Scientific Committee on Consumer Safety

SCCS

OPINION ON

peanut oil

(sensitisation only)

The SCCS adopted this opinion at its 5th plenary meeting
of 27 March 2014
Revision of the Opinion on peanut oil (sensitisation only)

About the Scientific Committees
Three independent non-food Scientific Committees provide the Commission with the scientific advice it needs when preparing policy and proposals relating to consumer safety, public health and the environment. The Committees also draw the Commission's attention to the new or emerging problems which may pose an actual or potential threat. They are: the Scientific Committee on Consumer Safety (SCCS), the Scientific Committee on Health and Environmental Risks (SCHER) and the Scientific Committee on Emerging and Newly Identified Health Risks (SCENIHR) and are made up of external experts.

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SCCS
The Committee shall provide opinions on questions concerning all types of health and safety risks (notably chemical, biological, mechanical and other physical risks) of non-food consumer products (for example: cosmetic products and their ingredients, toys, textiles, clothing, personal care and household products such as detergents, etc.) and services (for example: tattooing, artificial sun tanning, etc.).

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This opinion has been subject to a commenting period of six weeks after its initial publication. Comments received during this time have been considered by the SCCS and discussed in the subsequent plenary meeting. Where appropriate, the text of the relevant sections of the opinion has been modified or explanations have been added. In the cases where the SCCS after consideration and discussion of the comments, has decided to maintain its initial views, the opinion (or the section concerned) has remained unchanged. Revised opinions carry the date of revision.

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1. BACKGROUND

Peanut oil, with the Inci name Arachis hypogaea oil, and its derivatives have a widespread use in cosmetic products. The use of peanut oil in cosmetic products is not currently regulated in the Cosmetics Directive. Several Member States have recently indicated safety problems in relation to the use of this substance as an ingredient in cosmetic products.

Concerns were raised over the fact that an unexpected risk of food allergy to peanuts was reported in particular at young children (0-3 years), where it was suspected that the induction of the sensitisation might have appeared through the use of cosmetic products containing peanut oil in the first six months of life.

A public call for scientific data on the use of peanut oil in cosmetic products was made by the Commission Service during autumn/winter 2009-10.

2. TERMS OF REFERENCE

1. Does the SCCS consider the use of peanut oil and/or its derivatives to be safe for consumers in cosmetic products on the basis of the provided scientific data?

2. And/or does the SCCS has any scientific concerns with regard to the use of peanut oil and/or its derivatives in cosmetic products?
3. OPINION

3.1 Chemical and Physical Specifications

3.1.1 Chemical identity

Peanut (Arachis hypogaea) kernels contain approximately 45.5-50% fat, 25-30% protein, 8-12% carbohydrate, 5% water, 3% fiber and 2.5% ash (CIR 2001, Koppelman 2001). The protein content of refined oils, including peanut oil, was shown to be about 100 fold lower than that in cold pressed oil (Crevel et al. 2000). The protein content of refined oils was found to be 0.2-60 mg/L. However, in one study protein content of refined peanut oil was reported to be < 0.3µg/L (Peeters et al. 2004). The refining process, which also included heat treatment, did not destroy the allergenicity of the peanut allergens (Olszewski et al. 1998, Koppelman et al. 1999). This indicates that the major peanut allergens are heat stable even when present in trace amounts in refined peanut oil.

Thirteen peanut allergens (Ara h 1 to Ara h 13) have been identified (Table 1, WHO-IUIS. For detailed description of these allergens see also the review by de Leon et al. (2007).

Table 1. Peanut allergens (WHO-IUIS)

<table>
<thead>
<tr>
<th>Allergen</th>
<th>Biochemical name</th>
<th>Molecular Weight (SDS-PAGE), kDa</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ara h 1</td>
<td>Cupin (Vicillin-type, 7S globulin)</td>
<td>64.0</td>
</tr>
<tr>
<td>Ara h 2</td>
<td>Conglutin (2S albumin)</td>
<td>17.0</td>
</tr>
<tr>
<td>Ara h 3</td>
<td>Cupin (Legumin-type, 11S globulin, Glycinin)</td>
<td>60.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>37.0 (fragment)</td>
</tr>
<tr>
<td>Ara h 4</td>
<td>renamed to Ara h 3.02, number not available for future submissions</td>
<td></td>
</tr>
<tr>
<td>Ara h 4</td>
<td>Profilin</td>
<td>15.0</td>
</tr>
<tr>
<td>Ara h 6</td>
<td>Conglutin (2S albumin)</td>
<td>15.0</td>
</tr>
<tr>
<td>Ara h 7</td>
<td>Conglutin (2S albumin)</td>
<td>15.0</td>
</tr>
<tr>
<td>Ara h 8</td>
<td>Pathogenesis-related protein, PR-10</td>
<td>17.0</td>
</tr>
<tr>
<td>Ara h 9</td>
<td>Nonspecific lipid-transfer protein 1</td>
<td>9.8</td>
</tr>
<tr>
<td>Ara h 10</td>
<td>16 kDa oleosin</td>
<td>16.0</td>
</tr>
<tr>
<td>Ara h 11</td>
<td>14 kDa oleosin</td>
<td>14.0</td>
</tr>
<tr>
<td>Ara h 12</td>
<td>Defensin</td>
<td>8 (reducing), 12 (non reducing), 5.184 (mass)</td>
</tr>
</tbody>
</table>
The allergens Ara h1 and Ara h 2 have been shown to be the major allergens of peanut oil (Burks et al. 1991, 1995, Koppelman et al. 2001, Maleki et al. 2000). It has been shown that Ara h 2 and Ara h 6 are moderately homologous allergens and can act either synergistically or in a redundant fashion, and partial similarity between these two allergens has been demonstrated (Porterfield et al 2009, Chen et al. 2013, Koid et al. 2014). Koppelman et al. (2010) have recently shown that Ara h 2 and Ara h 6 are considerably more stable towards digestion than Ara h 1 and Ara h 3.

Cosmetic grade peanut oil is the refined (or hot pressed oil) fixed oil of one or more of the cultivated varieties of Arachis Hypogaea. Functions and uses of peanut oil according to the COSING Database is described in Table 2.

According to information supplied by Industry, peanut oil can be refined to protein levels below 1 ppm, and for some products to a level below the 0.5 ppm detection limit by ELISA (Ramazotti 2008).

### 3.2 Function and uses

Table 2: Function and uses of peanut oil in cosmetics according to the COSING database ([http://ec.europa.eu/consumers/cosmetics/cosing/index.cfm?fuseaction=search.details&id=38405](http://ec.europa.eu/consumers/cosmetics/cosing/index.cfm?fuseaction=search.details&id=38405))

<table>
<thead>
<tr>
<th>INCI Name</th>
<th>Description</th>
<th>CAS No.</th>
<th>EC No.</th>
<th>Cosmetic use/function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arachis Hypogaea Oil</td>
<td>Arachis Hypogaea Oil is the refined fixed oil obtained from the seed kernels of one or more of the cultivated varieties of the Peanut, Arachis hypogaea L., Leguminosae</td>
<td>8002-03-7</td>
<td>232-296-4</td>
<td>Emollient, Solvent</td>
</tr>
<tr>
<td>Hydrogenated peanut oil</td>
<td>Peanut oil, hydrogenated</td>
<td>68425-36-5</td>
<td>270-350-9</td>
<td>Emollient, Emulsifying, Skin conditioning, Viscosity controlling</td>
</tr>
<tr>
<td>Peanut Oil PEG-6 Esters</td>
<td>Peanut (Arachis hypogaea L., Leguminosae) oil, transesterification products with polyethylene oxide (6 mol EO average molar ratio)</td>
<td>68440-49-3</td>
<td>-</td>
<td>Emollient</td>
</tr>
<tr>
<td>Peanutamide MEA</td>
<td>Amides, peanut oil, N-2-hydroxyethyl</td>
<td>93572-05-5</td>
<td>297-433-2</td>
<td>Emulsifying, Emulsion stabilizing, Foam boosting, Surfactant, Viscosity controlling</td>
</tr>
<tr>
<td>Peanutamide MIPA</td>
<td>Amides, peanut oil, N-(2-hydroxypropyl)</td>
<td>-</td>
<td>-</td>
<td>Emulsifying, Emulsion stabilizing, Foam boosting, Surfactant, Viscosity controlling</td>
</tr>
</tbody>
</table>
Information on the concentration of peanut oil in various cosmetic product categories was not available. However, FDA data from 1984 (CIR 2001) indicated that peanut oil was used predominantly at concentration ≤ 25% (19 uses with 1 use at >50%). Frequency and use of peanut oil and hydrogenated peanut oil in cosmetic formulations in the US market are described in CIR 2001. No such data is available for the cosmetic products in the EU.

### 3.3 Toxicological Evaluation

#### 3.3.1 Irritation and corrosivity

##### 3.3.1.1 Skin irritation

The skin irritating potential of peanut oil has been evaluated in 4 different animal species (Motoyoshi et al., 1979).

Undiluted technical grade arachis oil (0.1 g) was applied to the dorsal surface of groups of 6 albino angora rabbits, male Hartley guinea pigs and male Wistar rats (arachis oil was one of 19 oils or 20 synthetic perfumes tested). Three test compounds and one control (n-hexadecane) were applied to the rabbits, whereas only one test substance and one control (n-hexadecane) were applied to the guinea pigs and rats. Sites were evaluated after 24 hours exposure and the test substances were re-applied 30 minutes after the reading. A second set of reading and application was made 48 hours later. After the 72-hour evaluation, the animals were injected with Evan’s blue, killed and a sample of dorsal skin was taken for histopathological examination. The dilating rate of blood vessels, the swelling rate (oedema), the bluing rate (as a result of increased capillary permeability), and the bleeding rate on the test sites were read using transmitting light. The total score of the averages of the reddening rate (erythema, 72-hour reading), the dilating rate, the swelling rate and the bluing rate for 6 animals in a group was referred to as the primary irritation index and was used for placing a compound in general groups with reference to irritant properties. The bleeding rate was used as a reference. Compounds producing the total score (primary irritation index) of 4 or less were mildly irritating whereas those with indexes from 4 to 8 were moderate irritants and those with scores above 8 were considered severe irritants. Arachis oil was moderately irritating to the rabbit and guinea pig and mildly irritating to the rat (Motoyoshi et al. 1979).

Arachis oil (technical grade, 0.05 g) was applied under occlusion for 48 hours to the dorsal surface clipped free of hair, of 6 miniature swine. Sites were evaluated at the time of patch removal and the animals were injected with Evan’s blue, killed and dorsal skin samples were taken for histopathological examination. Reactions were evaluated as described above. Arachis oil was not irritating to the miniature swine (Motoyoshi et al. 1979).

SCCS comment

The specific number and grade of reactions were not reported in the original article by Motoyoshi et al. (1979).
The ocular toxicity of ketoconazole has been tested in rabbits. One eye of each rabbit (18 animals) was treated with ketoconazole in an arachis oil vehicle (test eyes) and the other eye (control eyes) was treated with undiluted arachis oil. Drops were applied hourly for 6 consecutive hours daily for 3 weeks. Eyes were examined by slit-lamp biomicroscopy before the first instillation and twice weekly for the 3-week experimental period. A 4-point scoring system was used for various sites in the eye and for various parameters (discharge, oedema and hyperaemia). Six of the 18 control eyes that received arachis oil showed ‘a small degree of hyperaemia’ (grade 1 out of 4) involving the bulbar conjunctiva in 4 eyes and the eyelid in 2 eyes (Oji, 1982).

The author’s conclusion was that arachis oil ‘showed no ocular toxicity’.

SCCS comment
The finding of slight hyperaemia in 6 of 18 eyes to which arachis oil was applied seems to indicate that arachis oil can cause slight conjunctival irritation.

### 3.3.2 Skin sensitisation

**Local Lymph Node Assay (LLNA)**

No LLNA study was supplied or referenced for peanut oil, but some other studies are available.

Using peanut flour, Strid *et al.* (2005) demonstrated in female BALB/c mice that epicutaneous exposure to peanut protein induced potent Th2-type immunity with high levels of IL-4 and serum IgE. This was able to prevent the induction of oral tolerance.

Hsieh *et al.* (2003) showed that allergen exposure through the skin in BALB/c mice could serve as a pathway for sensitization for food allergy, but that 100 µg of ovalbumin applied to a 1 cm square patch for 1 week was required to induce a positive challenge. This is more than a million times higher than the detection limit of the ELISA described by Peeters *et al.* (2004).

### 3.3.3 Dermal / percutaneous absorption

No data provided.

### 3.3.4 Photo-induced toxicity

No data available.

### 3.3.5 Human data

**Background to consideration of the role of percutaneous sensitization**

Nipple creams often contain arachis oil, as do topical preparations for eczema (Lever, 1996). Out of 406 patients reporting symptoms on first contact with peanuts, only 121 (19%) had been knowingly exposed to peanuts before the first documented reaction (Hourihane *et al.*, 1997). Peanut allergy was much more common in the group of children fed vitamin D preparation containing peanut oil than those who took the vitamin D product free from peanut oil (De Montis *et al.* 1993). Lack *et al.* (2003) found a significant association between consumption of soya milk in the first 2 years of life and the development of peanut allergy. About 90% of individuals with peanut allergy were exposed
to skin creams containing peanut oil in the first 6 months of life. Almost 91% of children with peanut allergy (in a study of 49 children with symptoms of peanut allergy) had been exposed to arachis oil in the first 6 months of life. In addition they were exposed to more preparations containing arachis oil. Eczema was a risk factor as was intake of soya milk. Filaggrin gene mutations, with and without concomitant clinically manifest atopic dermatitis, have been shown to be associated with peanut allergy (Brown et al. 2011).

**Consideration of maternal factors**

According to Vadas *et al.* (2001), more than 70% of children with peanut allergy show reactions already at the first known time of ingestion, possibly sensitized via breast feeding since peanut protein is present in breast milk. In a questionnaire study on children with peanut allergy and egg-allergic controls conducted before the subjects were diagnosed as being peanut allergic, conducted by Fox *et al.* (2009), the mean household weekly peanut consumption was significantly elevated in the 133 peanut allergic group compared to 150 controls. Peanut butter posed the greatest risk.

The mothers of infants with peanut allergy reported a statistically significantly higher intake of peanuts during pregnancy and breast-feeding than controls (DesRoches *et al.*, 2010). The peanut allergic infants did not have a higher environmental exposure to peanut than did controls. However, a systematic review has shown that several studies have confounding variables. So much so that there is no clear evidence that maternal exposure or early/delayed exposure to peanuts has an influence on the subsequent development of peanut allergy (Thompson *et al.*, 2010). Therefore conclusions regarding maternal exposures are not possible at this stage. Notwithstanding, in a study of 140 infants with peanut allergy (as judged by specific IgE to peanut of >5 KU/mL), multivariate analysis showed an association with peanut consumption by the mother during pregnancy (odds ratio 2.9, 95% CI 1.7-4.9; P< 0.001) (Sicherer *et al.*, 2010). A yet further analysis has concluded that the risk of childhood peanut allergy was not modified by maternal exposure to peanut-containing food (Binkley *et al.*, 2011).

**SCCS comment**

There is some evidence that infant exposure via breast milk, nipple cream or vitamin D supplements may sensitise infants to peanut proteins. However, it has been pointed out that several studies have confounding factors which make a definite conclusion difficult.

**Symptomatology of peanut allergy**

Out of a series of 122 children with peanut and tree nut allergy, 89% had skin reactions, 52% had respiratory symptoms and 32% had symptoms related to the gastro-intestinal tract (Sicherer *et al.*, 1998).

**Oral challenge**

A double-blind cross-over trial in 10 peanut-allergic patients as judged clinically and by specific IgE was performed of the allergic potential of peanut oil. All subjects were negative to intradermal injection of peanut oil (and to olive oil as a control). Each was tested with each of 1, 2, and 5 mL capsules of peanut oil and olive oil as a control at 30 minute intervals without adverse reaction. Re-testing 2 weeks later was again negative. Ten patients who experienced systemic symptoms after peanut ingestion did not react to skin prick tests or on oral provocation to peanut oil (Taylor et al, 1981). A randomized double-blind crossover challenge study using 62 patients who were skin test positive for peanut allergy, in whom crude or refined peanut oil was administered orally, was negative in all those tested for the refined peanut oil (O’Hourihane et al, 1997).
In 41 children with positive tests for peanut allergy, none reacted to orally-administered refined peanut oil but 15 reacted to unrefined oil (Kull et al, 1999).

Combining the data from various published and unpublished data on clinical oral challenge studies in peanut-sensitised individuals, a New Zealand – Australian Expert Panel established a minimal oral reference dose of 0.2 mg peanut protein (Taylor et al., 2014).

**Dermal irritation**

Undiluted arachis oil (technical grade, 0.05 g) was applied to the back of 50 male volunteers for 48 hours (arachis oil was one of 19 oils or 20 synthetic perfumes tested). The patches were then removed and inspected after 30 minutes (and at 72, 96 and 120 hours if necessary). Compounds were classified in general groups according to their irritation potential. Compounds producing the percentages of positive reactions of 10% or less were considered almost non-irritating; those with percentages of 10-40% were considered as mildly irritating; those with percentages of 40-70% were considered as moderately irritating; and those with percentages above 70% were considered as severely irritating. The exact results are not shown in this paper. Arachis oil was regarded as not being a skin irritant in humans according to these authors (Motoyoshi et al. 1979).

CIR (2001) quotes a study by Frosch and Kligman (1976) in which 5 Caucasian volunteers participated in a chamber-scarification test using USP-grade peanut oil, applied in 100 µL quantities using aluminum chambers, daily for 3 days (length of exposure not stated). The 72-hour reading, done 30 minutes after last patch removal, was used for scoring, producing mean scores of 0-0.4, and assessed by the authors as non-irritating.

**SCCS comment**

Peanut oil is not irritant to the skin.

**3.3.6 Discussion**

Peanut proteins are known to cause severe potentially life-threatening type-I allergic reactions. However, refined peanut oils contain very low levels of the peanut proteins which are the moiety that has the allergenic potential (see below). Nonetheless, apparent allergy to peanut oil has been recorded, the first suspected case being that of a 32-year-old woman with asthma, who developed generalized urticaria related to IM injections of adrenaline in peanut oil in whom subcutaneous tests seemed to confirm allergy to peanut oil (Chafee FH 1941).

The Working Party on Herbal Medicinal Products of the European Medicines Agency (EMEA, 2004) states the view that ‘Since no safe threshold for the exposure to topical oil preparations can be defined and data point to the possibility of allergy induction due to the use of oil containing ointments in infants, all medications for topical use containing soya or peanut products should be treated as allergenic’. The report proceeds to state ‘It must be kept in mind that with chronic oral consumption of oil-based formulas e.g. vitamin D preparations in infants, containing only traces of protein the induction of new allergies cannot be excluded.’ Exposure to peanut allergen may not necessarily occur through exposure to peanut oil; it might occur through exposure to peanut butter (EMEA 2004).

Regarding the allergenicity of peanut proteins, Nordlee et al. (1981) performed a radioallergosorbent test to assess the allergenic potential of various moieties of peanut against the combined sera of 5 peanut allergic patients, as judged by the inhibition of binding of serum IgE to solid-phase peanut allergen. Defatted peanut flour, peanut butter, and raw and roasted peanuts were all allergenic but peanut oil was not as judged by this test. Teuber et al. (1997) tested IgE-binding capacity of nut extracts, using pooled serum from peanut allergic patients. Two minimally-processed peanut oils, with protein
concentrations of 11 microgram/mL, were positive in this study but the refined bleached peanut oils (protein levels 6 and 3 microgram/mL) were respectively negative and showed ‘a very light band of binding’.

Data to derive a safe level of exposure to food in sensitised individuals exist, however to derive a safe level of exposure of the skin (especially regarding induction) is problematic. There is mounting evidence of access of proteins to the immune system via the (even intact) skin (Kimber et al., 2014). This has implications for sensitisation to food proteins. A compromised skin barrier function, promoting this immunological access, is not uncommon in the general population; filaggrin loss-of-function mutations with or without clinically manifest atopic dermatitis have been shown to be a significant risk factor for peanut allergy (Brown et al., 2011). Evidence of enhanced epicutaneous sensitisation to protein (albeit not peanut) in filaggrin deficiency is supported by a study in mice (Oyoshi et al., 2009).

A one-time application of body lotion on the entire skin is approximately 8 ml and a one-time whole body cream application in dermatology patients is advised to be 20 gram (SCCS/1501/12 2012, Long 1991). Thus, a one-time skin application with refined peanut oil with 0.5 ppm would result in a total dose of max 10 microgram peanut protein, which is well below the ED1 level of 200 microgram for ‘safe’ elicitation/challenge studies in sensitised individuals.

4. CONCLUSION

There is no known safe threshold currently defined at which the skin of peanut allergic subjects can safely be exposed to peanut proteins, although such thresholds are available for oral intake.

The SCCS has followed the scientific debate about the importance of skin exposure as a route for induction of sensitisation to type I allergens such as peanut. The SCCS acknowledges that this is of concern, but that there are insufficient data to define a safe level of skin exposure in the non-sensitised population.

However, in view of the documented safe levels of oral intake of peanut protein in sensitised individuals and in view of the industry’s capability to refine peanut oil below a protein level of 0.5 ppm, the SCCS can accept this value as maximum allowable concentration in (refined) peanut oil for cosmetic use.

5. MINORITY OPINION

/
6. REFERENCES

Submission I


2. Adel-Patient K, Ah-Leung S, Bernard H, Durieux-Alexandrenne C, Croeminon C, Wal JM (2007). Oral sensitization to peanut is highly enhanced by application of peanut extracts to intact skin, but is prevented when CpG and cholera toxin are added. Int Arch Allergy Immunol 143: 10-20


4. Company (Croda) statement on the issue

5. Company (Wala) statement on the issue


17. Letter from the French authorities 2009


Additional references evaluated by SCCS


