necropsy; in a few rats fed the saturated fat, the adipose tissue had a mottled appearance. A foreign body–type granulomatous reaction was noted in the adipose tissue at microscopic examination of rats fed Hydrogenated Lard or the other saturated fats: the lesion consisted of multinucleated cells with nuclei arranged about the periphery or in clumps. Often, the fat material appeared partially dissolved, leaving radially oriented slitlike spaces that suggested fatty acid crystals. No acute inflammatory reaction, hemorrhage, or birefringent material were associated with the reaction. The reaction occurred within 16 weeks of feeding the Hydrogenated Lard diet and within 8 weeks of feeding the other saturated fats; it was not observed in control rats (Herting and Crain 1958).

REPRODUCTIVE AND DEVELOPMENTAL TOXICITY

Oral

Lard. The GRAS report on Lard (Informatics Inc. 1974) cited a 1940 study that investigated the influence of a high-Lard diet on the reproductive capacity of female rats. Five 2-month-old rats from a single litter were used. Two of the rats (rats 1 and 2) were fed bread, milk, and water ad libitum. The experimental rats (rats 3–5) were given 4 to 5 g of Lard daily in addition to the control diet. An adult female rat (rat 6) was also fed the high-fat diet and a male rat was alternately fed the control and high-fat diet (each for a 1-week period).

Beginning a few days into the study, test rats 3 to 5 reduced their feed intake, became emaciated, and crouched in a corner of the cage. They had swollen snouts, reddened and edematous eyelids with thinner eyelashes, rumpled fur, and extensively reddened and swollen external genitalia. The male had similar lesions when fed the high-fat diet and had conspicuous swelling of the scrotum and testicles and priapism. These alterations disappeared after 9 to 10 days, reoccurred periodically, and occurred in a "progressively more attenuated form in female rats." Reproductive activity was not effected as one test rat (3) gave birth at almost the same time as one of the control females. Dams and offspring were killed and necropsied shortly after birth.

Growth of test rats was "somewhat stunted." Livers of rats fed high-fat diets were enlarged, were uniformly yellow, diminished in consistency and the cut surface was moist, shining, and oily. A "more or less accentuated state of adiposis" was noted upon microscopic examination of hepatic cells.

No differences were noted in number of offspring between control and test rats. Offspring of test rats appeared smaller than control offspring and were weakened, sluggish, poorly nourished, and cyanotic with dry and wrinkled skin. Offspring from two litters whose dams had been fed the high-fat diet for 2 months were "in better condition" than those litters from dams that had been fed the high-fat diet for >3 months prior to delivery. Livers of pups from test group were small, yellow, and soft. "Adiposis" was noted upon microscopic examination. Hepatic cells were filled with granules and droplets of fat that were abundant at the periphery of the cell and formed vacuoles in some cells. The nuclei frequently were displaced to the periphery and the cytoplasm appeared reduced in amount.

In a follow-up study, the same investigators fed three test rats with 4 to 5 g of Lard/daily (in addition to laboratory feed) throughout pregnancy and lactation (2.5–3.5 months). The results were similar to those of the study reported above. In addition, pups of the test group had stunted growth, dry and wrinkled skin, cyanosis, and sluggish movements. Test group pups began to grow fine hair after 13 to 14 days and opened their eyes after 18 to 21 days, whereas the time frame for control rat pups was 10 to 11 days and 13 to 14 days, respectively. Some test pups died a few days after birth and the survivors (killed after 25–27 days of nursing) had "substantially" lower body weights compared to controls. The average weight was 23.3 g for test group pups and 34.6 g for control pups (Informatics Inc. 1974).

A four-generation study was conducted to determine nutritional requirements for reproduction and lactation. Wistar and Evans-Long rats were fed diets containing 10% Lard, 5% Lard (plus 10% Crisco, a commercial shortening), 2% Lard (plus 8% Crisco), and a combination of Crisco plus corn oil. A control group was fed unsupplemented diets. Growth was comparable among treated and control rats. Dams fed the experimental diets lost weight during lactation, and weaned fewer young than those fed the control diet. (The data for all supplemented feed groups were combined.) Dams regained weight when separated from their young. Supplementary Lard was supplied ad libitum and as much as 8 g/day was eaten; no improvement in lactation was noted and dams fed the supplement killed their young during lactation. The investigators considered that the consumption of such large quantities of Lard interfered with the ingestion of other food constituents (Vinson and Cerecedo 1944).

GENOTOXICITY

Lard. Noting that epidemiological studies implicated dietary fat as a contributor to the development of colonic cancer, Zhang et al. (1996) tested the mutagenicity of high-fat diets for intestinal epithelium at two loci (*Dlb-1* host gene of the colon and *lac*I transgene). Four transgenic mice (two of each sex; the F_1 generation of a cross of hemizygous *lac*I C57BLl/6 and SWR mice) were fed diets containing 31% Lard for 5 and 9 weeks. (Other fats were also tested; half of the calories were from fat in all experimental diets.) No significant increase in mutation frequencies was observed when compared to basal diet fed controls. The investigators concluded that uncooked fats were not mutagenic and were not initiators of carcinogenesis in the gut epithelium.

CARCINOGENICITY

Oral

Lard. Clayson et al. (1992) introduced an assay that measured increased cellular proliferation as an indicator of carcinogenesis. Greater [^{3}H]-thymidine incorporation in cells of the mammary gland and colorectal region was noted in young adult Swiss Webster mice that had been fed a modified diet containing 15% lard (for 30 days) compared to fat components rich in vegetable oils. Similarly, Lok et al. (1992) reported significantly greater ($p < .01$) [^{3}H]-thymidine labeling of mammary gland ductal cells and an increased number of labeled cells/crypt in the crypts of the colorectum in mice fed diets in which the 15% fat component was comprised solely of Lard or fish oils compared to when the fat source was either soybean oil or safflower oil, or a mixture of the vegetable oils and Lard. The addition of antioxidants to the Lard diets reduced the cell-labeling index, and in some instances, the index was similar to that of the soybean oil diet.

Jacobs (1993) studied whether short-term high-fat consumption could result in pancreatic cell and colonic cell hyperplasia. Groups of eight rats were fed diets containing either 27% Lard plus 3% corn oil or 30% corn oil for 4 weeks. Control rats were fed a 5% corn oil diet. Both high fat diets produced significant decreases ($p < .05$) in proximal jejunal mass. Pancreatic lipase activity increased (Lard group compared to control, $p < .05$) and amylase activity decreased (Lard group compared to control, $p < .05$) on the high-fat diets, but neither high-fat diet produced changes in cell growth or the incorporation of [^{3}H]-thymidine into the DNA of stomach, pancreas, or colon. The investigators considered that the data did not support the conclusion that dietary fat promoted pancreatic and colonic carcinogenesis by producing cell hyperplasia.

Pell, Brown, and Johnson (1994) investigated the role of dietary fats in the hyperproliferation of intestinal epithelium as a precursor to neoplasia. Groups of 10 male Wistar rats were fed fiber-free diets containing 80 g/kg of either Lard, corn oil, or fish oil. After 14 days, the crypt cell production rates (CCPR) at two sites in the small intestine, one site in the cecum, and two sites in the colon were determined by the metaphase arrest technique. A negative-control group was not included. Fish oil produced significantly less ($p < .05$) CCPR in the jejunum, ileum, and proximal colon. The CCPR were similar for the Lard and corn oil groups. Corn oil produced significantly lower CCPR in the cecum; the rates for the Lard-fed and fish oil–fed groups were similar. The Lard and fish oil groups had significantly smaller CCPRs in the distal colon compared to the corn oil group. Fish oil, which contains n-3 fatty acids, was considered to support a "relatively low rate of crypt cell proliferation in some regions of the alimentary tract."

COCARCINOGENICITY

This section describes the numerous studies that investigated the correlation between dietary fat and carcinogenicity. Reviews have been published by Reddy (1992) and Rogers (1983).

Mammary Cancer

Lard. Groups of 20 female Sprague-Dawley rats received an intragastric dose of the carcinogen 7,12-dimethylbenz[a]-anthracene (DMBA) and were then fed for 4 months one of seven diets: 20% Lard (diet 1), 3% sunflower seed oil supplemented with 17% of either tallow or coconut oil (diets 2 and 3), 20% tallow (diet 4), 20% coconut oil (diet 5), 3% sunflower seed oil (diet 6), or 20% sunflower seed oil (diet 7). Rats were palpated weekly for tumors and necropsied at the end of the study. Diets 1, 2, 3, and 6 had similar linoleic acid contents (2.1%–2.5% weight), diets 4 and 5 contained ~0.4 weight % linoleic acid, and diet 7 had a 13.8 weight % linoleic acid content. The incidence of mammary adenocarcinomas was similar for groups 1, 2, 3, and 7, and greater than that of groups 4, 5, and 6. The investigators concluded that a combination of high fat (20%) plus a moderate concentration of linoleic acid was necessary to enhance DMBA-induced carcinogenesis (Hopkins and Carroll 1979).

Similarly, Rogers and Wetsel (1981) reported a greater incidence of DMBA-induced mammary tumors in rats fed 20% Lard or corn oil versus 5% Lard or corn oil. Earlier sexual maturation (indicated by age at vaginal opening) was significantly earlier in rats fed 20% corn oil compared to 5% corn oil or either concentration of Lard. Serum prolactin concentrations after 3 or 10 weeks of dietary treatment were similar among groups. An untreated group was not included.

Cohen and Chan (1982) reported that the incidence of *N*-methylnitrosourea (MNU)-induced mammary tumors was significantly greater ($p < .0001$) in rats that had been fed 20% Lard versus those fed 4% Lard. The addition of cholesterol to either dietary group did not alter the findings. The triglyceride fraction (as opposed to the nonsaponifiable fraction) was considered the determining factor in promotion of mammary tumors.

Female Fischer rats (27 rats) that were fed Lard diet (32% by weight) followed by MNU administration had a mammary tumor incidence of 63%. The rate was not significantly different from the 33% incidence noted in the 23 rats that had been fed 5% corn oil (significance measured by Student-Neuman-Keuls analysis). A ranking of the dietary groups in order of the appearance of the first palpable tumor: high corn oil > high lard > high beef tallow > low corn oil > high coconut oil, correlated with the total oleate and linoleate content of the diets (Chan, Ferguson, and Dao 1983).

Chan, Didato, and Cohen (1975) investigated whether "the enhanced development of DMBA-induced mammary adenocarcinomas in rats fed a high fat diet might be mediated by prolactin via the hypothalamic-pituitary system rather than by a direct effect of the fat on the mammary gland itself." Groups of 20 female Sprague-Dawley rats received either 20% or 5% semisynthetic Lard. Blood samples taken weekly from the tail vein and daily vaginal smears were collected for 3 weeks at 2 and 5 months. The serum prolactin titer was increased significantly ($p < .05$) during the proestrus-estrus period in rats of the high-fat group than in those of the low-fat group at both 2 and 5 months. No difference in prolactin concentrations was noted between the two groups during the metestrus-diestrus phase.

Bieber (1986) investigated whether the enhanced mammary gland-tumor growth observed with lard was due to an estrogen-like activity. Groups of 15 prepubertal female mice were fed 5% or 20% lard for 7 days. Three lard samples were tested separately, one contained 35 ppb dieldrin, and the two commercial preparations contained 100 ppm each of butylated hydroxyanisole and butylated hydroxytoluene. Estrogen-like activity was measured by changes in uterine weight or in the ratio of uterine weight to body weight. No measurable effect was observed when compared to diethylstilboestrol, the positive control.

Sylvester et al. (1986) conducted a study to determine whether the effect of dietary fat on mammary tumorigenesis occurred prior to and during the initiation phase of carcinogenesis. Groups of 21-day-old female Sprague-Dawley rats received either 5% corn oil (normal fat control), or 20% Lard, corn oil, palm oil, or beef tallow. At 52 days of age, rats were given a oral (PO) dose of DMBA and 1 week later all rats were fed a 5% corn oil diet that was continued for the duration of the study. Rats fed 20% lard during the treatment period had a significant increase ($p < .05$) in number of mammary gland tumors 19 weeks after DMBA administration when compared to all other dietary groups. Because mammary tumor incidence was greater in the beef tallow group (nonsignificant), the investigators tested whether animal-derived fats altered sexual maturation and endocrine function. Average day of vaginal opening was earlier for rats of all the 20% fat groups compared to the control. Further, no differences were noted in basal or surge concentrations of prolactin, luteinizing hormone, or estradiol, or in diestrus uterine weight. Thus, the enhanced mammary tumorigenesis observed with animal-derived fat did not "result from endogenous or exogenous endocrine stimulation."

Colon Cancer

Lard. Reddy et al. (1976) reported that the incidence of 1,2-dimethylhydrazine (DMH)-induced tumors of the ear canal, small intestine, and colon was greater in rats fed 20% Lard or corn oil compared to rats fed 5% Lard or corn oil. The incidence of colonic tumors was slightly greater in rats of the 5% corn oil group compared to rats of the 5% Lard group. The data were not analyzed for statistical significance.

Bansal, Rhoads, and Bansal (1978) reported that DMH-induced tumors appeared earlier and the total number of gastrointestinal (GI) tumors was significantly greater ($p < .05$) in rats fed either 5% Lard (supplemented with carbohydrate) or 30% Lard compared to rats fed standard diet. The neoplasms metastasized more frequently and survival time was shortened in rats fed Lard. At 110 days of age, rats of the Lard groups

had increased cholesterol and serum triglyceride concentrations compared to controls, but by day 190 the concentrations in the 5% Lard group were comparable to controls. Serum immunoglobulin G (IgG) concentrations were significantly decreased in the Lard fed rats, but were similar to concentrations found in rats fed 30% corn oil or a semisynthetic diet of egg albumin and soybean oil (separate experiment). Compared to controls, rats of the 30% Lard group had increased lymphocyte counts during the early period of DMH carcinogenesis and a decline with the appearance of multiple GI neoplasms.

Reddy and Maruyama (1986) investigated whether Lard enhancement of azoxymethane (AOM)-induced colon carcinogenesis was at the initiation or promotion stage. Groups of male weanling F344 rats (treated with AOM for 2 weeks) were fed 5%, 12.6%, or 23.5% Lard and then transferred to a 5% Lard diet for an additional 34 weeks. A positive control (testing promotion) initially received 5% Lard and was transferred to a 23.5% Lard diet after carcinogen administration. Six randomly selected rats were examined by endoscopy at 20, 25, and 30 weeks after the last AOM injection. A dose-dependent increase in the incidence of colonic adenocarcinomas was observed and was significant ($p < .05$) for the 23.5% (then switched to 5%) dose group. Multiple colonic neoplasms were significantly greater in incidence in rats of the 13.6% Lard-fed group (then switched to 5%) and 23.5% Lard-fed group (then switched to 5%) compared to the 5% Lard-fed group. The greatest incidence was in rats of the positive-control group. No consistent differences were found in the incidence of small intestinal and ear duct tumors among rats fed Lard during either initiation or promotion.

Groups of 30 male 4-week-old Wistar rats were fed 15% (low), 27.5% (medium), or 40% (high) Lard in combination with small, medium, or large amounts of fiber. After 4 weeks on these diets, rats were given five weekly intrarectal instillations of *N*-methyl-*N'*-nitro-*N*-nitrosoguanidine. The greatest incidence and the greatest total number of colonic carcinomas were observed in the medium-fat/medium-fiber diet. The greatest number and a relatively greater polyp incidence were observed in rats of the high-fat/low-fiber diet. Fat concentration had no effect on tumor incidence when the fiber content was large (Sinkeldam et al. 1990).

Kristiansen, Thorup, and Meyer (1995) reported that adding 20% lard to a standard diet did not cause an increase in aberrant crypt foci (ACF) in DMH-initiated male Wistar rats. However, addition of 20% fiber to the Lard diet produced a significant reduction ($p \leq .05$) in the total number of ACF and the number of small and medium ACFs observed.

Pancreatic Cancer

Lard. Woutersen and van Garderen-Hoetmer (1988) reported a significant increase in azaserine and *N*-nitrosobis(2-oxopropyl)amine-induced preneoplastic foci in the exocrine pancreas of male rats and hamsters that were fed 20% lard (feeding began 12 days postcarcinogen administration) compared to the number of foci in the pancreas of animals fed 5% lard.

Groups of 40 male weanling SPF albino Wistar rats were fed either 20% Lard (high fat), 4.52% Lard/0.48% safflower oil plus 0.6% linoleic acid (low fat), or 1.4% Lard/3.6% corn oil diet with 2.0% linoleic acid (supplemented low fat). On days 19 and 26, rats received injections of azaserine. A control group treated with azaserine but fed basal diet was maintained. Necropsy was done on days 482 to 485 after azaserine administration. The number of azaserine-induced basophilic foci in the rat pancreas was significantly ($p < .05$) increased in rats of the supplemented low-fat group compared to the low-fat group. The supplemented low-fat group had significantly less atypical acinar cell nodules >1 mm compared to the 5% fat group ($p < .05$) and the 20% lard group ($p < .01$). The number of adenomas was significantly greater in the high-fat group compared to the low-fat ($p < .01$) and supplemented low-fat ($p < .001$) groups, and the incidence was significantly less in the supplemented group compared to the low-fat group ($p < .05$). The investigators concluded that the promoting effect of a high-Lard diet on azaserine-treated rats was not due to linoleic acid (Appel, nan Garderen-Hoetmer, and Woutersen 1990).

Hietanen et al. (1990) reported that *N*-nitrosodimethylamine (NDMA)-induced hemangiosarcomas in male rats increased from 43% to 67% when dietary Lard was increased from 2% to 25%; the increase in incidence was not significant. The increase in dietary fat also enhanced ethane exhalation (a measure of lipid peroxidation), but the increase was more pronounced in the sunflower seed oil group. The increase in hemangiosarcoma was significant ($p < .05$) in the sunflower seed group (2% vs. 25% fat).

SUMMARY

Lard Glyceride, Hydrogenated Lard Glyceride, Lard Glycerides, Hydrogenated Lard Glycerides, Lard, and Hydrogenated Lard are ingredients derived from the purified fat obtained from the abdomen of the hog. No information was available regarding potential impurities such as pesticides or heavy metals that may be found in animal tissue.

All seven ingredients function as skin-conditioning agents. All except Lard also function as viscosity-increasing agents. As of January 1998, Lard Glyceride was used in two formulations, Hydrogenated Lard Glyceride was used in six formulations, and Lard was used in three formulations.

Lard is a GRAS substance. In oral dose toxicity and reproductive/developmental studies, adverse effects were reported consistent with the feeding high-fat Lard diets to mice and rats.

Lard did not increase the mutation frequency in the intestinal epithelium of orally dosed transgenic mice. Two studies that examined cell proliferation as an indicator of carcinogenesis reported greater [^{3}H]-thymidine incorporation in cells of the mammary gland and colorectum region in mice fed 15% Lard (compared to mice fed plant-source fats). In another study, no increase in cell hyperplasia and thymidine incorporation were found in the stomach, pancreas, or colon of rats fed 27% Lard (compared to controls fed 5% corn oil). Rats fed fiber-free diets

containing 80 g/kg of Lard had crypt cell production rates (in the small intestine) comparable to that of rats fed corn oil. Lard-dosed rats had significantly smaller rates in the distal colon, whereas corn oil-dosed rats had significantly smaller rates in the cecum. Cocarcinogenic effects were observed when high-fat diets were fed with known carcinogens to mice, rats, and hamsters.

DISCUSSION

The Cosmetic Ingredient Review (CIR) Expert Panel was of the opinion that Lard Glyceride, Hydrogenated Lard Glyceride, Lard Glycerides, Hydrogenated Lard Glycerides, Lard, and Hydrogenated Lard can be used safely in cosmetic formulations. However, the Panel recognized the need to limit the presence of heavy metals and/or polychlorinated biphenyl (PCB) or other pesticide contamination.

Lead is limited to not more than 0.1 mg/kg (0.1 ppm). This value is adopted from the *Food Chemical Codex (FCC)* limit for unhydrogenated Lard (National Academy of Sciences 1996). Because the *FCC* did not address the presence of other heavy metals in Lard, the Panel adopted the limits found in 21 CFR 73&74. Those limits are $\leq$3 ppm arsenic (as As) and $\leq$1 ppm mercury (as Hg) (CIR 1998).

The Panel limited the total PCB/pesticide contamination to not more than 40 ppm with not more than 10 ppm for any specific residue. These limits are modeled after the USP standards for lanolin (Committee of Revision of the United States Pharmacopeial Convention 1995).

CONCLUSION

Based on the available data, the CIR Panel concludes that Lard Glyceride, Hydrogenated Lard Glyceride, Lard Glycerides, Hydrogenated Lard Glycerides, Lard, and Hydrogenated Lard are safe as used in cosmetic products, provided that established limitations imposed on heavy metal and pesticide concentrations are not exceeded.

REFERENCES

Appel, M. J., A. nan Garderen-Hoetmer, and R. A. Woutersen. 1990. Azaserine-induced pancreatic carcinogenesis in rats: Promotion by a diet rich in saturated fat and inhibition by a standard laboratory chow. *Cancer Lett.* 55:239–248.

Bansal, B. R., J. E. Rhoads, and S. C. Bansal. 1978. Effects of diet on colon carcinogenesis and the immune system in rats treated with 1,2-dimethylhydrazine. *Cancer Res.* 38:3293–3303.

Bieber, M. A. 1986. Lack of oestrogen-like activity in commercially refined vegetable oils. *Food Chem. Toxicol.* 24:251–253.

Budavari, S., ed. 1989. *The Merck index: An encyclopedia of chemicals, drugs and biologicals*, 10th ed., 847. Rahway, NJ: Merck and Co.

Chan, P. C., F. Didato, and L. A. Cohen. 1975. High dietary fat, elevation of rat serum prolactin and mammary cancer. *Proc. Soc. Exp. Biol. Med.* 149:133–135.

Chan, P. C., K. A. Ferguson, and T. L. Dao, 1983. Effects of different dietary fats on mammary carcinogenesis. *Cancer Res.* 43:1079–1083.

Clayson, D. B., E. Lok, F. W. Scott, R. Mongeau, et al. 1992. Calories, fat, fibers and cellular proliferation in Swiss Webster mice. *Adv. Exp. Med. Biol.* 322:83–93.

Cohen, L. A., and P. C. Chan. 1982. Dietary cholesterol and experimental mammary cancer development. *Nutr. Cancer* 4:99–106.

Committee of Revision of the United States Pharmacopeial Convention. 1995. *The United States Pharmacopeia*, 23rd ed., 817–872. Rockville MD: United States Pharmacopeial Convention, Inc.

Cosmetic Ingredient Review (CIR). 1998. *Final report on the safety assessment of Acid Violet 43*. Washington: CIR. (19 pages.)[2]

Food and Drug Administration (FDA). 1984. Cosmetic product formulation and frequency of use data. *FDA database*. Washington, DC: FDA.

FDA. 1992. Modification in Voluntary Filing of Cosmetic Product Ingredient and Cosmetic Raw Composition Statements. Final rule. *Fed. Register* 57:3128–3130.

FDA. 1998. Cosmetic product formulation data. *FDA database*. Washington, DC: FDA.

Herting, D., and R. C. Crain. 1958. Foreign-body type reaction in fat cells. *Proc. Soc. Exp. Biol. Med.* 98:347–348.

Hietanen, E., H. Bartsch, J. C. Bereziat, et al. 1990. Quantity and saturation degree of dietary fats as modulators of oxidative stress and chemically-induced liver tumours in rats. *Int. J. Cancer* 46:640–647.

Hopkins, G. J., and K. K. Carrol. 1979. Relationship between amount and type of dietary fat in promotion of mammary carcinogenesis induced by 7,12-dimethylbenz[a]anthracene. *J. Natl. Cancer Inst.* 62:1009–1012.

Imaki, M., T. Miyoshi, S. Tanada, et al. 1989. Effect of lard and corn oil intake on serum lipids in young men. *Acta Biol. Hung.* 40:271–282.

Informatics, Inc. 1974. Scientific literature reviews on generally recognized as safe (GRAS) food ingredients: Lard and Lard Oil. Available from the National Technical Information Service (NTIS). NTIS report no.: PB-234–891.

Jacobs, L. R. 1993. Effect of short-term dietary fat on cell growth in rat gastrointestinal mucosa and pancreas. *Am. J. Clin. Nutr.* 37:361–367.

Kaufman, L. N., M. M. Peterson, and S. M. Smith. 1994. Hypertensive effect of polyunsaturated dietary fat. *Metabolism* 43:1–3.

Kristiansen, E., I. Thorup, and O. Meyer. 1995. Influence of different diets on development of DMH-induced aberrant crypt foci and colon tumor incidence in Wistar rats. *Nutr. Cancer* 23:151–159.

Lewis, R. J., Sr. 1993. *Hawley's condensed chemical dictionary*, 12th ed., 682. New York: Van Nostrand Reinhold.

Lok, E., W. M. Ratnayake, F. W. Scott, R. Mongeau, et al. 1992. Effect of varying the type of fat in a semi-purified AIN-76A diet on cellular proliferation in the mammary gland and intestinal crypts in female Swiss Webster mice. *Carcinogenesis* 13:1735–1741.

National Academy of Sciences (NAS). 1996. *Food chemicals codex*, 4th ed., 217. Washington, DC: National Academy Press.

Pell, J. D., J. C. Brown, and I. T. Johnson. 1994. Polyunsaturated fatty acids of the n-3 series influence intestinal crypt cell production in rats. *Carcinogenesis* 15:1115–1119.

Reddy, B. S. 1992. Dietary fat and colon cancer: Animal model studies. *Lipids* 27:807–813.

Reddy, B. S., and H. Mauyama. 1986. Effect of different levels of dietary corn and lard during the initiation phase of colon carcinogenesis in F344 rats. *J. Natl. Cancer Inst.* 77:815–822.

Reddy, B. S., T. Narisawa, D. Vukusich, J. H. Weisburger, and E. L. Wynder. 1976. Effect of quality and quantity of dietary fat and dimethylhydrazine in colon carcinogenesis in rats. *Proc. Soc. Exp. Biol. Med.* 151:237–239.

Rogers, A. E. 1983. Influence of dietary content of lipids and lipotropic nutrients on chemical carcinogenesis in rats. *Cancer Res.* 43:2477s–2484s.

Rogers, A. E., and W. C. Wetsel. 1981. Mammary carcinogenesis in rats fed different amounts and types of fat. *Cancer Res.* 41(9, part 2):3735–3737.

Seiquer, I., M. Mañas, E. Martinez-Victoria, et al. 1994. Effects of adaptation to diets enriched with saturated, monounsaturated and polyunsaturated fats on

[2]Available for review: Director, Cosmetic Ingredient Review, 1101 17th Street, NW, Suite 310, Washington, DC 20036-4702, USA.

lipid and serum fatty acid levels in miniature swine. *Comp. Biochem. Physiol. Comp. Physiol.* 108:377–386.

Sessions, V. A., and A. M. Salter. 1994. The effects of different dietary fats and cholesterol on serum lipoprotein concentrations in hamsters. *Biochim. Biophys. Acta* 1211:207–214.

Silberberg, M., and R. Silberberg. 1954. Factors modifying the life span of mice. *Am. J. Physiol.* 177:23–26.

Silberberg, R., and M. Silberberg. 1955. Life span of mice fed a high fat diet at various ages. *Can. J. Biochem. Physiol.* 33:167–173.

Sinkeldam, E. J., C. F. Kuper, M. C. Bosland, V. M. Hollanders, and D. M. Vedder. 1990. Interactive effects of dietary wheat bran and lard on N-methyl-N′-nitro-N-nitroguanidine-induced colon carcinogenesis in rats. *Cancer Res.* 50:1092–1096.

Swern, D., ed. 1979. *Bailey's industrial oil and fat products*, 4th ed., Vol. 1, 332–340. New York: John Wiley & Sons.

Sylvester, P. W., M. Russell, M. M. Ip, C. Ip. 1986. Comparative effects of different animal and vegetable fats fed before and during carcinogen administration on mammary tumorigenesis, sexual maturation, and endocrine function in rats. *Cancer Res.* 46:757–762.

Thomasson, H. J. 1955. The biological value of oils and fats. I. Growth and food intake on feeding with natural oils and fats. *J. Nutr.* 56:455–468.

Vinson, L. J., and L. R. Cerecedo. 1944. Growth, reproduction and lactation in rats maintained through four generations on highly purified diets. *Arch. Biochem.* 3:389–397.

Wenninger, J. A., G. N. McEwen, Jr., eds. 1997. *International cosmetic ingredient dictionary and handbook*, 7th ed., 624–625, 721. Washington, DC: CTFA.

Woutersen, R. A., and A. van Garderen-Hoetmer. 1988. Inhibition of dietary fat promoted development of (pre)neoplastic lesions in exocrine pancreas of rats and hamster by supplemental selenium and beta-carotene. *Cancer Lett.* 42:79–85.

Zhang, X. B., K. Tao, C. Urlando, P. Shaver-Walker, and J. A. Heddle. 1996. Mutagenicity of high fat diets in the colon and small intestine of transgenic mice. *Mutagenesis* 11:43–48.

2017 VCRP Data for Lard-Derived Ingredients

03A - Eyebrow Pencil	HYDROGENATED LARD GLYCERIDE	1
10A - Bath Soaps and Detergents	LARD	4
12C - Face and Neck (exc shave)	LARD GLYCERIDE	1
12F - Moisturizing	LARD GLYCERIDE	1
		2

There were no reported uses in the 2017 VCRP for:

Lard Glycerides
Hydrogenated Lard Glycerides
Hydrogenated Lard

Data from 1984 indicated use of **Lard Glyceride** at concentrations of ≤10%, **Hydrogenated Lard Glyceride** at ≤1%, and **Lard** at ≤10% (FDA 1984).

No historical or currents uses or concentrations of use reported for:

Hydrogenated Lard
Lard Glycerides
Hydrogenated Lard Glycerides

Personal Care Products Council
Committed to Safety, Quality & Innovation

Memorandum

TO: Lillian Gill, D.P.A.
Director - COSMETIC INGREDIENT REVIEW (CIR)

FROM: Beth A. Lange, Ph.D.
Industry Liaison to the CIR Expert Panel

DATE: February 17, 2016

SUBJECT: Concentration of Use by FDA Product Category: Lard Ingredients

Concentration of use by FDA Product Categories – Lard Ingredients*

Lard Glyceride
Hydrogenated Lard Glyceride
Lard Glycerides
Hydrogenated Lard Glycerides
Lard
Hydrogenated Lard

Ingredient	Product Category	Maximum Concentration of Use
Lard Glyceride	Tonics, dressings and other hair grooming aids	1.6%

*Ingredients included in the title of the table but not found in the table were included in the concentration of use survey, but no uses were reported.

Information collected 2015-2016
Table prepared February 16, 2016