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Coconut oil

MAK Value Documentation – Translation of the German version from 2019

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Abstract

The German Commission for the Investigation of Health Hazards of Chemical Compounds in the Work Area has evaluated coconut oil [8001-31-8] to derive a maximum concentration at the workplace (MAK value), considering all toxicological endpoints. Available publications are described in detail. There are no inhalation studies with coconut oil available. Since coconut oil is a viscous liquid that is also used in metalworking fluids, exposure to a coconut oil aerosol is possible at these workplaces. As with white mineral oil, inhalation of the hardly water-soluble coconut oil could result in overload in the lung, inflammatory reactions and microgranulomas. To prevent this overload, a MAK value of 5 mg/m^3 is derived for the respirable fraction by analogy with white mineral oil and Peak Limitation Category II as well as an excursion factor of 4 are set. There are no developmental toxicity studies with coconut oil. The inhalative uptake by exposure at the MAK value is far lower than the recommended consumption of total fat for women. The degradation products, the saturated fatty acids capric, lauric, myristic and palmitic acid, as well as the unsaturated oleic and linoleic acid, are not expected to be teratogenic. Secondary effects on the foetus by hypoxia, which can be induced by the overload effect in the lung, could be excluded at the level of the MAK value by the data on other lubricant oils. Therefore, damage to the embryo or foetus is unlikely when the MAK value is not exceeded and coconut oil is classified in Pregnancy Risk Group C. Coconut oil is not genotoxic in bacteria. From limited carcinogenicity studies, a carcinogenic potential of coconut oil is not expected. There are no indications of a contact sensitizing potential of coconut oil. Skin contact is not expected to contribute significantly to systemic toxicity.

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MAK value (2018)	**5 mg/m³ R (respirable fraction)**
Peak limitation (2018)	**Category II, excursion factor 4**
Absorption through the skin	–
Sensitization	–
Carcinogenicity	–
Prenatal toxicity (2018)	**Pregnancy Risk Group C**
Germ cell mutagenicity	–
BAT value	–

Synonyms	copra oil
Chemical name	coconut oil
CAS number	8001-31-8
Formula	no data
Molar mass	no data
Melting point	21–25 °C (NCBI 2020)
Boiling point	no data
Density at 20 °C	0.9219 g/cm³ (ECB 2000)
Vapour pressure	no data
log K_{OW}	no data
Solubility	insoluble in water; 1.1×10^{-12} mg/l water at 25 °C (calculated, NCBI 2020)
Stability	on contact with air, the oil is readily oxidized and turns rancid (NCBI 2020)
Production	hydraulic pressing or expeller extraction from the coconut pulp (copra) of the coconut palm (Cocos nucifera), followed by alkali refining, bleaching and deodorization (NCBI 2020)
Purity	no data
Impurities	free fatty acids, low concentrations of sterols, tocopherol, squalene (Burnett et al. 2011; CIR Expert Panel 1986)
Uses	ingredient of soaps, ointments, emulsions, cosmetic products, food, candles, detergents, paints, varnish, lubricants (NCBI 2020)

Coconut oil can be contained in metal-working fluids up to a maximum proportional mass of 20% (Hartwig 2014, available in German only).

Coconut oil is obtained from the coconut pulp of the coconut palm and comprises its extracts and its physically modified derivatives. It consists mainly of triglycerides with mostly saturated fatty acid residues (in total 90%), for example the saturated fatty acids capric acid ($C_{10}H_{20}O_2$, 6%–10%), lauric acid ($C_{12}H_{24}O_2$, 44%–52%), myristic acid ($C_{14}H_{28}O_2$, 13%–19%) and palmitic acid ($C_{16}H_{32}O_2$, 8%–11%), as well as the monounsaturated oleic acid ($C_{18}H_{34}O_2$, 5%–8%) and the diunsaturated linoleic acid ($C_{18}H_{32}O_2$, traces up to 2.5%) (CIR Expert Panel 1986). There is an ECHA infocard on the substance (ECHA 2017). Coconut oil is a mixture, whose qualitative or quantitative composition is unknown or variable (UVCB: Unknown or Variable Composition, Complex Reaction Products or Biological Materials) (NCBI 2020).

Documentation is available for myristic acid and palmitic acid (Greim 1999, available in German only). For oleic acid, documentation is available from 2001 (Greim 2002) and a supplement from 2016 (Hartwig and MAK Commission 2017 a).

1 Toxic Effects and Mode of Action

In rabbits the substance is not irritating to the skin and eyes.

There is no evidence that coconut oil has a sensitizing effect on skin or the airways.

Comprehensive toxicological studies after repeated administration are not available. In animal experiments, systemic effects such as elevated cholesterol levels in the blood occur only at doses in the gram per kg body weight range after the administration of coconut oil with the feed. In view of its structure, high systemic toxicity is not suspected. After inhalation of coconut oil, macrophage accumulation in the lungs and the formation of microgranulomas are to be expected. However, inhalation studies are not available.

Coconut oil is not mutagenic in bacteria and does not lead to the formation of etheno-DNA adducts in leukocytes, the liver, the prostate and colon epithelial cells in rats.

There are no carcinogenicity studies with coconut oil carried out according to valid test guidelines.

2 Mechanism of Action

In the liver there is a mechanism that regulates the toxic effects of medium-chain fatty acids (C10 to C14), steatosis and inflammation. The C10 and C12 fatty acids capric and lauric acid contained in coconut oil induce the hepatic ω-oxidation genes for CYP4A10 and CYP4A14, thus increasing the production of dicarboxylic fatty acids. In addition, these fatty acids activate ω and β-oxidation pathways via the peroxisome proliferator activated receptor (PPAR)α and PPARγ. This activation loop is normally controlled by the degradation of dicarboxylic fatty acids. Degradation is catalysed by the peroxisomal enzyme enoyl-CoA-hydratase/L-hydroxyacyl-CoA-dehydrogenase (L-PBE). L-PBE-deficient mice, which were given feed containing coconut oil (334 g coconut oil/kg diet; doses of about 40 000 mg/kg body weight and day, conversion factor 0.12 for subacute studies according to EFSA (2012)) for 21 to 24 days, accumulated dicarboxylic fatty acids. This leads to the activation of all fatty acid oxidation pathways and thus to steatosis, fibrosis and inflammation in the liver, although the mechanism is still unknown. It is possible that reactive oxygen species are formed. Thus, the correct homoeostasis of dicarboxylic fatty acids is a means to regulate the efficient utilization of C8 to C12 fatty acids in food. Its deregulation highlights the complicated relationship between disturbed metabolism and inflammation (Ding et al. 2013).

3 Toxicokinetics and Metabolism

3.1 Absorption, distribution, elimination

Absorption

In clinical studies, in which volunteers orally received 50 000 to 140 000 mg coconut oil for 3 days, about 98% was absorbed (no other details; CIR Expert Panel 1986).

In rats, after a single dose of coconut oil of 6000 mg/kg body weight, oral absorption amounted to 60% (no other details; CIR Expert Panel 1986).

After oral uptake, glycerides in the duodenum are cleaved by pancreatic lipase into fatty acids, glycerol and monoacylglycerols or diacylglycerols and absorbed into the enterocytes (Greim 1999). It is generally assumed that the chain length determines oral absorption. While free fatty acids with a chain length of C8 to C12 (in coconut oil: capric and lauric acid) reach the liver directly via the portal vein, free fatty acids with a chain length of C14 and longer (in coconut oil: myristic and palmitic acid) react via esterification with glycerol to form triglycerides (Roche and Clark 1994). In serum, the triglycerides are bound to lipoproteins or transported as chylomicrons via the lymphatic system and stored in fatty tissue. Free fatty acids, which are released from the fatty tissue, are either bound to serum albumin or remain in the blood as non-esterified fatty acids. The physiological concentration of free fatty acids in blood plasma is 10 to 300 mg/l. The enteral absorption of free fatty acids decreases with increasing chain length (Greim 1999).

There are no experiments available for the elimination and half-life of coconut oil.

3.2 Metabolism

The breakdown of fatty acids takes place in fat metabolism via successive β-oxidation of the respective terminal C2 unit as the acetic acid thioester of coenzyme A. The breakdown of fatty acids can also take place in the liver to a lesser extent via ω-oxidation and in the brain via α-oxidation. In the form of their triglycerides, fatty acids are natural components of vegetable and animal fats (neutral fats) and are subject to the general fatty acid metabolism (Greim 1999).

4 Effects in Humans

4.1 Single exposures

There are no data available.

4.2 Repeated exposure

No workplace studies are available for coconut oil.

The advantages and disadvantages of the intake of coconut oil via food are controversial. As it contains a high proportion of saturated fatty acids, an increased intake is considered potentially harmful to health. On the other hand, studies are available that prove there are beneficial effects (Ding et al. 2013).

In humans, the high dietary intake of saturated fatty acids is associated with elevated blood cholesterol levels and is associated with an increased risk of coronary heart disease (EFSA 2011).

4.3 Local effects on skin and mucous membranes

On contact with skin or eyes, coconut oil can be irritating. Inhalation may cause irritation of the respiratory tract (no other details; NCBI 2020). This is a standard database entry without any further details of the origin of the information. This statement has therefore not been used in this evaluation.

4.4 Allergenic effects

Sensitizing effects on the skin

There are no clinical findings.

Negative results in repeated insult patch tests with cosmetic products containing 2.5% coconut oil or 10% hydrogenated coconut oil as well as studies of photocontact sensitization in which 1% to 3% preparations of soaps containing 13% coconut oil were used (Burnett et al. 2011; CIR Expert Panel 1986) cannot be included in the evaluation due to the low substance concentrations or the preceding purification step.

Sensitizing effects on the airways

There are no data available.

4.5 Reproductive and developmental toxicity

There are no data available.

4.6 Genotoxicity

There are no data available.

4.7 Carcinogenicity

There are no workplace studies available.

There are also no meaningful studies regarding the relationship between individual fats in the diet and the occurrence of various diet-related tumours.

5 Animal Experiments and in vitro Studies

5.1 Acute toxicity

5.1.1 Inhalation

There are no data available.

5.1.2 Oral administration

Ten rats were given gavage doses of undiluted coconut oil of 5000 mg/kg body weight. During the 7-day follow-up period none of the animals died (no other data; CIR Expert Panel 1986). The oral LD_{50} value is therefore higher than 5000 mg coconut oil/kg body weight.

5.1.3 Dermal application

A single dose of 3000 mg undiluted coconut oil/kg body weight was applied to the skin of 12 guinea pigs. No deaths occurred within the 7-day follow-up period (no other data; CIR Expert Panel 1986). The dermal LD_{50} value is higher than 3000 mg coconut oil/kg body weight.

5.2 Subacute, subchronic and chronic toxicity

5.2.1 Inhalation

There are no data available.

5.2.2 Oral administration

Studies according to valid test guidelines are not available.

Coconut oil was frequently used in studies as an example substance of a fat with a high proportion of saturated fatty acids and led to increases in the serum cholesterol concentration (no other details; CIR Expert Panel 1986). For example, in a total of 6 male and female pigs, the administration of 10% coconut oil in the feed resulted after 8 weeks in a 50% higher serum cholesterol concentration compared with that in the group not given coconut oil (Jurgens et al. 1970).

The studies with repeated oral administration are summarized in Table 1. Comprehensive toxicological investigations are not available. NOAELs (no observed adverse effect levels) cannot be derived from these studies. In general, it can at least be stated that the increased intake of coconut oil via feed leads to increased cholesterol levels in the blood and could result in fat deposits in the liver. However, this is only to be expected in dosages in the gram per kg body weight range. In view of the structure of the individual components, the glycerides, coconut oil is not expected to cause high systemic toxicity.

Tab. 1 Effects of coconut oil after repeated oral administration

Species, strain, number per group	Exposure	Findings	References
rat, Wistar, 13 ♂	**4 weeks**, 0, 31, 218 g/kg diet (about 0, 3720, 26 160 mg/kg body weight and day) [a], comparison group: sunflower oil	only activity of protein kinase C investigated, no effects	Pajari and Mutanen 1999
rat, Wistar, 8 ♀	**7 weeks**, 0, 300 g coconut oil/kg diet, (about 0, 15 908 mg/kg body weight and day calculated on the basis of 682 g total feed consumption over 7 weeks and a final body weight of 263 g)	**15 908 mg/kg body weight**: body weights ↑, weight of perimuscular white fatty tissue ↑, feed consumption ↓, total energy intake ↑, weight of interscapular brown fatty tissue ↑, UCP1 activity ↑; no histological examinations	Portillo et al. 1998
rat, Wistar, 12 ♂ and 13 ♀	**90 days**, 0, 250 g coconut oil/kg diet, about 0, 22 500 mg/kg body weight and day [b], controls: standard diet, examinations after 15, 30, 60 and 90 days	**22 500 mg/kg body weight**: liver: slight infiltration of fat in cytoplasm; histological examinations of the heart, lungs, kidneys, spleen, gastrointestinal tract, skin, muscle, subcutaneous fatty tissue, bones (femur) and body weights without unusual findings; organ weights were not determined	Harris and Mosher 1940

Tab. 1 *(continued)*

Species, strain, number per group	Exposure	Findings	References
monkey, Cebus, 5–7 ♂	**at least 3 years**, 0, 126 g coconut oil/kg diet, (about 0, 6300 mg/kg body weight and day assuming a body weight of 5 kg and feed consumption of 250 g/day), controls: corn oil with and without 0.1% cholesterol	**about 6300 mg/kg body weight**: plasma: total cholesterol ↑ (+217% compared with in the corn oil group), HDL cholesterol ↑, VLDL + LDL cholesterol ↑, Apo A-I ↑, proportion of triglycerides in HDL ↓, proportion of phospholipids in HDL ↑, proportion of protein in HDL ↓, liver: Apo A-I mRNA ↑; liver biopsies were performed, however no results reported	Stucchi et al. 1991

[a)] conversion factor 0.12 for subacute studies according to EFSA (2012)
[b)] conversion factor 0.09 for subchronic studies according to EFSA (2012)
Apo A-I: apolipoprotein A-I; HDL: high density lipoprotein; LDL: low density lipoprotein; UCP1: uncoupling protein 1; VLDL: very low-density lipoprotein

5.2.3 Dermal application

There are no data available.

5.3 Local effects on skin and mucous membranes

5.3.1 Skin

In an unpublished occlusive patch test in 9 rabbits with undiluted coconut oil applied to the skin for 24 hours, no irritation was observed (no other details; CIR Expert Panel 1986).

5.3.2 Eyes

Undiluted coconut oil was instilled into the conjunctival sac of one eye of 6 rabbits per treatment group. Without washing out the substance, irritation values of 2 and 1 were obtained for the two treatment groups (of a maximum 110). This was interpreted as an indication of slight eye irritation (no other details; CIR Expert Panel 1986). The original reports of the two studies cited are not available. The test substance was not washed out as described in the OECD test guideline. Therefore, the very low irritation values are to be interpreted as evidence that coconut oil does not cause irritation in the rabbit eye.

5.4 Allergenic effects

5.4.1 Sensitizing effects on the skin

In a maximization test with 10 Dunkin Hartley guinea pigs, coconut oil was used as a 5% preparation in propylene glycol for intradermal induction treatment and undiluted for topical induction treatment. Before the topical induction treatment, a 5% preparation of sodium lauryl sulfate was applied non-occlusively to the animals' skin. During the 24-hour occlusive challenge treatment with 50% and undiluted coconut oil two weeks later, neither irritant nor allergic reactions were observed (Burnett et al. 2011; CIR Expert Panel 1986).

A modified Buehler test with 9 occlusive 6-hour applications of a 5% ethanolic preparation of hydrogenated coconut oil did not lead to more pronounced reactions in any of the 15 treated animals than in the 5 control animals during the challenge treatment two weeks later with the same preparation (no other details) (Burnett et al. 2011; CIR

Expert Panel 1986). However, due to the low test concentration and the preceding hydrogenation of the substance, this result cannot be used for the evaluation.

5.4.2 Sensitizing effects on the airways

There are no data available.

5.5 Reproductive and developmental toxicity

5.5.1 Fertility

There are no data available.

5.5.2 Developmental toxicity

Studies carried out according to valid test guidelines are not available.

Six pregnant C57BL/6J mice per dose and time of examination were given coconut oil doses of 0, 20, 80, 160 or 240 g/kg diet (corresponding to about 0, 2400, 9600, 19 200, 28 800 mg/kg body weight and day, conversion factor 0.12 for subacute studies according to EFSA (2012)) from the day of insemination until 2, 4 or 6 weeks after birth. A similarly high calorie intake in the control group was achieved by adding casein to the feed. Animals given safflower oil, which in contrast to coconut oil has a high proportion of polyunsaturated fatty acids, served as a comparison group. Several parameters for recording immune function, for example serum concentrations of the immunoglobulins IgG1 and IgG2 and the percentage of immunoglobulin-positive spleen cells, were changed. Therefore, the authors concluded that the fats in the diet affect the modulation of immune function (Erickson et al. 1980).

5.6 Genotoxicity

5.6.1 In vitro

Coconut oil was separated by silica gel column chromatography into a non-polar and a polar fraction. Both fractions, dissolved in tetrahydrofuran, were tested on the Salmonella typhimurium strains TA97 and TA100 at concentrations of 0, 100, 1000, 5000 and 10 000 µg/plate both with and without the addition of a metabolic activation system and were found not to be mutagenic. No cytotoxicity was observed up to the highest concentration tested (Hageman et al. 1991).

In a bacterial mutagenicity test with the Salmonella typhimurium strains TA98, TA100, TA1535 and TA1537, which was not considered valid in the European Chemicals Bureau report, coconut oil was found not to be mutagenic up to a concentration of 5000 µg/plate (no other details; ECB 2000).

5.6.2 In vivo

Groups of 4 to 6 male and female BD-VI rats were given gavage doses of 0 or 500 mg coconut oil (saturated fatty acids), linoleic acid (ω−6 fatty acid 18 : 2, number of carbon atoms:number of double bonds) or oleic acid (ω−9 fatty acid 18 : 1) per kg body weight and day for 30 days. Coconut oil and oleic acid led to the formation of twice as much 1,N6-ethenodeoxyadenosine, an etheno-DNA adduct, in epithelial cells of the mammary gland of female animals compared with linoleic acid. The only slight increase was explained by an altered composition of the phospholipid membrane or the metabolism of free fatty acids in the liver. In leukocytes, the liver, the prostate and colon epithelial cells, coconut oil did not cause the formation of etheno-DNA adducts (Fang et al. 2007).

5.7 Carcinogenicity

5.7.1 Short-term tests

In female rats a high fat content in the diet had a promoting effect on the development of mammary gland tumours in animals initiated with 7,12-dimethylbenz[a]anthracene. Coconut oil with predominantly saturated fatty acids proved to be less effective than polyunsaturated fatty acids such as ethyl linoleate (Carroll and Hopkins 1979; CIR Expert Panel 1986; Hopkins and Carroll 1979; Hopkins et al. 1981).

Female F344 rats were divided into 5 groups of 30 animals and were fed from the age of 21 to 25 days with low levels of corn oil (group 1, control), high levels of corn oil (group 2), lard (group 3), beef tallow (group 4) and coconut oil (group 5). At the age of 50 days the animals received a single intravenous injection of *N*-nitrosomethylurea (50 mg/kg body weight). After 28 weeks, the tumour incidences of adenocarcinomas in the mammary gland were 33%, 85%, 63%, 50% and 43% in groups 1 to 5, respectively. Only in the group with the high level of corn oil (group 2), was the increase in the incidence statistically significant compared with that in the control group. The *N*-nitrosomethylurea-induced carcinogenesis of mammary gland tumours in rats was enhanced by high concentrations of fat in the diet. The extent of amplification depended on the type of fat added to the diet. A positive correlation was found between the total dietary intake of oleic acid or linoleic acid and the tumour incidence (Chan et al. 1983).

In another study, 30 female F344 rats were given 23% safflower oil, corn oil, olive oil or coconut oil per kg feed (similarly high fat content, similar calorie intake) for 22 weeks. After initiation with *N*-nitrosourea, there was an increased incidence of adenocarcinomas of the mammary gland compared with that in animals fed coconut oil and a reduced latency period in animals fed safflower or corn oil. Therefore, the authors concluded that the composition of fatty acids and not the fat content plays a role in the promotion of mammary gland tumours (Cohen et al. 1986 a, b).

Forty-four female C3H mice were fed a diet containing 10% coconut oil (body weight of 34 g after 27 weeks and a feed intake of 4 g/day) or other fats for 65 weeks. With an increasing content of linoleic acid (18 : 2), and a decreasing content of other fatty acids, the incidence of adenocarcinomas of the mammary gland increased and the time to the appearance of tumours decreased. The saturated fatty acids lauric acid (12 : 0), myristic acid (14 : 0) and palmitic acid (16 : 0), and the monounsaturated fatty acid oleic acid (18 : 1) had no significant effects. In the case of the saturated fatty acid stearic acid (18 : 0) there was even a reduction in the tumour incidence (Tinsley et al. 1981).

5.7.2 Long-term tests

A carcinogenicity study is not available.

6 Manifesto (MAK value/classification)

As coconut oil is a viscous oil, aerosol exposure may be expected. As with white mineral oil, this could lead to the accumulation of macrophages in the lungs and the formation of microgranulomas. However, inhalation studies with coconut oil are not available. The systemic toxicity is low.

MAK value. As it is a viscous oil, coconut oil can lead to effects in the lungs similar to those caused by white mineral oil. In the case of white mineral oil, an overload effect in the lungs is observed (Hartwig and MAK Commission 2017 b). While white mineral oil is not hydrolysable, coconut oil may be subject to hydrolysis. When the hydrolysis capacity is exceeded, overload can occur also with coconut oil. The limit of the hydrolysis capacity is not known, which means it is unclear when the overload effect is to be expected. The assumption that coconut oil can lead to an overload effect therefore represents a “worst case”.

In analogy to white mineral oil, a provisional MAK value of 5 mg/m³ R (respirable fraction) has therefore been set for exposure to coconut oil alone. This concentration is not reached in the case of exposure to a mixture of substances such as with metal-working fluids as long as the corresponding technically based limit value of 10 mg/m³ I (inhalable fraction) is not exceeded, because metal-working fluids contain a maximum of 20% coconut oil. The expected amount of coconut oil inhaled and absorbed dermally at the workplace is several orders of magnitude lower than that taken in with food. If the technically based limit value for metal-working fluids of 10 mg/m³ I is observed, an intake of 20 mg (10 m³/8 hours, 100% absorption), corresponding to 0.3 mg/kg body weight and day at a body weight of 70 kg, can be calculated for this workplace. Even in the case of exposure to coconut oil alone, absorption by inhalation would be in the mg per kg body weight range. Intake via food, on the other hand, is in the gram per kg body weight range. A systemic effect is therefore not to be expected.

A possible component of natural vegetable oils are lecithins. Due to their polar and non-polar properties, lecithins (phosphatidylcholines) can have surfactant-like effects and therefore enter into a reaction with the surfactant in the lungs. No data are available for the lecithin content of coconut oil. The raw oil of the soybean, which is the main source in the production of lecithin, contains about 1.8% (1.2% to 3.2%) lecithin (Shurtleff and Aoyagi 2016). The lecithin content in coconut oil is therefore probably far below 2%. A surfactant-like effect resulting from lecithins is therefore unlikely.

Peak limitation. As described for pharmaceutical white mineral oil, effects are cumulative and occur late (Hartwig and MAK Commission 2017 b), so that coconut oil has been assigned to Category II. In analogy to pharmaceutical white mineral oil and the polyalphaolefins, an excursion factor of 4 has been set for peak limitation, as very high short-term concentrations might change the distribution behaviour in the alveoli and thus their dwelling time, and the formation of microgranulomas in the lungs must be prevented (Hartwig 2011, available in German only).

Prenatal toxicity. There are no developmental toxicity studies carried out according to valid test guidelines available for coconut oil.

As a result of possible effects of the substances contained in coconut oil, analogy with pharmaceutical white mineral oil, as was assumed for the MAK value, is not possible for the prenatal toxicity of coconut oil.

As there are no developmental toxicity studies available, coconut oil would formally be assigned to Pregnancy Risk Group D.

Fats are one of the main constituents of the human diet. The EFSA recommends a total uptake of fat in the range of 20% to 35% of the daily energy intake (EFSA 2010). For 25 to 51-year-old women, 30% of the daily energy intake at an energy reference value of 1800 kcal and low physical activity (a physical activity level of 1.4) corresponds to a total daily intake of 63 g fat (Deutsche Gesellschaft für Ernährung 2017).

At the level of the MAK value of 5 mg coconut oil/m³ R, an absorbed amount of 50 mg is obtained for a woman, corresponding to 0.8 mg/kg body weight and day (assuming 10 m³/8 hours, 100% absorption, 60 kg body weight). Even if absorption of the inhalable fraction is also taken into consideration, the inhalation exposure would still be several orders of magnitude below the recommended total fat intake of 63 g/day, corresponding to 1050 mg/kg body weight and day at a body weight of 60 kg.

In addition to this, teratogenic effects are not expected from the degradation products of coconut oil, such as the saturated fatty acids capric, lauric, myristic and palmitic acid and the monounsaturated oleic acid and the diunsaturated linoleic acid.

In addition to the direct effects of coconut oil, secondary effects on the foetus due to possible hypoxia caused by overload in the lungs have to be taken into consideration. There are no data available for coconut oil, however for other viscous oils. In rats, rabbits, mice, gerbils and dogs, no signs of hypoxia such as cyanosis were observed after inhalation exposure to concentrations of up to 100 mg mineral oil/m³ for 12 to 24 months (Stula and Kwon 1978;

Wagner et al. 1964). Abnormal results in pulmonary function tests in the form of an increased end-expiratory volume were obtained in rats after inhalation of an oil mist of light lubricating oil for 13 weeks in a concentration of 1500 mg/m³ (Selgrade et al. 1990). In several studies, experimentally induced hypoxia in rats occurred together with an increased end-expiratory volume (Bonora and Vizek 1999). Assuming that the increased end-expiratory volume in rats in the above-mentioned 13-week inhalation study is evidence of hypoxia, the LOAEC (lowest observed adverse effect concentration) for this effect is 1500 mg/m³ and the NOAEC (no observed adverse effect concentration) 500 mg/m³. There is, therefore, a 100-fold margin between the NOAEC and the MAK value of 5 mg/m³, and this provides sufficient protection against possible hypoxia caused by coconut oil.

Coconut oil has therefore been assigned to Pregnancy Risk Group C.

Carcinogenicity. A carcinogenicity study carried out according to valid test guidelines is not available for coconut oil. Several initiation–promotion studies in female rats have shown that coconut oil does not have the potential to cause the induction of mamma tumours (Carroll and Hopkins 1979; Chan et al. 1983; CIR Expert Panel 1986; Cohen et al. 1986 a, b; Hopkins and Carroll 1979; Hopkins et al. 1981). The substance is not genotoxic. In view of its structure, carcinogenicity is not suspected. Coconut oil has not been classified in one of the categories for carcinogens.

Germ cell mutagenicity. Studies with germ cells are not available. Coconut oil is not mutagenic in bacteria (Hageman et al. 1991) and in the rat does not lead to the formation of etheno-DNA adducts in leukocytes, the liver, the prostate or in epithelial cells of the colon. In epithelial cells of the mammary gland of female rats, a slight increase in etheno-DNA adducts was induced via an indirect mechanism (Fang et al. 2007). Since the mechanism in question was indirect and its structure does not suggest it may be genotoxic, coconut oil has not been classified in one of the categories for germ cell mutagens.

Absorption through the skin. Quantitative data for dermal absorption are not available. Due to the complex and variable composition of coconut oil, model calculations cannot be used. After dermal application, coconut oil was not found to be acutely toxic up to the highest dose tested of 3000 mg/kg body weight. In view of the regular uptake of coconut oil or components of coconut oil via the diet in the gram per kg body weight range, it is to be expected that even very high levels of penetration through the skin would not lead to systemic effects. Coconut oil has therefore not been designated with an "H" (for substances which can be absorbed through the skin in toxicologically relevant amounts).

Sensitization. There are no positive findings available for sensitizing effects of coconut oil on the skin and airways. The substance is therefore not designated with "Sh" (for substances which cause sensitization of the skin) or "Sa" (for substances which cause sensitization of the airways).

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