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Evidence for Cosmetics as a Source of Mineral Oil Contamination in Women

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Abstract

Background: There is strong evidence that mineral oil hydrocarbons are the greatest contaminant of the human body, amounting to approximately 1 g per person. Possible routes of contamination include air inhalation, food intake, and dermal absorption. The present study aims to identify the most relevant sources of mineral oil contamination.

Methods: One hundred forty-two women undergoing elective cesarean section were enrolled. A specimen of subcutaneous fat was removed prior to wound closure. On days 4 and 20 postpartum, milk samples were collected from the women. Fat and milk samples were analyzed for mineral oil saturated hydrocarbons (MOSH). All women completed a questionnaire on personal data, nutrition habits, and use of cosmetics. MOSH concentrations in fat tissue were compared with data from the questionnaire and with MOSH concentrations in corresponding milk samples.

Results: The predominant predictor for MOSH contamination of fat tissue was age ($p < 0.001$). Furthermore, body mass index ($p = 0.001$), country of main residence ($p = 0.03$), number of previous childbirths ($p = 0.029$), use of sun creams in the present pregnancy ($p = 0.002$), and use of hand creams and lipsticks in daily life ($p = 0.011$ and $p = 0.007$, respectively) were significant independent determinants. No association was found with nutritional habits. A strong correlation was seen between MOSH concentration in fat tissue (median 52.5 mg/kg) and in the corresponding milk fat sample from day 4 (median 30 mg/kg) ($p < 0.001$) and day 20 (median 10 mg/kg) ($p = 0.028$).

Conclusions: The increase in MOSH concentration in human fat tissue with age suggests an accumulation over time. Cosmetics might be a relevant source of the contamination.

Introduction

There is strong evidence that mineral oil hydrocarbons are the greatest contaminant of the human body.[1] We have previously shown that mineral oil saturated hydrocarbons (MOSH) amount to approximately 1 g per person and even 10 g in extreme cases.[1] This strong contamination raises the question of the origin of the mineral oil products accumulating in our bodies.

Mineral oil products mainly consist of mineral oil saturated hydrocarbons (MOSH) and mineral oil aromatic hydrocarbons (MOAH). MOSH are open-chain hydrocarbons (paraffins) that include straight-chain alkanes (n-alkanes), branched alkanes (iso-alkanes), and cycloalkanes (naphthenes). MOAH, which range from benzene to polyaromatics of several rings, are mostly alkylated. MOSH are present in crude mineral oil, but also in highly refined mineral oil products. They can be modified by raffination processes such as cracking and polymerization processes using cracking products or hydrogenation.

Humans are exposed to a broad range of mineral oil products. Possible exposure routes include the air, food, and

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the skin (cosmetics and technical products). None of these routes has been satisfactorily investigated.

During winter–spring 2007–2008 some 100,000 metric tons of sunflower oil in the Ukraine were contaminated with mineral oil at concentrations often exceeding 1000 mg/kg. This mineral oil had a composition corresponding to that of base oils for manufacturing lubricating or hydraulic oils and was probably added fraudulently.[2,3] A broad control campaign was initiated throughout Europe to protect consumers, and the European Commission introduced a legal limit of 50 mg/kg for mineral paraffins in Ukrainian sunflower oil. This case drew our attention to many other foods that have also been contaminated with mineral oil.

Until the 1990s, industrial bakeries used release agents consisting of paraffin oil to facilitate removing finished products from baking pans, thereby producing bread and other products with paraffin oil concentrations of 700–3000 mg/kg.[4] Edible oils are frequently contaminated with mineral oil at rates of up to 300 mg/kg and occasionally more than 1000 mg/kg.[5–7] Grapeseed oils, for example, are commonly contaminated with mineral oil material at 30–150 mg/kg, presumably from pesticide formulations.[8] Food packaging material releases mineral waxes and oils at concentrations frequently reaching 60 mg/kg. At present, migration from recycled paperboard and offset printing inks into dry foods is of prime concern.[9–13] Furthermore, paraffin oil is used as a direct food additive in numerous food categories with maximum use levels of 200–3000 mg/kg; e.g., as a dust binder for wheat and rice.[14]

Mineral oils are also widely used in cosmetics and pharmaceutical products. They are excellent moisturizers and emollients and provide a lipophilic base in which to deliver active ingredients. Formulas for creams and lotions, bath oils, lipsticks and lip glosses, sun creams, and hair products often contain mineral oils. In some cases—Vaseline, for instance—they virtually consist exclusively of MOSH.[15]

Only MOSH of a narrow molecular mass range (from about n-C_{17} to n-C_{30}, centered at n-$C_{23/24}$) were found in human body fat, and this composition was almost identical for all women participating in our previous study.[1] This indicates a selective absorbance and elimination process resulting in the accumulation of hydrocarbons that the metabolism cannot handle; it also means that this composition does not provide information about the hydrocarbon source.

Toxicology data regarding MOSH are derived from feeding studies in animals, mainly F344 rats.[16–19] These studies provide evidence that some hydrocarbons not only accumulate, but also cause local foreign body reactions (histopathological granulomata in the liver, histiocytosis in lymph nodes) and induce toxicity (altered serum enzyme levels, altered blood picture, inflammatory lesions at the base of the mitral valve in the hearts of F344 rats). Sensitivity towards MOSH was found to differ between species and rat strains.[20,21] The sensitivity of humans to MOSH is unknown.

There are virtually no data on the toxicity of MOAH. MOAH probably contain genotoxic components, but since they are composed of an enormous number of substances and this composition is often changed during raffination (e.g., by hydrogenation/dehydrogenation) their evaluation is extremely difficult.[22]

In the present study, fat from the abdominal wall was sampled during cesarean section in 142 women and analyzed for MOSH. In addition, human milk from days 4 and 20 after childbirth was collected and analyzed. Our previous manuscript[1] reported data on the concentration and molecular mass distribution of MOSH accumulating in human body fat. The present paper reports on relevant sources for this contamination of the human body. It is based on a questionnaire on personal data, nutrition habits, and use of cosmetics that was completed by all participating women. MOSH concentrations found in fat tissue were correlated with the questionnaire responses and with MOSH concentrations in corresponding milk fat samples.

Materials and Methods

Study design

Between October 2005 and June 2006, 142 women undergoing elective cesarean section (at an average of 38 weeks of gestation) at the Departments of Obstetrics and Gynecology of the Hospital of Bregenz and the Innsbruck Medical University, Austria, were enrolled in the present study after providing informed consent. The study was approved by the Institutional Review Board.

Sample collection

During cesarean section, 1 g of subcutaneous fat was removed when closing the abdominal wall after delivery of the neonate. On days 4 and 20 postpartum, 15-mL samples of milk were collected at the end of breastfeeding. The day 4 milk was collected using a pump (Symphony, Medela), and the day 20 milk either with a pump or by direct expression from the breast. To prevent contamination with MOSH, women were instructed to avoid contact with their hands and to refrain from using breast creams shortly before collecting the sample. Storage vials for fat and milk samples, the pump, and surgeons' gloves were tested to ensure the absence of MOSH.

Tissue and milk fat specimens were analyzed by on-line normal phase high-performance liquid chromatography gas chromatography flame ionization detection as described in our previous manuscript.[1] Analytical measurement uncertainty varied between 15% and 25%.

Questionnaire

One day before the planned cesarean section all women completed a questionnaire on personal data, nutrition habits, and cosmetics use. Women were asked about their consumption of particular food items in their overall habitual diet. Food items addressed were meat, fish, fast food, and convenience food. Information requested was the frequency of consumption per week. The use of cosmetics in the present pregnancy and their use in daily life before pregnancy were addressed separately. Additional questions regarding the amount of milk produced, occurrence of breast inflammation, and use of breast and nipple salves were asked when milk samples were collected on days 4 and 20 after birth. On day 20 information was generated by telephone interview.

Statistical analysis

Because the distribution of MOSH concentrations was non-Gaussian, the Spearman rank test was used for correlations with personal data, nutrition habits, and use of cosmetics. Relationships between two categorical variables were assessed using the chi-square test. All variables significantly related to

MOSH concentrations in univariate analyses ($n=12$) were included in a linear regression analysis. Only variables with a p value <0.15 were kept in the final multiple linear regression analysis ($n=7$). Because multivariate analysis calls for Gaussian distribution of the dependent variable, log-transformed MOSH concentrations were used. Although several subgroup analyses of MOSH in human body fat were performed, no corrections for multiple comparisons were applied due to the explorative character of these analyses. p values <0.05 were considered statistically significant.

Results

Information derived from the questionnaire

Personal data are summarized in Table 1. Information on nutrition habits and cosmetics use is given in Table 2. When sampling milk on days 4 and 20 after childbirth women were asked about their use of nipple creams and anti-inflammatory breast creams (e.g., to treat mastitis; included in Table 2).

Correlations with MOSH concentrations in fat tissue

Age and body mass index (BMI) prior to the pregnancy was significantly correlated with MOSH concentration in fat tissue, showing a higher concentration with increasing age and lower BMI (Spearman correlation coefficient $r_s=0.365$, $p<0.001$; Spearman correlation coefficient $r_s=-0.283$, $p=0.001$, respectively, see Table 3). The same was true for bra cup size (cup sizes A and B versus C, D, and E; Spearman correlation coefficient $r_s=-0.202$, $p=0.017$).

Women with their main residence (where the women spent the vast majority of their life) in Austria had a significantly higher MOSH concentration in fat tissue than did women from other countries (Spearman correlation coefficient $r_s=0.172$, $p=0.041$). Austrians had significantly higher concentrations than did Turks (Spearman correlation coefficient $r_s=-0.259$, $p=0.007$) or Germans (Spearman correlation coefficient $r_s=-0.259$, $p=0.007$).

Women with university admission qualification showed significantly higher MOSH concentrations in their fat tissue than did women with a lower education level (Spearman correlation coefficient $r_s=-0.273$, $p=0.001$).

Women who smoked during their present pregnancy had significantly lower paraffin concentrations in their fat tissue than did nonsmokers (Spearman correlation coefficient $r_s=-0.228$, $p=0.006$). The status of smoking before pregnancy did not correlate with MOSH concentration.

Women with two or more previous childbirths exhibited a significantly higher MOSH concentration in fat tissue than did those with only one previous childbirth (Spearman correlation coefficient $r_s=0.169$, $p=0.044$). Total breastfeeding time (sum of breastfeeding months following all previous childbirths) did not correlate with MOSH concentration in fat tissue.

A significant association was observed between MOSH concentrations and the use of sun cream during the present pregnancy (Spearman correlation coefficient $r_s=0.258$, $p=0.002$), as well as between MOSH concentrations and the use of body lotions (Spearman correlation coefficient $r_s=0.207$, $p=0.014$), hand and face creams (Spearman correlation coefficient $r_s=0.205$, $p=0.014$; Spearman correlation coefficient $r_s=0.241$, $p=0.004$, respectively), and lipsticks (Spearman correlation coefficient $r_s=0.174$, $p=0.038$) in daily life.

Table 1. Personal Data of the 142 Women Included in the Study

	No. of women[a]	*Median (range)*
Age	142	31 years (19–47)
Height	141	165 cm (147–180)
Weight		
Before the present pregnancy	141	62 kg (43–148)
Before the cesarean section	137	78 kg (56–155)
BMI	141	23 (17–52)
Bra cup size	138	
A	12 (9%)	
B	74 (54%)	
C	41 (30%)	
D	10 (7%)	
E	1 (<1%)	
Parity	142	
First pregnancy	59 (42%)	
1 previous childbirth	53 (37%)	
2 previous childbirths	20 (14%)	
3–5 previous childbirths	10 (7%)	
Breastfeeding of previous children	83	Total breast-feeding time[b]
No	15 (18%)	
Yes	68 (82%)	4 months (0.3-108)
Country of main residence[c]	142	
Austria	99 (70%)	
Germany	10 (7%)	
Turkey	11 (8%)	
Other[d]	22 (15%)	
Smoking status	142	
In the present pregnancy	24 (17%)	
Before the present pregnancy	84 (59%)	
Education level	142	
University admission qualification	52 (37%)	
No university admission qualification	90 (63%)	
Underlying diseases	142	
Metabolic disease[e]	16 (11%)	
Bowel disease (celiac disease)	1 (<1%)	

[a]Information was not available in all 142 women.
[b]Sum of breastfeeding months after all previous childbirths.
[c]The country where the women spent the vast majority of their life.
[d]Croatia ($n=1$), Bosnia ($n=3$), Serbia ($n=1$), Bulgaria ($n=1$), Switzerland ($n=1$), Italy ($n=2$), Spain ($n=1$), Hungary ($n=1$), England ($n=1$), Ireland ($n=1$), Egypt ($n=1$), Morocco ($n=1$), Russia ($n=1$), United States ($n=3$), Mexico ($n=1$), South-Africa ($n=1$), and the Philippines ($n=1$).
[e]Diabetes mellitus in pregnancy ($n=3$), hyperlipidemia ($n=1$), hyperthyroidism ($n=2$), hypothyroidism ($n=9$), and rheumatorid arthritis ($n=1$).

No correlation was found between MOSH concentration in fat tissue and reported nutrition habits (i.e. the amount of meat, fish, fast food, or convenience food consumed). Multivariate analysis showed age ($p<0.001$), BMI ($p=0.001$), country of main residence ($p=0.03$), number of previous childbirths ($p=0.029$), use of sun cream in the present pregnancy ($p=0.002$), and use of hand cream ($p=0.011$) and lipstick ($p=0.007$) in daily life to remain significant (Table 3). Overall,

Table 2. Use of Cosmetics and Nutrition Habits

	No. of women using distinct cosmetics/consuming distinct food	*Times per week: Median (range)*	*Duration in weeks: Median (range)*
Cosmetics use in the present pregnancy			
Creams to prevent striae	74/142 (52%)	7 (1–21)	28 (3–40)
Breast creams	7/142 (5%)	4 (1–14)	16 (2–40)
Creams for medical reasons[a]	20/142 (14%)	7 (2–18)	12 (3–40)
Sun creams	57/142 (40%)	n.e.[b]	
Cosmetics use in daily life			
Body lotions	99/142 (70%)	4 (1–21)	
Hand creams	95/142 (67%)	7 (1–42)	
Face creams	104/142 (73%)	7 (1–21)	
Lipstick or lip balm	105/142 (74%)	7 (1–84)	
Sun creams	101/142 (71%)	n.e.	
On day 4 after childbirth[c]			
Nipple creams	59/100 (59%)		
Anti-inflammatory breast creams	4/106 (4%)		
On day 20 after childbirth[c]			
Nipple creams	30/101 (30%)		
Anti-inflammatory breast creams	2/100 (2%)		
Nutrition habits			
Consumption of meat	140/142 (99%)	3 (0–10)	
Consumption of fish	129/142 (91%)	1 (0–4)	
Consumption of fast food	86/142 (61%)	0.25 (0–2)	
Consumption of convenience food	85/141 (60%)	0.5 (0–23)	

[a]Medical reasons for the application of creams in pregnancy: varicosities ($n=4$), itching ($n=3$), dermatosis in pregnancy ($n=3$), psoriasis ($n=3$), vulvovaginal candidiasis ($n=2$), dry skin ($n=1$), eczema ($n=1$), herpes labialis ($n=1$), hemorrhoids ($n=1$), burn ($n=1$).
[b]n.e., not evaluated.
[c]Information was not available in all 108 breastfeeding women.

the multiple linear regression model explained 37% of the variation in fat tissue MOSH concentrations ($R^2=0.373$). Results of univariate and multivariate analyses are given in Table 3.

Correlations between MOSH concentrations in milk and fat tissue

Milk samples from day 4 postpartum were available from 107 of 142 (75.4%) women; 34 had stopped breastfeeding and one had too little milk to provide a sample. On day 20 postpartum, milk samples were collected from 71 of 142 (50%) women.

A higher MOSH concentration in fat tissue (median 52.5 mg/kg) correlated with a higher concentration in milk fat on day 4 (median 30 mg/kg; Spearman correlation coefficient $r_s=0.476$, $p<0.001$; Fig. 1) as well as on day 20 (median 10 mg/kg; Spearman correlation coefficient $r_s=0.261$, $p=0.028$). MOSH concentrations in milk fat rapidly decreased between day 4 and day 20.

Table 3. Significant Predictors of Mineral Oil Saturated Hydrocarbons Concentration in Tissue Fat From Univariate Analysis and From the Multiple Regression Analysis

	Univariate analysis		*Multivariate analysis*	
	Spearman r	p *value*	*Standardized coefficient*[a]	p *value*
Age	0.365	< 0.001	0.375	< 0.001
BMI	− 0.283	0.001	− 0.233	0.001
Cup bra size (A, B vs. C, D, E)	− 0.202	0.017		
Country of main residence (Austria vs. others)	0.172	0.041	0.155	0.03
Education level (university admission qualification vs. other)	− 0.273	0.001		
Smoking in the present pregnancy (yes vs. no)	− 0.228	0.006		
Number of previous childbirths (> 2 vs. ≤ 2)	0.169	0.044	0.159	0.029
Cosmetics in the present pregnancy (yes vs. no)				
Sun cream	0.258	0.002	0.218	0.002
Cosmetics in daily life (yes vs. no)				
Body lotion	0.207	0.014		
Hand cream	0.205	0.014	0.183	0.011
Face cream	0.241	0.004	0.195	
Lipstick	0.174	0.038		0.007

[a]The standardized coefficient shows the relative importance of the variables for the prediction of fat tissue MOSH concentrations. MOSH, mineral oil saturated hydrocarbons.

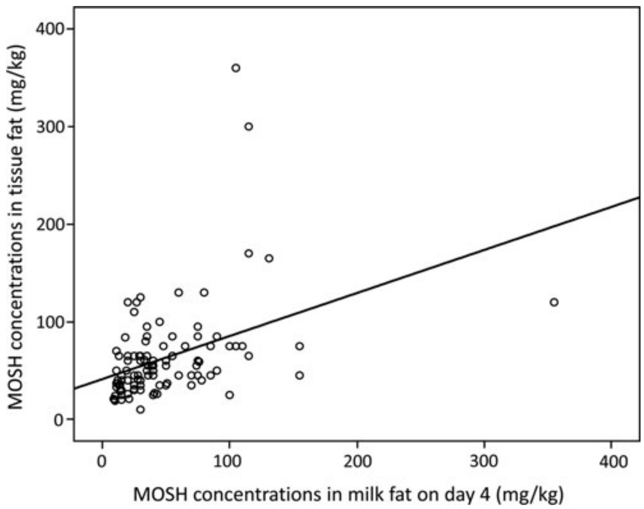

FIG. 1. Scatterplot of 107 women showing strong correlations between MOSH concentration in the fat tissue and the corresponding milk sample on day 4 postpartum. Each dot represents one specific women (in whom both was available, fat tissue and a milk sample on day 4). A Spearman correlation coefficient (r) with values close to +1 indicates a perfect linear correlation.

For 5 of 142 (3.5%) women the MOSH concentration in day 4 milk fat exceeded that in the women's fat tissue by more than twofold. Moreover, for 6 of 71 (8.5%) women the MOSH concentration in day 20 milk fat exceeded that in day 4 milk fat by more than twofold. As shown previously, in at least some of these cases the increase was probably related to contamination from an external source such as breast salves.[1]

On day 4 postpartum, women were asked for their subjective estimation of the amount of milk they produced. The milk fat of women who estimated their amount of milk as little (little versus medium, much, and very much milk) showed a significantly higher MOSH concentration (Spearman correlation coefficient $r_s = -0.354$, $p < 0.001$).

Discussion

The aim of the present study was to identify the major sources of mineral oil contamination in the human body by means of a statistical correlation of anamnestic data derived from a questionnaire and the MOSH concentrations found in human fat tissue and milk fat.

General correlations

The increase seen in MOSH concentration in human fat tissue with age suggests an accumulation over time. The amount of body fat (BMI and bra cup size) was negatively correlated with the MOSH concentration in fat tissue, suggesting that a given exposure is diluted by more fat. This assumes a homogeneous distribution throughout the body's fat, which so far has not been confirmed. Transport within the body is assumed to be slow, which would explain why MOSH concentrations in milk fat strongly decreased between days 4 and 20. The hydrocarbons stored in the breast are mobilized during lactation and depleted in a short time, with reallocation from the body being slow.

Dietary factors

No statistically significant correlation with nutritional habits was seen. This should not be construed as an argument against food being a principal source of contamination, however, since mineral oils are present in many foods and concentrations can vary strongly from one product to another of the same type.

The absorption of mineral oil through the gastrointestinal tract is one of the proven routes for MOSH to enter the body.[16,23] In a rat feeding study 1.5% of the orally administered dose of mineral oil was absorbed, and merely 0.1% of the initial dose remained in the body after 3 weeks.[23] Grob et al.[24] showed that concentrations in egg yolk only reached 1.5% to 3% of the concentration in animal feed.

Cosmetics

MOSH concentrations were significantly correlated with the use of particular cosmetics, although these correlations were weak (Spearman correlation coefficients <0.3). However, even in multivariate analyses the correlation remained significant for the use of sun cream in the present pregnancy and the use of hand cream and lipstick in daily life before pregnancy, suggesting that specific cosmetics could be a relevant source of MOSH accumulation in the human body. The scarce data available indicate that dermal absorption following topical application of mineral hydrocarbons is less than 1%.[25–28] *In vitro* human skin penetration studies with labeled n-pentadecane and n-undecane showed 0% and less than 0.01% absorption, respectively.[29] Microscopic studies suggest that penetration of hydrocarbons, such as octadecane, is limited to the stratum corneum.[30]

In contrast to these findings, Noti et al.[31] and our group[1] provided evidence that mineral hydrocarbons of higher molecular mass applied to the breast skin are transferred to the human milk. In the most drastic case, the MOSH concentration in the milk from a woman with breast inflammation and extensive treatment with anti-inflammatory ointments increased from 40 mg/kg on day 7 to 1300 mg/kg on day 20 after childbirth. The mineral hydrocarbons in the day 20 milk sample had a higher molecular weight (centered on the C33 n-alkane) than those in the day 7 milk sample (centered on the C24 n-alkane), reflecting the composition of the salves. Our previous paper reported several cases of MOSH concentrations in milk fat that significantly exceeded those in corresponding fat tissue, suggesting that salves or ointments had interfered.[1] In at least three of these cases the composition of the MOSH concentration clearly deviated from that normally observed. For the day 4 milk samples we can rule out contamination during sample collection because they were taken under supervision.

Inhalation of contaminated air

Another potential source of exposure to mineral oil is the atmosphere, particularly incompletely burned diesel fuel and heating oil, as well as lubricating oil emitted by diesel engines.[32] No data are available on the importance of this source, but the MOSH concentration in the body fat of animals (hens, pigs, and cattle), breathing the same air is lower by a factor of at least 50 (below 1 mg/kg, unless raised with contaminated feed).[24] This suggests that the contribution from air inhalation is small.

Other correlations

Interestingly, Austrian women and women with several children showed significantly higher MOSH concentrations in fat tissue, even in multivariate analysis. This could be due to indirect effects of their geographic and social background. Women with several children do not substantially reduce their MOSH contamination by breastfeeding, which is supported by the relatively small amount of MOSH fed to their babies, as compared to the total body burden.

Limitations

The study also shows the difficulty of using a questionnaire to investigate significant correlations in complex systems. First, it is difficult to categorize nutrition and cosmetics use habits, especially because these vary in different periods of life. Furthermore, MOSH concentrations in foods and cosmetics vary broadly from one product to another of the same type. For instance, a woman who applies a lot of cosmetics but exclusively uses products free of paraffin oils may not be exposed to MOSH from this source. A person consuming bread from one of the few producers using large amounts of paraffin oils as release agent, is not adequately recognized by a questionnaire as being particularly highly exposed. In addition, a possible measurement error needs to be considered when interpreting our data. Besides the analytical measurement uncertainty (below 25%) we cannot exclude natural variations in MOSH concentrations in fat tissue, since the invasive character of the probe collection did not allow sampling fat tissue at different locations.

Conclusions

Exposure to MOSH can take at least three pathways: oral (food, pharmaceuticals), dermal contact (cosmetics, technical oils), and air inhalation. Our statistical evaluation of the responses to a questionnaire did not provide a simple answer as to which source predominates.

Animal studies indicate that certain mineral oil hydrocarbons in food are transferred to the body fat, although selectively and at a low percentage. However, because this uptake is selective and most hydrocarbons are efficiently eliminated by the body, the average content of about 1 g in humans may not be explained by foods alone.[1] The present study provides statistical evidence that cosmetics are a relevant source. Even mineral hydrocarbons of high molecular mass seem to pass the dermal barrier and should therefore be avoided for dermal application—at least on the breasts of nursing mothers.

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Disclosure Statement

No competing financial interests exist.

References

1. Concin N, Hofstetter G, Plattner B, et al. Mineral oil paraffins in human body fat and milk. Food Chem Toxicol 2008;46:544–552.
2. Biedermann M, Grob K. Comprehensive two-dimensional GC after HPLC preseparation for the characterization of aromatic hydrocarbons of mineral oil origin in contaminated sunflower oil. J Sep Sci 2009;32:3726–3737.
3. Biedermann M, Grob K. How "white" was the mineral oil in the contaminated Ukrainian sunflower oils? Eur J Lipid Sci Technol 2009;111:313–319.
4. Grob K, Artho A, Biedermann M, Egli J. Food contamination by hydrocarbons from lubricating oils and release agents: determination by coupled LC-GC. Food Addit Contam 1991;8:437–446.
5. Wagner Ch, Neukom HP, Grob K, Moret S, Populin T, Conte LS. Mineral paraffins in vegetable oils and refinery by-products from animal feeds. Mitt Lebensm Hyg 2001; 92:499–514.
6. Moret S, Populin T, Conte L, Grob K, Neukom H. Occurrence of C15 – C45 mineral paraffins in olives and olive oils. Food Addit Contam 2003;5:417–426.
7. Grundböck F, Fiselier K, Schmid F, Grob K. Mineral oil in sunflower seeds: the sources. Eur Food Res Technol 2010; 231:209–213.
8. Fiorini D, Fiselier K, Biedermann M, Ballini R, Coni E, Grob K. Contamination of grape seed oil with mineral oil paraffins. J Agric Food Chem 2008;56:11245–11250.
9. Grob K, Huber M, Boderius U, Bronz M. Mineral oil material in canned foods. Food Addit Contam 1997;14:83–88.
10. Jickells S, Nichol J, Castle L. Migration of mineral hydrocarbons into foods. 5. miscellaneous applications of mineral hydrocarbons in food contact materials. Food Addit Contam 1994;11:333–341.
11. Biedermann M, Grob K. Is recycled newspaper suitable for food contact materials? Technical grade mineral oils from printing inks. Eur Food Res Technol 2010;230:785–796.
12. Lorenzini R, Fiselier K, Biedermann M, Barbanera M, Braschi I, Grob K. Saturated and aromatic mineral oil hydrocarbons from paperboard food packaging: estimation of long-term migration from contents in the paperboard and data on boxes from the market. Food Addit Contam A 2010;27:1765–1774.
13. Vollmer A, Biedermann M, Grundböck F, et al. Migration of mineral oil from printed paperboard into dry foods: survey of the German market. Eur Food Res Technol 2011;232: 175–182.
14. Codex Alimentarius Commission. Food additive details: Mineral oil, high viscosity (905d). Available at http://www.codexalimentarius.net/gsfaonline/additives/details.html?id = 292. (accessed in 2011).
15. deNavarre MG. Oil cosmetics. In: deNavarre MG, ed. The chemistry and manufacture of cosmetics. Vol. III. 2nd ed. Orlando, FL: Continental Press, 1975:323–325.
16. Low L, Shymanski P, Kommineni C, Naro P, Mackerer C. (1992). Oral absorption and pharmacokinetic studies of radiolabelled normal paraffinic, isoparaffinic and cycloparaffinic surrogates in white oil in Fischer 344 rats. Transcript of the Toxicology Forum, Special Meeting on Mineral Hydrocarbons, Oxford, 1992:86–101.
17. Baldwin MK, Berry PH, Esdaile DJ, et al. Feeding studies in rats with mineral hydrocarbon food grade white oils. Toxicol Pathol 1993;20(3 Part 1):426–435.
18. Firriolo J, Morris C, Trimmer G, Twitty L, Smith J, Freeman J. Comparative 90-day feeding study with low-viscosity white mineral oil in Fischer-344 and Sprague-Dawley-derived crl:cd rats. Toxicol Pathol 1995;23:26–33.
19. Smith J, Mallett A, Priston R, et al. Ninety-day feeding study in Fischer-344 rats of highly refined petroleum-derived food-grade white oils and waxes. Toxicol Pathol 1996;24(2):214–230.
20. Miller M, Lonardo E, Greer R, et al. Variable responses of species and strains to white mineral oils and paraffin waxes. Regul Toxicol Pharmacol 1996:23(1):55–68.

21. Griffis LC, Twerdok LE, Francke-Carroll S, et al. Comparative 90-day dietary study of paraffin wax in Fischer-344 and Sprague–Dawley rats. Food Chem Toxicol 2010;48:363–372.
22. Bundesinstitut für Risikobewertung. Übergänge von Mineralöl aus Verpackungsmaterialien auf Lebensmittel. Available at www.bfr.bund.de/cm/216/uebergaenge_von_mineraloel_aus_verpackungsmaterialien_auf_lebensmittel.pdf. (accessed in 2010).
23. Ebert A, Schleifer C, Hess S. Absorption, disposition, and excretion of 3H mineral oil in rats. J Pharm Sci 1966;55:923–929.
24. Grob K, Vass M, Biedermann M, Neukom H. Contamination of animal feed and food from animal origin with mineral oil hydrocarbons. Food Addit Contam 2001;18:1–10.
25. Rossmiller JD, Hoekstra WG, Hexadecane-induced hyperkeratinization of guinea pig skin. II. Arginase activity in normal and hexadecane-treated epidermis. J Invest Dermatol 1964;45:24–27.
26. Yang JJ, Roy TA, Neil W, Krueger AJ, Mackerer CR. Percutaneous and oral absorption of chlorinated paraffins in the rat. Toxicol Ind Health 1987;3:405–412.
27. Brown BE, Diembeck W, Hoppe U, Elias PM. Fate of topical hydrocarbons in the skin. J Soc of Cosmet Chem 1995;46:1–9.
28. Nash JF, Gettings SD, Diembeck W, Chudowski M, Kraus AL. A toxicological review of topical exposure to white mineral oils. Food Chem Toxicol 1996;34:213–225.
29. Scott RC. In vitro absorption of some chlorinated paraffins through human skin. Arch Toxicol 1989;63:425–426.
30. Zesch A, Bauer E. Quantitative aspects of the percutaneous uptake of wool wax alcohols (cetyl alcohol) and paraffins (octadecane) from different ointment bases. Derm Beruf Umwelt 1985;33:15–20.
31. Noti A, Grob K, Biedermann M, Deiss U, Brueschweiler B. Exposure of babies to C15 – C45 MOSH from human milk and breast salves. Regul Toxicol Pharmacol 2003;3:317–325.
32. Neukom H, Grob K, Biedermann M, Noti A. Food contamination by C20 – C50 MOSH from the atmosphere. Atmos Environ 2002;36:4839–4847.

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