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## The safety issue in aromatherapy

### Introduction

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Many aromatherapists and members of the public consider natural essential oils to be completely safe. This is based on the misconception that all herbs are safe – because they are ‘natural’. However, it is dangerous to assume, just because a tea or alcoholic extract of a plant used as a herbal medicine is harmless, that the essential oil derived from that plant is also safe. The dramatic increase in concentration of the essential oil compared with that in the whole plant (often the yield is 0.01%) demonstrates that essential oils are not equivalent to the whole herb. Essential oils are also volatile and fat-soluble and therefore differ from the mainly water-soluble whole herb extracts used in herbal medicine. As suggested in Chapter 1, the comparison is akin to massaging butter into the skin of a baby and believing that this is equivalent to giving the baby whole milk to drink.

The toxicity of essential oils can also be entirely different to that of the herb, not only because of their high concentration, but also because of their ability to pass across membranes very efficiently due to their lipophilicity.

Some aromatherapists believe that aromatherapy is self-correcting, unlike conventional therapy with medicines, and if errors are made in aromatherapy, they may be resolved through discontinuation of the wrongful application of the oil. There is also the belief that if an inflammation follows the use of an irritant oil, it will dissipate as soon as the oil is discontinued without having caused lasting damage. It is said that the occasional mistake is never injurious, but instead provides valuable guidance about how to correctly use the often underestimated power of essential oils (e.g. Schnaubelt, 1999).

This is a very dangerous view due to the considerable amount of evidence of the risks of essential oils.

Essential oil safety has been monitored in a variety of different ways, all of which have been geared to the perfumery, cosmetics and the food industries. The continuous synthesis of new aromachemicals and their widespread usage in ‘natural essential oils’ together with many diluents, has brought about many problems, the worst being sensitisation. The whole aspect of safety is now being stringently reviewed and new regulations may soon impede the sale and usage of many essential oils and cosmetic products as well as their use in foods.

The toxicity of essential oils does not entirely depend on high concentrations. All essential oils are toxic at very high doses, especially if taken orally. Many essential oils are inherently toxic at very low concentrations due to very toxic components: these are not normally used in aromatherapy (see Appendices 29 and 30). Many essential oils which are considered to be non-toxic can have a toxic effect on some people: this can be influenced by previous sensitisation to a given essential oil, a group of essential oils containing similar components or some adulterant in the essential oil. It can also be influenced by the age of the person: babies and young children are especially vulnerable and so are very old people (who are also more affected by drugs, etc.). The influence of other medicaments, both conventional and herbal, is still in the preliminary stages of being studied. It is possible that these medicaments, and also probably household products, including perfumes and cosmetics, can influence the adverse reactions to essential oils. Very small doses of essential oils taken/used over many months or years could have toxic effects, as shown by many recent studies on sensitisation.

Aromatherapists themselves have also been affected by sensitisation (Crawford *et al.*, 2004): in a 12-month period under study, prevalence of hand dermatitis in a sample of massage therapists was 15%

by self-reported criteria and 23% by a symptom-based method and included use of aromatherapy products in massage oils, lotions or creams. In contrast, the suggestion that aromatherapists have any adverse effects to long-term usage of essential oils was apparently disproved by a non-scientific survey, where adverse reactions to essential oils were blamed on reactions to the clients themselves (Price and Price, 1999). Most aromatherapists apparently experienced only beneficial effects both on the skin and other organs and tissues. This type of survey may be considered unscientific for reasons of bias of the respondents to the survey, notably because aromatherapists who had experienced adverse effects would have left the profession; secondly, most of the respondents had practised for under 4 years and had given fewer than ten treatments per week (as reported by Price and Price, 1999).

The International Organization for Standardization (ISO) has set up standards to make essential oils more consistent (see monographs), but this often encourages adulteration (see Chapter 5). The ISO stipulates that there is a named botanical source, but in commerce the actual plant source is often confused. For example, citrus plants can be grown as scions on a parent plant of a different species. Furthermore hybrids and cultivars are often used, as well as clones obtained by micro-propagation (e.g. tea tree).

### **General guidance for essential oil purchase and storage**

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Do not buy essential oils from market stalls – these cheap essential oils are often useful only for usage in burners and not for skin application. Many of the essential oils are mixed with considerable volumes of various diluents, which include petroleum spirits. Buy bottles with child-proof caps and efficient droppers. On the other hand do not assume that essential oils sold from high street stores are pure, unadulterated essential oils (see Chapter 5). All essential oils should be sold in brown bottles or platinum containers: do not buy them in clear glass or plastic containers.

Essential oils should always be stored in the refrigerator (preferably in an enclosed plastic container to prevent the odours mingling with stored foods) or in a cool, dark place. Storage areas must be out of reach for children. Do not expose the bottles to light or air for long periods, to prevent oxidation

of the components – as this may make them more toxic. Citrus essential oils are very unstable and may last for only a few months. Many already contain added antioxidants, but one can add vitamin E (squeezed from capsules) to the essential oils as a safe and efficient antioxidant; it also supposedly helps the skin to remain young and healthy.

### **Toxicity testing in animals**

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Most aromatherapy suppliers claim to have managed in some way to obtain essential oils, which ‘have never been tested on animals’, information which they pass on to their clientele. Nearly all the essential oils and extractives commonly used in aromatherapy have however been tested on animals and their monographs are to be found in the journal *Food and Cosmetics Toxicology* from 1973, renamed *Food and Chemical Toxicology* in 1982. This fact is not known by many aromatherapists, who, in their innocence, think they are using only essential oils that have not been tested on animals, sold to them by reputable dealers. This is not only erroneous, but it contravenes the Trades Description Act and also Health and Safety regulations, as only essential oils tested on animals are legally sold and used for foods, perfumes and cosmetics.

Apparently suppliers can get round the legislation using a loophole that involves the issue of certificates stating that ‘the essential oils have never been tested on animals if they have not been tested **in the last seven years**’. As most were tested from 1973 to 1992, this seems to be a good ploy by the suppliers. The results of more recent animal tests, published as monographs, include essential oil components and further genotoxicity, mutagenicity and pharmacological evaluations on both essential oils and components. Most cosmetic products are now no longer tested on animals, but all their ingredients have been tested.

As most essential oils were tested over 30 years ago, the toxicity data may now be meaningless, as different essential oils are now used, some of which contain different quantities of synthetic components. There is also the question as to whether all synthetic components are always made in the same way. If not, then there is the possibility of contamination with other chemicals, which changes the composition and

therefore the adverse effects, either making them worse or better.

The Living Flavour and Living Flower series (International Flavor & Fragrance Inc.) are produced by trapping the natural odours of the living plant using SPME (solid phase micro extraction) and then assembling them using totally synthetic components. Synthetic products could perhaps account for the increased toxicity of the essential oils bought today, especially in the area of sensitisation.

Published monograph data usually include: LD<sub>50</sub> (lethal dose for 50% of the test population) and acute symptoms after oral dosing in rats and dermal dosing in rabbits, subacute toxicity data after oral dosing, irritation studies usually after application on the backs of hairless mice or intact/abraded rabbit skin (Appendix 22). Sensitisation tests use a maximisation test on human volunteers at 1–8% in petrolatum, phototoxicity on hairless mice/swine and antimicrobial activity. On occasion, carcinogenicity and mutagenicity studies are included, together with other references as to the composition and bioactivities, including pharmacological and insecticidal studies and clinical trials, etc.

### Toxicity studies in animals: critique

The major drawbacks of trying to extrapolate toxicity studies in animals to humans concern feelings – from headaches to splitting migraines; feeling sick, vertigo, profound nausea; tinnitus; sadness, melancholia, suicidal thoughts; feelings of hate – which are clearly impossible to measure in animals.

The toxicity of an individual essential oil/component is also tested in isolation in animals and disregards the possibility of modification by other substances, including food components and food additive chemicals, the surrounding atmosphere with gaseous and other components, fragrances used in perfumes, domestic products, in the car, in public transport (including the people), workplace, etc. These could cause modification of the essential oil/component, its bioavailability and possibly the enhancement or loss of its function.

The detoxification processes in the body are all directed to the production of a more polar product(s), which can be excreted mainly by the kidneys regardless of whether this (these) are more toxic or less toxic than the initial substance. Any biotransformation in the body is affected by individual enzymes, which attack certain chemical groups. These include: aromatic,

acyclic and heterocyclic hydroxylation; *N*-, *S*- and *O*-dealkylation; *N*-oxidation and *S*-oxidation; amine oxidation, alcohol and aldehyde oxidation; *N*-hydroxylation; desulphuration and deamination. The process usually occurs through two phases: the primary phase involves these enzymatic biotransformations, the most important being microsomal oxidation using cytochrome P450; this is followed by the secondary phase, involving conjugation. There can be numerous biotransformations following the conjugations as well, giving rise to hundreds of metabolites: the main metabolite(s) vary in different animals, therefore extrapolation from animal to humans becomes difficult if the major metabolite(s) are entirely different. These major metabolites can be influenced by the presence of other components. The latter can also affect the biological half-life, and thereby its activity and accumulation in different tissues in the body.

### Dermal absorption and detoxification

Cutaneous enzymes include esterases and other enzymes, including oxidases using cytochrome P450. The activity of these enzymes in the skin is much lower than in the liver, but the large surface area of the skin makes it a significant detoxification process. Any chemicals absorbed will then be dealt with by the liver and other organs/tissues.

Absorption of essential oil components can be quite substantial and is influenced by numerous internal and external factors: idiosyncrasy; skin/air temperature, humidity, contact time and concentration, area and site of body as well as the physicochemical nature of each component. There is also the variability introduced by age, follicle number and skin surface status (e.g. undamaged, damaged, shaven, suntanned, protected by creams, etc.) (Hewitt *et al.*, 1993). The more lipophilic molecules are absorbed quickly, but also volatilise more readily; the more hydrophilic components may be very slow in penetrating, if at all, but are also influenced by the presence or absence of occlusion. Coumarin, present in cassia and other oils, is rapidly absorbed to 46% (human unoccluded), β-phenylethanol 64% (rat unoccluded), benzyl acetate 12% (human unoccluded), cinnamaldehyde to 24% (human unoccluded). Some components will accumulate to form a cutaneous reservoir pool (Hewitt *et al.*, 1993) in the lipid-rich stratum corneum. Others components permeate deeper into the skin to be

biotransformed by the P450 enzyme systems in the dermis and epidermis, and eventually this mixture of biotransformed and unchanged molecules reaches the systemic circulation via the dermal microvasculature.

### Inhalation: absorption and detoxification

Similar enzymes occur in the alveolar cells, modifying any chemicals absorbed through inhalation. There is almost a direct entry into the lung cells for lipophilic molecules in the essential oils as there is only one cell membrane thickness to traverse. This is why the effect of vaporisers or simply breathing in fragrances added to bath water can be substantial. Damage can occur to the lungs due to excessive use of certain chemicals in essential oils, but the actual concentration has not been worked out and very few studies are available (Cooper *et al.*, 1995). The risk of respiratory cancer in workers after 5 years of exposure to industrial terpenes from conifers is greatly increased (Kauppinen *et al.*, 1986). However, in another study, exposure to  $\alpha$ -pinene enantiomers for 20 minutes at 10–450 mg/m<sup>3</sup> did not cause acute changes in lung function (Falk *et al.*, 1990). Studies on the absorption of inhaled essential oil components are very rare, but one showed that 1,8-cineole was rapidly absorbed from eucalyptus essential oil, with plasma concentrations at their peak after 18 minutes (Jaeger *et al.*, 1996). The direct entry of lipophilic components from essential oils via the olfactory mucosa is quite substantial and they can act like anaesthetics very rapidly. Entry via the blood–brain barrier can also be substantial, especially in neonates and young children where it is undeveloped.

### GRAS status/NOELs

Most essential oils have GRAS (generally recognised as safe) status granted by the Flavor and Extract Manufacturers Association (FEMA) and approved by the US Food and Drug Administration (FDA) for food use, and many appear in the Food Chemical Codex. This was reviewed in 1996 after evaluation by the Expert Panel of the FEMA. The assessment was based on data of exposure, and as most flavour ingredients are used at less than 100 ppm, predictions

regarding their safety can be assessed from data on their structurally related group(s) (Munro *et al.*, 1996). The NOELs (no-observed-adverse-effect levels) are more than 100 000 times their exposure levels from use as flavour ingredients (Adams *et al.*, 1996). Critical to GRAS assessment are data of metabolic fate and chronic studies rather than acute toxicity. Most essential oils and components have an LD<sub>50</sub> of 1–20 g/kg body weight or roughly 1–20 mL/kg, with a few exceptions as follows:

Boldo leaf oil	0.1/0.9 (oral/dermal)
Calamus	0.8–9/5
Chenopodium	0.2/0.4
Pennyroyal	0.4/4
Savory (summer)	1.4/0.3
Thuja	0.8/4

Teratogenicity studies are infrequent and often deceptive, as they often involve the study of unusual species of plant essential oils. For example, *Salvia lavandulifolia* Vahl or Spanish sage, containing 50% of sabinyl acetate, injected s.c. during pregnancy with 15, 45 and 135 mg/kg essential oil (Pages *et al.*, 1992; see monograph) showed an abortifacient effect, no fetal toxicity but significant maternal toxicity. This amount of sabinyl acetate was similar to that found in *Juniperus sabina* and *Plectranthus fruticosus*, which had a teratogenic effect (neither of these are frequently used, especially in aromatherapy).

Reproductive organ and hormone studies have shown that there are several xenoendocrine disrupters *in vitro* on male reproductive systems; citral has caused enlargement of the prostate gland in animal models and has oestrogenic effects (Nogueira *et al.*, 1995); several fragrances are carcinogenic (e.g. methyl eugenol in mice), whilst others are possible carcinogens (Burkey *et al.*, 2000).

### Poisonous chemicals

The National Institute of Occupational Safety and Health (1989) recognised 884 poisonous substances (many synthetics from petrochemicals) from 2983 chemicals used in the fragrance industry. Of these, many cause cancer, birth defects, CNS disorders,

allergic respiratory reactions, skin and eye irritation. The Research Institute for Fragrance Materials (RIFM) tests the safety of fragrance materials, but only about 1500 of more than 5000 materials used in fragrances have been tested. This is in contrast to their statement that: 'Over the approximately 30 years since its inception, RIFM has tested virtually all important fragrance materials in common use but it has always been the policy of RIFM that if a material is used by only one company, it is that company's responsibility to see that the material is adequately tested and evaluated' (Frosch *et al.*, 1998). However, patented chemicals are not tested until the patent expires, which may be after 17 years.

The testing done by the RIFM is generally limited to acute oral and dermal toxicity, irritation and dermal sensitisation, and phototoxicity. Testing is limited to individual materials and there is little effort to address synergistic and modifying effects of materials in combination, though the RIFM is aware that they occur. Materials used in combinations often have synergistic and modifying effects and more positive sensitisation reactions occur than when the materials are tested individually (Johansen *et al.*, 1998).

Most chemical data sheets and Material Safety Data Sheet (MSDS) information on fragrance materials clearly state that the chemical, physical and toxicological properties have not been thoroughly investigated. Many materials that were widely used for decades in the past had severe neurotoxic properties and accumulated in body tissues (Spencer *et al.*, 1979; Furuhashi *et al.*, 1994). In spite of this, most fragrance materials have never been tested for neurological effects, despite the fact that olfactory pathways provide a direct route to the brain (Hastings *et al.*, 1991).

## Toxicity in humans

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### Dermatitis and sensitisation

A recent clinical review of the adverse reactions to fragrances has been published (de Groot and Frosch, 1997) and many examples of cutaneous reactions to essential oils have been reported elsewhere (Guin, 1982, 1995). In the USA about six million people have a skin allergy to fragrance. Many of these people reported that this has a major impact on their quality

of life. Symptoms include headaches, dizziness, nausea, fatigue, shortness of breath and difficulty concentrating. Fragrance materials are readily absorbed into the body via the respiratory system and once absorbed cause systemic effects. Migraine headaches are frequently triggered by fragrances. Fragrances are known to modify cerebral blood flow and several common fragrance materials are known to have potent sedative effects via inhalation (Buchbauer *et al.*, 1993a). Recent studies in the US by the Institute of Medicine sponsored by the Environmental Protection Agency (EPA) suggest that fragrance materials can act on the same receptors in the brain as alcohol and tobacco, altering mood and function.

### Effects on asthmatics

Perfumes and fragrances are recognised as triggers for asthma by the American Lung Association and several other organisations concerned with respiratory health. The vast majority of materials used in fragrances are respiratory irritants and there are a few that are known to be respiratory sensitisers. Most have not been evaluated for their effects on the lungs and the respiratory system.

Respiratory irritants are known to make the airways more susceptible to injury and allergens, as well as to trigger and exacerbate such conditions as asthma, allergies, sinus problems and other respiratory disorders. In view of the recently recorded increase in asthma and other respiratory disorders, reduction in exposures to irritants is essential. In addition, there are a subset of asthmatics that are specifically triggered by fragrances (Shim and Williams, 1986; Bell *et al.*, 1993; Baldwin *et al.*, 1999), which suggests that fragrances not only trigger asthma, they may also cause it in some cases (Millqvist and Lowhagen, 1996). Placebo-controlled studies using perfumes to challenge people with asthma-like symptoms showed that asthma could be elicited with perfumes without the presence of bronchial obstruction and these were not transmitted by the olfactory nerve as the patients were unaware of the smell (Millqvist and Lowhagen, 1996).

People who are sensitive to fragrance often experience great difficulty in obtaining fragrance-free home and personal care products, and suffer health effects as a result of using scented products. Products labelled 'unscented' or 'hypoallergenic' that actually contain fragrance materials are particularly problematical

(HEAL, 2005). Several fragrance chemicals affect the immune response of the skin when inhaled, but the systemic and long-term effects of most fragrance materials are not known.

Adverse reactions to fragrances are difficult or even impossible to link to a particular chemical – often due to secrecy rules of the cosmetic/perfumery companies and the enormous range of synthetic components, constituting about 90% of flavour and fragrance ingredients (Larsen, 1998). The same chemicals are used in foods and cosmetics – there is therefore a greater impact due to the three different modes of entry: oral, inhalation and skin.

### Increase in allergic contact dermatitis in recent years

A study of 1600 adults in 1987 showed that 12% reacted adversely to cosmetics and toiletries, 4.3% of which were used for their odour (i.e. they contained high levels of fragrances). Respiratory problems worsened with prolonged fragrance exposure (e.g. at cosmetic/perfumery counters) and even in churches. In another study, 32% of the women tested had adverse reactions and 80% of these had positive skin tests for fragrances (deGroot and Frosch, 1987). Problems with essential oils have also been increasing. For example, contact dermatitis and allergic contact dermatitis caused by tea tree oil has been reported, which was previously considered to be safe (Carson and Riley, 1995a). It is unclear whether eucalyptol was responsible for the allergenic response (Southwell, 1997); out of seven patients sensitised to tea tree oil, six reacted to limonene, five to  $\alpha$ -terpinene and aromadendrene, two to terpinen-4-ol and one to *p*-cymene and  $\alpha$ -phellandrene (Knight and Hausen, 1994).

Many studies on allergic contact dermatitis (ACD) have been done in different parts of the world (deGroot and Frosch, 1987):

- Japan (Sugiura *et al.*, 2000): the patch test with lavender oil was found to be positive in increased numbers and above that of other essential oils in 10 years.
- Denmark (Johansen *et al.*, 2000): there was an 11% increase to the patch test in the last year and of 1537 patients, 29% were allergic to scented.
- Hungary (Katona and Egyud, 2001): increased sensitivity to balsams and fragrances was noted.

- Switzerland (Kohl *et al.*, 2002): ACD incidence has increased over the years and recently 36% of 819 patch tests were positive to cosmetics.
- Belgium (Kohl *et al.*, 2002): increased incidence of ACD has been noted.

Occupational increases have also been observed. For example, two aromatherapists were reported to have developed ACD: one to citrus, neroli, lavender, frankincense and rosewood and the other to geraniol, ylang ylang and angelica (Keane *et al.*, 2000).

Allergic air-borne contact dermatitis from the essential oils used in aromatherapy was also reported (Schaller and Korting, 1995). Allergic contact dermatitis occurred in an aromatherapist due to French marigold essential oil, *Tagetes* (Bilsland and Strong, 1990). A physiotherapist developed ACD to eugenol, cloves and cinnamon (Sanchez-Perez and Garcia Diez, 1999).

There is also the growing problem that patients with eczema are frequently treated by aromatherapists using massage with essential oils. A possible allergic response to a variety of essential oils was found in children with atopic eczema, who were massaged with or without the oils. At first both massages proved beneficial, though not significantly different; but on re-applying the essential oil massage after a month's break, there was a notable adverse effect on the eczema, which could suggest sensitisation (Anderson *et al.*, 2000).

### Photosensitisers

Berlocque dermatitis is frequently caused by bergamot or other citrus oil applications on the skin (often due to their inclusion in eau de Cologne) followed by exposure to UV light. This effect is caused by psoralens or furanocoumarins (Klarmann, 1958). Citrus essential oils labelled furanocoumarin-free (FCF) have no phototoxic effect, but are suspected carcinogens (Young *et al.*, 1990). Other phototoxic essential oils include yarrow and angelica, neroli, petitgrain, cedarwood, rosemary, cassia, calamus, cade, eucalyptus (species not stated), orange, anise, bay, bitter almond, ylang ylang, carrot seed and linaloe (the latter probably due to linalool, which, like citronellol, has a sensitising methylene group exposed) (Guin, 1995). Photosensitiser oils include cumint, rue, dill, sandalwood, lemon (oil and expressed), lime (oil and expressed), opopanax

and verbena (the latter being frequently adulterated) (Klarmann, 1958). Even celery soup eaten before UV irradiation has been known to cause severe sunburn (Boffa *et al.*, 1996).

Many of these photosensitisers are now banned or restricted. New International Fragrance Research Association (IFRA) proposals for some phototoxic essential oils include: rue oil to be 0.15% maximum in consumer products, marigold oil and absolute to be 0.01% and petitgrain mandarin oil to be 0.165%.

### Commonest allergenic essential oils and components

The most common fragrance components causing allergy are: cinnamic alcohol, hydroxycitronellal, musk ambrette, isoeugenol and geraniol (Scheinman, 1996). These are included in the eight commonest markers used to check for allergic contact dermatitis, usually as a 2% mix. Other components considered allergenic are: benzyl salicylate, sandalwood oil, anisyl alcohol, benzyl alcohol and coumarin.

The IFRA and the Research Institute for Fragrance Materials (RIFM) have forbidden the use of several essential oils and components, including costus root oil, dihydrocoumarin, musk ambrette and balsam of Peru (Ford, 1991; see also Appendices 28 and 29). There is also a concentration limit imposed on the use of isoeugenol, cold-pressed lemon oil, bergamot oil, angelica root oil, cassia oil, cinnamic alcohol, hydroxycitronellal and oakmoss absolute. Cinnamic aldehyde, citral and carvone oxide can only be used with a quenching agent. Photosensitivity and phototoxicity occurs with some allergens such as musk ambrette and 6-methyl coumarin and has been removed from skin care products. Children were often found to be sensitive to Peru balsam, probably due to the use of baby-care products containing this (e.g. talcum powder used on nappy rash).

As fragrances and foods contain essential oils and components, it is not surprising that fragrance materials have been found to interact with food flavourings. This is of increasing concern. For example, a 'balsam of Peru-free diet' has been devised in cases where cross-reactions are known to occur (Veien *et al.*, 1985). 'Newer' sensitisers include ylang ylang (Romaguera and Vilplana, 2000), sandalwood oil (Sharma *et al.*, 1987) (caution should be considered in accepting this as so much of this essential oil is

adulterated or completely synthetic), Lyrall (Frosch *et al.*, 1999; Hendriks *et al.*, 1999) and eucalyptol (Vilaplana and Romaguera, 2000).

Some sensitisers have been shown to interact with other molecules. For example, cinnamaldehyde interacts with proteins (Weibel *et al.*, 1989), which indicates how the immunogenicity occurs.

The international authorities are not satisfied that the cosmetics industry has been vigilant enough in their protection of the public, hence the proposed new EC legislation (7th Amendment), to label cosmetics/perfumes containing sensitisers and reduce or ban them altogether (see Appendices 27–29).

### Synthetic musks: a special problem

There have been very few published reports on neurotoxic aromachemicals such as musk ambrette (Spencer *et al.*, 1984), although many synthetic musks took over as perfume ingredients when public opinion turned against the exploitation of animal products. Musk ambrette was found to have neurotoxic properties in orally fed mice in 1967. However, it was in 1985, after studies were again published on its neurotoxic effects, that it was also realised that musk ambrette was readily absorbed through the skin. The IFRA then recommended that musk ambrette should not be used in direct skin contact products, even though it had been used since before the 1920s. In 1991, the FDA still found musk ambrette in skin contact products, proving that the recommendations by the IFRA are not binding.

A similar story occurred with acetyethyltetramethyltetralin (AETT), another synthetic musk, also known as versalide, patented in the early 1950s. During routine tests for irritancy in 1975, it was noted that with repeated applications the skin of the mice turned bluish and they exhibited signs of neurotoxicity. On further application, the internal organs also turned blue and there was severe neurological damage. The myelin sheath was damaged irreversibly in a manner similar to that which occurs with multiple sclerosis. In spite of legitimate concerns, the industry does not demand testing for the neurological and respiratory effects of fragrance materials.

Musk xylene, one of the commonest fragrance materials, is found in blood samples from the general population (Kafferlein *et al.*, 1998) and bound to human haemoglobin (Riedel *et al.*, 1999). Nitro- and

non-nitrobenzenoid musk compounds are also found in human adipose tissue (Riedel *et al.*, 1999) and nitro musk metabolites are found in human breast milk (Liebel and Ehrenstorfer, 1993). These musk products have been found to have an effect on the life stages of experimental animals such as the frog, *Xenopus laevis*, and the zebra-fish, *Danio rerio* (Chou and Dietrich, 1999) and the rat (Christian *et al.*, 1999). The effects on animal development have been extended to studies on reproduction and fertility, including hyperplasia of the prostate and testicular effects (Ford *et al.*, 1990; Api *et al.*, 1996). The hepatotoxic effect of musks is under constant study (Steinberg *et al.*, 1999).

### Toxicity in young children: a special case

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It is clear that there are severe dangers associated with the bad or ill-informed advice given by many aromatherapy books about the treatment of babies and children. For example, one book recommends giving 5–10 drops of ‘chamomile oil’ three times a day in a little warmed milk to their babies to treat colic. As there is no indication as to which of the three commercially available chamomile oils is to be used and because, depending on the dropper size, the dose could easily approach the oral LD<sub>50</sub> for the English and German chamomile oils, this could result in a fatality. In the same publication ‘syrup of elderflower and peppermint’ was recommended for ‘fever’. The peppermint could possibly be given by mothers in the form of peppermint oil, which has been known to kill a week-old baby (Evening Standard, 1998).

Dosages given in terms of drops can vary widely according to the size of the dropper in an essential oil bottle (see Appendices 9 and 10) and dilutions for massage also vary widely from author to author (e.g. 4–6 drops in 10 mL carrier oil; 1 drop for every 20 mL of vegetable oil). This could make a considerable difference to the toxicity regarding children, especially babies.

### Children’s cosmetics and toys

Many ‘cosmetics’ designed for use by children contain fragrance allergens (Rastogi *et al.*, 1999). In Denmark,

samples of children’s cosmetics were found to contain geraniol, hydroxycitronellol, isoeugenol and cinnamic alcohol (Rastogi *et al.*, 1999). Children are more susceptible than adults to any chemical, so the increase in childhood asthma reported in recent years could be caused by fragrance components in fast foods (whose consumption is escalating). There is also an increase in fragrance chemicals in everyday products from air-fresheners, soaps, cosmetics, bathroom products, ‘new-car smells’, all of which may interact.

### Selected toxicities of certain essential oils and their components

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#### Limonene

This is a common industrial cleaner and is also the main citrus oil component, the latter being often used in aromatherapy in pregnancy and childbirth. D-Limonene is used for degreasing metal before industrial painting; it oxidises to R-(–)-carvone, *cis*- and *trans*-isomers of limonene oxide. D-Limonene causes allergic contact dermatitis, particularly when aged (Chang *et al.*, 1997). In one series of studies, 2% of car mechanics with eczema on their hands tested positive to oxidised D-limonene, as did 2% of dermatitis patients (Karlberg *et al.*, 1994a,b).

Allergic contact dermatitis was noted in a histopathology laboratory technician using Parasolve (containing D-limonene) instead of xylene (Wakelin *et al.*, 1998). Pulmonary exposure of human volunteers to D-limonene caused a decrease in the lung vital capacity at highest doses (Falk-Filipsson *et al.*, 1993). The major volatile component of lactating mothers’ milk in the USA was found to contain D-limonene and the component is used as a potential skin penetration promoter for drugs such as indometacin, especially when mixed with ethanol (Falk-Filipsson *et al.*, 1993). Lastly, cats and dogs are very susceptible to insecticides and baths containing D-limonene giving rise to neurological symptoms including ataxia, stiffness, apparent severe CNS depression, tremors, coma (von Burg, 1995; see also Beasley, 1999).

In contrast to all the toxicity, D-limonene was shown to have anticarcinogenic properties *in vivo* when applied subcutaneously to mice which were then injected with benzopentaphene. Although the lung tumours took longer to develop and therefore the



animals lived longer, it did not prevent the cancer from forming in the first place (Homburger *et al.*, 1971).

## Linalool

It has been established that linalool hydroperoxide increases as the linalool decays and the rate of chemical decay is rapid compared with the potential shelf-life and age of oils used in aromatherapy. When linalool was oxidised for just 10 weeks the linalool content fell to 80% and the remaining 20% consisted of a range of breakdown chemicals including linalool hydroperoxide, which was confirmed as a sensitising agent. The fresh linalool was not a sensitiser, therefore the EC regulations which are warning about sensitisation potential are looking for potential harm even on storage (Skoeld *et al.*, 2002).

The sensitising activity of linalool was assessed using a commercial grade of 97% purity, which included linalool hydroperoxide. The sample of commercial linalool was then purified and the only sensitisation reaction was obtained when it was used at 100%; a dramatic reduction in sensitisation occurred when the linalool was 98.6% pure and the dihydrolinalool was below 1.4%. It was suggested that some of the other oxidation chemicals may also have sensitisation potential apart from linalool hydroperoxide (Basketter *et al.*, 2002).

## Safrole

This is the main component of sassafras oil and sweet basil and was used previously in perfumes and food: it is now limited to 1 mg/kg in foods (Council Directive 92/109/EEC, 14 December 1992), except in foods containing mace, nutmeg (15 mg/kg) and alcoholic drinks containing more than 25% of alcohol by volume (5 mg/kg) and other alcoholic drinks (2 mg/kg). Safrole and isosafrole were first studied in 1885 by Heffer, and numerous further studies reported their effects on tumorigenesis in animals and humans (Chen *et al.*, 1999) and the mode of action (Luo and Guenther, 1996). Safrole and sassafras oil are controlled under the Controlled Drugs Regulations (1993) (Controlled Drugs with subsequent European Directives 3677/90 as amended by Council Regulation 900/92 and Regulation (EC) No 273/2004 of the European Parliament and of the Council of

11 February 2004 on drug precursors) and are listed as category 1 substances, as they are precursors for illicit manufacture of hallucinogenic, narcotic and psychotropic drugs (e.g. ecstasy).

## Thujone

In large quantities thujone can cause convulsions associated with lesions of the cerebral cortex (Keith and Starraky, 1935; Opper, 1939) but thujone is permitted at 0.5 mg/kg in foods as  $\alpha$  or  $\beta$  or combination (Council Directive, 88/388/EEC, of 21 June 1988) and at 35 mg/kg in bitters.

## Wormwood

Wormwood essential oil, obtained via the Internet by a young man who thought it was equivalent to absinthe, caused acute renal failure and rhabdomyolysis when just 10 mL was drunk (Weisboro *et al.*, 1997). In another incident a 2-year-old child survived after drinking 15 mL of the oil. Absinthe was drunk by Van Gogh, together with camphor and turpentine (Bonkovsky *et al.*, 1992), which may explain his style of painting!

## Abortifacient and teratogenic oils

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### Apiol

When apiol (in dill) was studied in animals (Patoir *et al.*, 1936) it was found that the largest doses (not stated) gave rise to bleeding and hepatonephritis as well as abortion (i.e. it was an overall poison). A woman with fatal acute haemolytic anaemia, thrombocytopenic purpura, nephrosis and hepatitis was found to have taken a high dose of a compound containing apiol for amenorrhoea for three months. Other overdose cases of apiol resulted in CNS effects (Lowenstein and Ballew, 1958).

### Pennyroyal oil

A large dose of pennyroyal oil taken by a woman in the USA to induce an abortion proved fatal (Gold and

Cates, 1980). Other cases of toxicity of pennyroyal to women who tried unsuccessfully to induce abortion have been described (Tisserand and Balacs, 1995).

### Camphorated oil

Taken by mistake instead of castor oil during pregnancy, camphorated oil resulted in one fatality of the baby at birth out of four cases (Weiss and Catalano, 1973). Several cases of accidental poisoning in children are also reported, causing excitation of the CNS resulting in delirium and convulsions followed by depression including uncoordination and coma. Camphor oil was also reported to cause intoxication in 500 people in one year in the USA (Tisserand and Balacs, 1995).

### Nutmeg

Nutmeg intoxication during pregnancy (1 tablespoonful of grated nutmeg instead of 1/8th teaspoon in cookies) resulted in acute anticholinergic hyperstimulation, i.e. palpitations, agitation and blurred vision (Lavy, 1987). Treated with morphine and activated charcoal, the expectant mother was well after 24 hours and the baby was born later after slight pre-eclampsia symptoms. Nutmeg oil/grated seed can cause hallucinations and convulsions in large doses; myristicin itself was also shown to produce narcotic effects (Weil, 1965) and in large quantities, nutmeg and mace also showed these effects, which were comparable to alcohol intoxication.

### Antifertility oils

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#### $\beta$ -Myrcene

$\beta$ -Myrcene above 0.25 g/kg was found to be detrimental to the fertility and progeny number and development in the rat when given during pregnancy by gavage (Delgado *et al.*, 1993).

#### Phenol methyl ethers

Phenol methyl ethers, in particular anethole, found in fennel and anise are related to the oestrone and oestradiol methyl ethers; however, dill oil is inactive

(Zondek and Bergman, 1938; Albert-Puleo, 1980). The activity of anethole was much less than that of oestrone itself, and had a more profound effect on rats than mice, but there may be a remote danger of producing more oestrogens in the body by fennel. *trans*-Anethole has some minor oestrogenic properties (Zondek and Bergman, 1938).

### Genotoxic oils

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#### Dill, peppermint and pine

Genotoxicity of dill, peppermint and pine essential oils has been reported using chromosome aberration and sister-chromatid exchange tests in human lymphocytes *in vitro* and *Drosophila melanogaster* somatic mutation and recombination tests *in vivo* (Lazutka *et al.*, 2001). All these oils were cytotoxic for human lymphocytes. Other cytotoxic and/or genotoxic studies have recently been published on methyl eugenol (Burkey *et al.*, 2000), mint (Franzios *et al.*, 1997), camphor, 1,8-cineole, citral, citronellal, menthol and terpineol (Nogueira *et al.*, 1995; Gomes-Carneiro *et al.*, 1998), oregano essential oils (Karpouhtsis *et al.*, 1998), allyl benzene etheric oils estragole, basil and *trans*-anethole, D-limonene (Whysner and Williams, 1996). Negative evidence for *in vivo* DNA-damaging and mutagenic and chromosomal effects of eugenol were also shown (Maura *et al.*, 1989; Abraham, 2001).

### Neurotoxic oils

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#### Thuja, sage, cedar, hyssop

The toxicity of *Salvia officinalis*, thuja, *Arbor vitae* and cedar *Chaemocypris thyroides*, hyssop *Hyssopus officinalis* (containing pinocamphone and isopinocamphone), as well as thujone-containing Dalmatian sage, were investigated in 65 male and female rats intraperitoneally at progressively increasing doses; the components thujone and pinocamphone were also tested (Millet *et al.*, 1981; see also sage oil monograph). The animals were equipped with four skull electrodes for EEG and convulsions were elicited by all the oils, but varied according to the plant. For hyssop, the dose for no effect was 0.08 g/kg; convulsions were at

0.13 g/kg and it was lethal at 1.25 g/kg. For sage oil it was 0.3, 0.5 and 3.2 g/kg respectively. A daily repeated subclinical dose of hyssop oil (0.02 g/kg) for 15 days precipitated convulsions after just 5 days, but after cessation of injections, the rats returned to normal.

Convulsions were shown for thujone and pinocamphone at lower doses: thujone: at 0.2 g/kg and pinocamphone at 0.05 g/kg. This suggests that small doses every day for a short time could produce convulsions.

Eight cases of poisoning by the four essential oils showed a period of latency of a few minutes to 2 hours, the patients vomited and had convulsions, resembling epileptic fits, sometimes with cyanosis (Millet *et al.*, 1981). In six clinical cases the patients had ingested the oils for therapeutic purposes (e.g. 10 mL thuja oil to be 'in shape', 30 drops of hyssop oil for a common cold). Repetitive intake occurred in an asthmatic 6-year-old girl who had 2–3 drops of hyssop oil per day, but during a dyspnoeic crisis, she received half a teaspoonful of hyssop. Ten drops of hyssop oil was taken 'for flu' during two consecutive days; on the second day the convulsions appeared. A woman with facial acne took 20 drops of undiluted thuja at lunch and dinner for 5 days; after the tenth dose she got convulsions (Millet *et al.*, 1981).

Three case studies associated with the induction of epileptic seizures in normal people, including a child, were reported due to sage and other essential oils. One adult took 'a mouthful' of sage essential oil for hyperlipaemia over several years but after a larger dose she had tonic seizures and became unconscious for an hour, but recovered. Lastly, a baby given five long baths with addition of eucalyptus, pine and thyme over a 4-day period to cure her upper respiratory infection had a fit, followed by two more that day. She was hospitalised and the tests were normal, but she started to have multiple fits with a maximum of 133 in a day and was treated with phenobarbital and phenytoin. Her development was very depressed (Burkhard *et al.*, 1999). Eucalyptus and camphor have been reported to have an effect on the rat cerebral cortex (Steinmetz *et al.*, 1987).

### Absolutes and concretes

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Absolutes and concretes are potentially dangerous as they have not all been tested on the skin (Lis-Balchin,

1999a). Many solvent extracts are sensitisers (IFRA) and are usually used in lower percentages in all perfumes and cosmetic products. About 4% is the usual recommendation (ISA, 1993): except for neroli, lavender, hyacinth, benzoin (strong sensitiser), myrrh, olibanum at about 8%, also mastic although it is a strong sensitiser and causes irritation. Genet is used at 12% and vanilla at 10%. The absolutes and concretes of verbena are decreased to 2% and tobacco leaf to 1%. Some have not been tested for toxicity dermally, e.g. violet leaf, honeysuckle, orris, narcissus, mimosa, but are used at about 1–2% in perfumes.

Untested common essential oils include: catnip, chamomile (Maroc), *Eucalyptus* oils other than *E. globulus* and *E. citriodora*, *Imula graveolens*, kanuka (*Kunzea ericoides*), manuka (*Leptospermum scoparium*), melissa, naouli, *Ravensara aromatica* and other *Ravensara* species, spikenard, thyme chemotypes, valerian and yarrow.

### Phytols

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Herbal oils (real phytols or infused oils) are coming into fashion with certain aromatherapists. These include arnica (*Arnica montana*), calendula (*Calendula officinalis*, pot-marigold), centella (*Centella asiatica*, gotu kola or hydrocotyle), comfrey (*Symphytum officinalis*), Devil's claw (*Harpagophytum procumbens*), echinacea (*Echinacea purpurea*), fenugreek (*Trigonella foenum-graecum*), lime blossom (*Tilia* sp.), meadowsweet (*Filipendula ulmaria*) and St John's wort (*Hypericum perforatum*) (Lis-Balchin, 1999a). Most are tea-like or alcoholic extracts, not essential oils, and have no aroma.

All are well known as herbal remedies, usually taken internally or applied to burns or bruises, as poultices or compresses, but many are potentially toxic orally and are sensitisers (Newall *et al.*, 1996; Lis-Balchin, 1999a) and should be given only at the advice of a qualified herbalist – not an aromatherapist. There is no toxicological evaluation for their aromatherapeutic application and their possible dermal irritation or sensitisation is often unknown, therefore their use should be restricted in pregnancy (especially meadowsweet and St John's wort, which are both said to be uteroactive).

Phytol is also the name given to a particular solvent extraction technique, which does not involve the

usual benzene or hexane (now considered toxic if not potentially carcinogenic) to produce concretes, which are normally re-extracted with absolute alcohol to give absolutes (Wilde, 1994). The main object of this method was to be able to provide 'absolutes' without the use of alcohol for people who, for religious reasons, cannot drink alcohol or even use perfumes with alcohol. The actual 'phytols' are very similar to the absolutes and CO<sub>2</sub> extracts and none of them has been tested for toxicity, especially on the skin. These phytols contain similar 'plant impurities' to those of absolutes (i.e. the solvent-soluble pigments, alkaloids, etc.) which could cause sensitisation. They could however be useful as food additives, provided safety studies are done, or people accept them as being equivalent to absolutes or CO<sub>2</sub> extracts.

### Possible microbiological dangers of hydrolysates or hydrolysats

Many companies are now selling the by-products of essential oil distillation (e.g. rosewater) as alternative, gentle, aromatherapy products. As these products are meant to be pure and wholesome, no preservative is usually added. This means that microbial contamination is very likely, even before leaving the factory, let alone after several months of usage. Many aromatherapists recommend these for treating eye and other infections and spraying the rooms of asthmatics: this could have very serious effects due to possible bacterial contamination. Over a third of samples of bottled water tested contained *Cryptosporidium*, *Giardia* and other cysts (Rose *et al.*, 1993): the same level of contamination can happen to aromatherapy hydrolysates. As many of these waters are used for skin complaints, the addition of microbes could greatly exacerbate the condition – not alleviate it.

Hydrosols from unusual and toxicologically untested plants (e.g. verbenone-type rosemary, raven-sara and thyme chemotypes) should be avoided. Statements like: 'They are like homeopathic essential oils' are incorrect as their flower remedies have added alcohol as a preservative. It seems that to date the Food Safety Laws do not apply to hydrosols. This situation should be remedied as they are often drunk as well as applied to the skin. Insurance policies covering aromatherapy practices do not permit therapists to practise herbal medicine (i.e. to treat internally), especially using unknown products.

Some dangerous advice is given in aromatherapy books, such as swabbing down the whole of the lower body with flower water following parturition, including swabbing lightly over stitches and even leaving a clean swab in place. *Helichrysum italicum* and geranium (*Pelargonium graveolens*) waters have also been recommended for the care of open wounds. Other floral waters stated to be useful for wound healing included rose (*Rosa damascena*), myrtle (*Myrtus communis*) and rosemary borneol (*Rosmarinus officinalis* ct. borneol).

### Interactions between essential oils and conventional medicines or medical conditions

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There is growing evidence that adverse effects are often caused by mixing alternative and conventional therapies. This is shown by reports on herbal medicine interactions, including Chinese herbs (Lis-Balchin, 1999a). A report on the adverse action of a massage with wintergreen oil (containing 98% methyl salicylate) on a patient taking warfarin (given as an anti-coagulant), which caused haematomas, is a serious reminder of such dangers (Yeo *et al.*, 1994). Many of the cautions reported were for oral intake of herbal remedies, but where absorption of the active components occurs in large concentrations, essential oils must also come under the same risk category.

Some examples include cautions against the use, via any route, of cornmint or peppermint in cardiac fibrillation (Tisserand and Balacs, 1995) and against the use of annual wormwood, balsamite, camphor, ho leaf, hyssop, cotton lavender in people with epilepsy or patients with fever. All those examples, plus Indian dill, parsley leaf and seed, sage (Spanish) and savin, should not be used in pregnancy and caution is also given for *Lavandula stoechas*, oakmoss, rue and treemoss (Tisserand and Balacs, 1995).

Indian dill, parsley leaf and seed is cautioned against in people with kidney or liver disease and *Backhousia citriodora*, *Eucalyptus stagierana*, lemon-grass, may chang and melissa in prostatic hyperplasia (Tisserand and Balacs, 1995). Advice is also given to avoid certain essential oils via dermal administration in certain cases: basil, fennel, ho leaf (camphor/safrole) and nutmeg (East Indian) except at very low concentrations of up to 2% (Tisserand and Balacs,

1995). For cancers and for oestrogen-dependent cancer patients, caution is given against fennel, anise and star anise. For patients with glaucoma, avoidance of *Backhousia citriodora*, lemongrass, may chang and melissa is recommended and in patients with glucose-6-phosphate dehydrogenase (G6PD) deficiency, avoidance of cornmint and peppermint is advised.

Oral intake of certain essential oils is also prohibited (mainly as stated for dermal application before) and caution is advised with bay (West Indian), betel leaf, cinnamon leaf, clove (bud, leaf and stem), garlic, *Ocimum gratissimum*, onion, pimento (berry and leaf) and tejpat leaf, where anticoagulants are used (which include aspirin, heparin and warfarin).

There is considerable reluctance to accept all these cautions as they are probably overexaggerated and the dosage of essential oils is overlooked, however, wormseed (*Chenopodium*) was used to treat intestinal worms and caused poisoning in many children (Mele, 1952) and experiments on monkeys have shown that citral given in small daily doses causes symptoms similar to those of glaucoma (Geldof *et al.*, 1992). Occasionally, completely unfounded contraindications are given in aromatherapy books and it is advisable to cross-reference any such advice with more scientific works (e.g. Tisserand and Balacs, 1995).

### The phenomenon of 'quenching': true or false?

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Many essential oils do not cause sensitisation even though their main components are very potent sensitisers (Opdyke, 1974). Quenching is the term given to this amelioration or complete stoppage of sensitisation of sensitisers when used in a mixture of components as in essential oils or even perfumes. One should not rely on quenching, however, as most commercial essential oils are commonly adulterated and may contain potent sensitiser chemicals anyway.

Some components like aldehydes (e.g. citral) can quench potent sensitisers in essential oils (Opdyke, 1974). For example, cinnamal was apparently quenched by eugenol not limonene and citral was quenched by limonene.

The latest survey at Unilever Toxicology Unit (2000) pours doubt on the original study as there are no signs of interaction (e.g. Schiff base formations). Studies using modern methods (e.g. murine lymph

nodes in mice) have shown no quenching at all. The RIFM produced limited studies that show citral quenched by limonene, but Unilever tests on Opdyke quenchers using guinea-pig models failed to support his work. Ageing perfume mixtures showed no quenching effect either; studies on humans showed no quenching (Basketter and Allenby, 1991). In conclusion, there is no satisfactory physicochemical or immunobiological proof for quenching; it is, at best, a hypothesis, and no 'good components' have been found as yet. Furthermore, as many commercial essential oils are adulterated, potential sensitisers can be added: so it's a no-win situation.

In one instance a high D-limonene-containing (unknown) oil was added to lemongrass (containing citral) by an aromatherapist on the advice of a perfumer, to counteract, by quenching, the sensitising effect on the skin (Price, 1993). One can only wonder if this was a wise move in view of D-limonene's toxicity.

### Possible dangers of novel essential oils and plant extracts

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There is a trend by some 'clinical aromatherapists' to use uncommon essential oils, often derived from plants which are grown wild and which have a tendency to produce numerous cultivars with different chemical compositions (i.e. chemotypes). These oils are very much more expensive, as they are produced in small amounts; the quality will be variable as well as the yield and composition. The real benefit remains a mystery. The International Federation of Aromatherapists (IFA), which represents well-trained therapists, has published articles in its journal promoting such potentially hazardous oils and even the use of *Verbena* essential oil on the skin (Autumn 1999 edition), stating that this oil 'used sensibly, is safe' despite well-documented evidence proving that this oil should never be applied to the skin due to sensitising and phototoxic potential (IFRA, see Appendix 30).

The vast majority of the commonest essential oils have been well tried and tested and safety levels have been ascertained; however, when an aromatherapist uses novel essential oils, they are using their clients as human guinea-pigs (Lis-Balchin, 1999b). This is also unethical unless the client is told that the safety of

such oils is unknown, and legally it could possibly leave the therapist open to court action if the essential oils caused a harmful reaction.

South American and other novel plants and their essential oils, introduced at recent aromatherapy conferences as potential cures for numerous ailments, include muna (*Minthostachys spicata*), a peppermint-smelling herb consisting mainly of pulegone, whose folk usage is as a digestive and spasmolytic when drunk as a tea (Lunny, 1997), and the Bolivian herb *Satureya boliviana*, which also has a high pulegone content. The pulegone-containing European wormwood (*Artemisia absinthium*) must not be used in aromatherapy as aromatherapists believe pulegone to cause abortions. It seems rather odd that *Mentha pulegium* (pennyroyal) is banned, but a new exciting Bolivian herb with a similar composition is thought to be good! Another novel plant, molle (*Schinus molle*), otherwise known as Peruvian mastic, is also suggested as beneficial in aromatherapy (Lunny, 1997), but it is closely related botanically and toxicologically to poison ivy (*Toxicodendron radicans*), one of the most potent dermal irritants and sensitisers known (Lis-Balchin, 1999b).

### **Dangerous practices of some aromatherapists**

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A number of particularly dangerous items and suggestions have been published in the many aromatherapy books and journals and presented at aromatherapy conferences. Among them are aromatic perfusion, oral/rectal/vaginal usage, and the use of essential oils during pregnancy and childbirth.

#### **Aromatic perfusion**

This practice involves using up to 20 mL of undiluted essential oils directly on the skin for specific conditions (Guba, 2000). Apparently the use of red thyme, ajowan, clove bud oil, oregano at concentrations not exceeding 10% for massage, compared with the use of 90% of non-irritant essential oils (true lavender, *Eucalyptus radiata*, tea tree) and up to 10% of phenolic essential oils is considered to be perfectly acceptable. This treatment could, however, cause severe burns/irritation to the skin, especially if the person is slightly sensitive.

#### **Oral/rectal/vaginal usage**

Oral/rectal/vaginal usage of essential oils is advocated by aromatherapists in France and Germany, who are sometimes medically qualified, but also by some medically unqualified aromatherapists in the UK. The use of essential oils in this way is potentially dangerous due to possible damage to the delicate mucosal cells, adulteration of the oils and the use of high dosages. There is also the potential for illegality in the use of untested oils, which are often advocated.

The effects of chemicals, especially cosmetics, are tested on various larger mammals for their potential effects on the sensitive mucous membranes such as in the eyes and mouth. Other specialist products are tested on the membranes of the anus, vagina, and so forth. The animals tested are specially chosen for each type of membrane. For eyes, the rabbit is the only choice due to its large eyes and sensitivity, whilst oral products are tested on the hamster, due to its natural way of storing anything in its food pouches. Dogs or cats are often used for testing penile or vaginal membranes.

Not all essential oils are tested on all membranes, as most are used in cosmetics and foods and are not intended to be used medicinally within the body. However an increasing number of aromatherapists are using these very strong chemicals on tampons inserted into the vagina or anus, and there is a possibility of a patient having a severe reaction to the chemicals on the delicate membranes. This practice is professionally irresponsible, as aromatherapists should not be diagnosing and treating venereal disease unless medically qualified to do so.

#### **Use of essential oils during pregnancy and childbirth**

It has been shown that uterine contractions may possibly be decreased or stopped by essential oils (Lis-Balchin and Hart, 1997b). Over 40 essential oils and 20 components studied *in vitro* on the rat uterus had a spasmolytic effect, and at higher doses they completely stopped the spontaneous contractions, following a rapid reduction in their intensity. This could be a dangerous event during childbirth, where strong contractions are so vital. It was also found that during the oestrus cycle, some animals developed

irregularity in their uterine spontaneous contractions in response to some essential oils: this suggests their potential danger during pregnancy, as a spontaneous abortion could be initiated. There is also the possibility of anaesthetising the baby in the womb if essential oils are used during parturition, resulting in the baby's inability to display the crying reflex on birth.

Almost all of the claims made in aromatherapy books and journals regarding the use of restricted or banned essential oils during pregnancy are largely based on the traditionally claimed effects of the water-soluble herbal extracts, which were mainly taken internally. Such extracts are totally different from the plant essential oils extracted from them. The common essential oils used in aromatherapy are all used as food flavourings and in perfumery, therefore one would have imagined that the slightest evidence of toxicity of these essential oils to the baby would have restricted their usage by legislation. However, not many teratogenic studies have been conducted on essential oils, although they have been used for years as food additives. The main problem these days is seen as the sensitisation potential of many essential oils, and of course these could also have possible effects on the unborn child.

### **'Safe' essential oils and their toxicity**

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The two main essential oils considered by aromatherapists to be safe (and therefore recommended for usage during pregnancy, parturition and on small babies) are lavender and geranium oil, but even these are not without risk. In one case a hairdresser with allergy problems on her hands due to a variety of products reacted most strongly to a lavender shampoo and lavender oil itself (Rudzki *et al.*, 1976). Lavender was said to be a photosensitiser (Brandao, 1986) and patch tests have shown a few allergies due to photosensitisation (Lovell, 1993). Pigmentation has also been reported (Klarmann, 1958).

Most of the references to geranium oil are to contact dermatitis and sensitisation (Romaguera *et al.*, 1986) especially due to one of the main components, geraniol. Although patch tests to geraniol proved negative, dermatitis caused by perfumes containing geranium oil has been observed in a few cases (Klarmann, 1958). Ointments containing geraniol (e.g. *Blastoestimulina*) have been reported to cause sensitisation

when used in the treatment of chronic leg ulcers (Romaguera *et al.*, 1986; Guerra *et al.*, 1987). Geraniol may be an allergen, as cross-reactions occur with citronella (Keil, 1947), however, the main sensitiser found is citronellal (mainly in palmarosa, *Eucalyptus citriodora* and melissa), with citronellol less reactive; geraniol was even weaker, as was citral. In two cases, strong reactions were obtained with 1% solutions of citronellal and weaker ones with citronellol, geraniol and geranyl acetate (see Appendix 22). In 23/23 cases no response was found using lemon oil, which suggests specificity of the response. However, sensitisation to geraniol using a maximisation test proved negative.

Most cosmetics and perfumes are tested on human 'guinea-pigs' using similar tests to those described for animals. These are demanded by the RIFM as a final test before marketing a product. Further data are accumulated from notifications from disgruntled consumers who report dermatitis, itching or skin discoloration in use. These notifications can result in legal claims, although most cases are probably settled out of court and not reported to the general public.

### **Possible dangers of using essential oils internally and externally in large doses**

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Essential oils are used in the food industry usually in very minute amounts of 10 ppm, and even up to 1000 ppm and above in the case of mint confectionery or chewing gum. This does not make it safe to use drops of undiluted essential oils on sugar lumps for oral application or on suppositories for anal or vaginal application. Severe damage to mucous membranes could result from such practices, and the dangers are magnified if cheap, adulterated oils are used.

The Internet has made it possible for a trusting, though often ill-informed, public to purchase a wide range of dubious plant extracts and essential oils. Even illegal essential oils can now be obtained. Furthermore, unqualified people can offer potentially dangerous advice, such as internal usage or the use of undiluted essential oils on the skin for 'mummification', or in order to rid the body of toxic waste. The latter can result in excruciating pain from the burns produced and the subsequent loss of layers of skin.

In one study, aromatherapy trials in high-dose chemotherapy patients were not only unscientific but also very dangerous: essential oils, diluted in water, were given internally or applied externally to very sick patients in a rather haphazard way (reported in *Aroma '93*). The 'experiments' were conducted by an aromatherapist who probably had little knowledge of essential oil chemistry, function or toxicity. Such studies should not have been passed by the hospital safety committee in the first instance.

In an aromatherapy book steam distillation at home was illustrated by a drawing of a kettle perched on a gas flame over the stove; this was loosely attached to a tube passing through ice in a pan, and the essential oil was then collected into a jamjar close by (Rose, 1992a). From the point of health and safety, this was extremely dangerous.

In another book, readers were advised that a woman in labour can be given jasmine or lemon verbena compresses or massages in the sacral area when suffering pain. Even taking lemon verbena internally was recommended to stimulate uterine contractions (Fischer-Rizzi, 1992). Such advice was very dangerous, especially as no concentrations were given and lemon verbena is a potent allergen. To suggest that it is to be used internally for uterine contractions is totally irresponsible as there has been no scientific verification.

The same book gives a recipe for suntan oil, including bergamot, carrot seed and lemon essential oils (Fischer-Rizzi, 1990). These are all phototoxic essential oils. The author then advises that bergamot oil is added to suntan lotion, to get the bonus of the substance called 'furocumarin', which lessens the skin's sun sensitivity while it helps one to tan quickly. This could cause severe burns.

Elsewhere, sassafras (*Ocotea pretiosa*) was said to be only toxic for rats, due to its metabolism and not dangerous to humans (Pénoel, 1991) and a 10% solution in oil was suggested for treating muscular and joint pain and sports injuries. Safrole (and sassafras oil) is, however, controlled under the Controlled Drugs Regulations (1993) and listed as a category 1 substance, as it is a precursor for the illicit manufacture of hallucinogenic, narcotic and psychotropic drugs like ecstasy (see also nutmeg oil monograph).

French practitioners and other therapists have apparently become 'familiar' with untested oils (Guba, 2000). The use of toxicologically untested Nepalese essential oils, etc. includes lichen resinoids, sugandha kokila oil, jatamansi oil and Nepalese

lemongrass (*Cymbopogon flexuosus*), also *Tagetes* oil (Basnyet, 1999).

*Melaleuca rosalina* (*M. ericifolia*), 1,8-cineole 18–26%, is apparently especially useful for the respiratory system (Pénoel, 1998). Because it is so mild to the skin, 2–3 drops can be applied neat on the side of the neck at the area of the lymphatic nodes when treating infections of the upper respiratory system. This essential oil is untested and could be a sensitiser.

'Nurses warn against rash use of herbal oil treatments' (*The Guardian*, 12 March 1999, p. 9) was the unexpected title of a report from the Royal College of Nursing Congress, as nurses are usually very keen to support aromatherapy. Several dangerous scenarios were mentioned, including the intensive marketing of aromatherapy treatments in a 3-year trial on cancer patients and the danger of non-qualified nurses implementing the treatment. An incident concerning a nurse's daughter, who had been given aromatherapy treatment in a hospital during a 5-day stay, was reported. Apparently no choice, no parental consent and no agreement was given. There was also a warning about the possible use of aromatherapy oils poured over children's heads against headlice by inexperienced parents.

### Reporting of adverse effects by aromatherapists

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There have been no reports on any adverse effects in the extensive aromatherapy literature, despite the many reports appearing in the scientific literature. There is at present no 'yellow card' scheme or other regarding the thousands of aromatherapists practising in the UK or elsewhere in the world, although this could provide useful data. There seems to be a reluctance by aromatherapists to participate in any scheme which could prove damaging to their profession and thereby jeopardise their income. The few clinical studies on aromatherapy carried out have not only shown no benefits in using essential oils in massage compared with massage alone, but have also yielded no significant data on adverse effects.

The yellow card scheme operates for those few essential oils sold as licensed products, including peppermint oil (e.g. as Colpermin), but these oils are all GRAS and therefore unlikely to be hazardous unless grossly misused.



## Safety warnings in the aromatherapy industry

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Many aromatherapy suppliers, especially on the Internet, continue to sell dangerous essential oils without adequate warnings. There are also examples of inadequate labelling as to sell by or use by dates, which should always be given especially for essential oils of the citrus and pine families, which develop skin sensitising chemicals on ageing. Shelf-life can be lengthened by the addition of artificial antioxidants; these are disliked by aromatherapists, but vitamin E could be used instead. Warnings should be given that essential oils without antioxidants should not be used on the skin after about six months, but can still be used as fragrances and that storing such oils in optimum conditions, such as in sealed containers in a refrigerator will slow down the chemical changes in the oil.

## Legislation: present and future

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The Health and Safety at Work Act (1974) together with COSHH (Control of Substances Hazardous to Health) Regulations (1994) and CHIP (Chemical Hazard Information and Packaging) Regulations 1998 now control all chemicals in the UK. The New 7th Amendment: European Parliament 2002: To Annex III – Part 1 (Directive 76/768/EEC) covers the conditions of use and warnings which must be printed on the label.

The following components must be indicated in the list of ingredients and a warning given that they can cause an allergic reaction (see also Appendices 28 and 29):

- Benzyl alcohol
- Cinnamyl alcohol
- Eugenol
- Hydroxycitronellal
- Isoeugenol
- Benzyl salicylate
- Cinnamaldehyde
- Coumarin
- Geraniol
- Anisyl alcohol
- Benzyl cinnamate
- Farnesol
- Linalool

- Benzyl benzoate
- Citronellol
- D-Limonene
- Oakmoss and treemoss extract.

This list is greatly increased by the fragrance industry, which has now produced a *Labelling Manual* (EFFA-IOFI-IFRA, 26.3.2001) for health and environmental effects. The proposals could form the basis of an individual supplier's classification and labelling, and it is recommended that they label their goods within six months of the proposal. Sensitisers are labelled 'Xi (irritant); R43' and it is recommended that a 1% concentration is adhered to. Aspiration hazard is 'Xn (harmful); R65' and is recommended now for all substances with a hydrocarbon greater than 10%. Some chemicals do not require labelling; these include citronella oil (Java type) and farnesol. Citronella oil Ceylon type is however Xn. *Pinus nigra*, terpenes and terpenoids, limonene fraction, turpentine oil are now Xn, N. Most of the other components have an Xi or Xn label, with exceptions like safrole which is T (see Appendices 24–26).

The Medicines and Healthcare Products Regulatory Agency in the UK may bring about changes in aromatherapy practice similar to their threat on herbal remedies. The freedom to describe what the essential oils used in aromatherapy are apparently capable of doing will be restricted. Legislation in the USA has already led to several prosecutions following false claims by aromatherapy distributors (see Chapter 3). Legislation brought in by the Health and Safety Executive (HSE) (R65), which came into force on 20 June 1999, classified 89 essential oils as 'harmful'. The danger of this legislation is that aromatherapists are now using some harmful products in their therapy. This immediately places them at serious risk if there is any untoward reaction to their specific treatment. It virtually means that bottles and containers of essential oils now rank with domestic bleach for labelling purposes. It also means that companies are now obliged to self-classify their essential oils on their labels and place them in suitable containers; this applies both to large distributing companies as well as individual aromatherapists reselling essential oils under their own name.

The EU Biocide Directive, which covers about 40 oils, including lavender, was adopted for implementation on 20 May 2000 but has not yet been implemented. It implies that essential oils cannot

be used as biocides, as they are not included in the list.

Finally, new legislation has gone to the Council of Ministers and may imply that only qualified people will be able to use essential oils, and retail outlets for oils will be pharmacies. Their definition of 'qualified' is limited to academic qualifications – doctors or pharmacists.

## **Conclusion**

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In the past essential oils have been considered to be relatively safe, with a few exceptions, but with new

technological advances in the manufacture of synthetic components and 'designer' essential oils to suit every pocket, there is an increasing danger of toxicity, which can manifest itself as sensitisation. New legislation has now become a reality, and warning consumers of the dangers of essential oils on bottle labels, as well as the labels on other cosmetic products, is imminent. The next stage could be the restriction of sales of concentrated essential oils to pharmacies, under the supervision of pharmacists and medically qualified personnel only.