# Chapter 104

# TURPENTINE and PINE OIL (*Pinus* Species)

## HISTORY

Oil of turpentine (terebinthinates) has been an herbal treatment for a variety of illnesses since ancient times. Hippocrates (460–370 BC) mentioned the value of turpentine oil as an emmenagogue (stimulant of menstrual flow) and inhibitor of nasal discharge.[1] The Greek physician, Dioscorides used turpentine oil as an aphrodisiac, diuretic, decongestant, and antiarthritic agent. The Romans used this oil for a wide variety of internal and external diseases including stroke, lethargy, depression, and pleurisy. From the late 17th century through the US Civil War, surgeons used turpentine in the treatment of wounds and amputations. In the Confederate Army, the external application of turpentine was a substitute for quinine in the treatment of malaria.[2] During the 18th and 19th centuries, turpentine oil was administered for the treatment of a variety of infections including croup, yellow fever, typhus, dysentery, and various parasites. Turpentine enemas were a treatment for abdominal distension, hysteria, and amenorrhea during the late 19th century.[3]

## BOTANICAL DESCRIPTION

**Common Name:** Pines

**Scientific Name:** *Pinus* species

**Botanical Family:** Pinaceae

**Physical Description:** Pine trees have distinctive bundles of long, narrow needles and large, woody cones with tough scales. Branches commonly grow in rings around the trunk.

**Distribution and Ecology:** Pines are coniferous species, which are native to Central and North America, Europe, Asia, and small areas of North Africa. Pines are widely cultivated around the world including in Africa and South America. There are a variety of *Pinus* hybrids, and *Pinus* species are the most common conifers worldwide.

## EXPOSURE

### Sources

Oil of turpentine (gum turpentine) is the volatile, oily, liquid fraction derived from the steam distillation of pine resin. The latter material is obtained by tapping living species in the genus *Pinus* including *P. brutia* Tenore (calabrian pine),*Pinus elliottii* Engelm. (slash pine), *P. halepensis* P. Miller (Aleppo pine), *P. massoniana* D. Don (Masson pine, Chinese red pine), *P. merkusii* Junghuhn & Vriese ex Vriese (Merkus pine), *P. pinaster* Aiton (maritime pine), *P. radiata* D. Don (Monterey pine), and *P. sylvestris* L. (Scots pine). Other sources of turpentine include the extraction or destructive distillation of wood (wood turpentine) and by-products of chemical pulping of pine trees. Although technically not a petroleum distillate, most reviews include turpentine with petroleum distillates because turpentine is an aromatic hydrocarbon with properties, toxic effects, and uses similar to petroleum distillates. The skin sensitizer, colophony, is the nonvolatile, solid residue (rosin, gum rosin) that remains after the evaporation of volatiles from the distillation of crude turpentine.[4] Both

*Medical Toxicology of Natural Substances*, by Donald G. Barceloux, MD

colophony (rosin) and turpentine oil are distillation products of *Pinus* species, and the rosin used on violins and equestrian equipment includes both products. The high-temperature distillation of turpentine or other pine oil resins produces pine oil. Tall oil is dark-brown, liquid rosin, which is a by-product of pinewood in the wood pulp industry.

## Uses

### *Traditional*

Traditional uses of turpentine include use as an abortifacient,[5] antihelminthic agent,[6] rubefacient, decongestant, antipyretic, insect repellant,[7] and a treatment for human myasis (i.e., infection with dipterous larvae, such as maggots).[8] A tea brewed from the tips of pine branches was a substitute for coffee.

### *Current*

Turpentine is a constituent of paint and other coatings, paint thinners, and solvents. The use of turpentine for these applications has decreased as other, less-expensive solvents have become available. However, the use of turpentine as a solvent remains common in developing countries. Other products containing turpentine include perfumes, liniments, and cleaning products. Turpentine is a raw product for the isolation of chemicals (e.g., camphor, citral, citronellal, isobornyl acetate, linalool, menthol, pine oil) in a wide range of commercial products. Pine oil is a solvent for varnish and polish, and synthetic pine oil is a constituent of fragrances and flavorings. Purification of monoterpene compounds in pine oil or the acid catalysis of pinene produces *l*-limonene, whereas citrus pulp or peels are the sources for the *d*-isomer of limonene. More recent formulations of pine oil-cleaning products contain smaller concentrations of pine oil and higher concentrations of constituents that reduce the viscosity and aspiration potential of the product.

# PRINCIPAL INGREDIENTS

## Chemical Composition

Turpentine is the volatile, predominantly terpenic, fraction or distillate resulting from the solvent extraction of softwoods (e.g., pine). The exact composition of turpentine oil depends on a variety of factors including extraction and refining methods, plant part, species, geographical location, and season. The major constituents of turpentine (CAS RN:8006-64-2) are terpene compounds ($C_{10}H_{16}$), such as α-pinene, β-pinene, camphene, 3-carene, and dipentene (*d*,*l*-limonene).[9,10] Other constituents include other acyclic, monocyclic, or bicyclic terpenes, oxygenated terpenes, and anethole. Monoterpenes in turpentine are volatile substances that occur naturally in air as a result of the emission of these substances from vegetation.[11] *d*-Limonene is a minor constituent of turpentine, but this compound is a major constituent of caraway, dill, celery, and oil from several fruits of the genus *Citrus* (lemon, orange, grapefruit).

Pine oil contains highly lipophilic secondary and tertiary cyclic terpene alcohols. Depending on the source of turpentine and the production methods, pine oil can contain terpene ethers and other hydrocarbons. Analysis of a sample of pine oil demonstrated 57% α-pinene, 26% carene, 8% β-pinene, 6% limonene, and 3% other hydrocarbons as measured by gas chromatography/mass spectrometry.[12] Individual constituents of the essential oil vary with *Pinus* species, extraction methods and plant parts. Table 104.1 lists the common constituents of the essential oil from the Balkan pine (*Pinus peuce* Gris.), North American pine (*Pinus ponderosa*, subspecies not reported), red pine (*Pinus resinosa* Aiton), and the eastern white pine (*Pinus strobus* L.).[13,14] Pine oil is usually present in household products along with other constituents (e.g., isopropyl alcohol, chloroxylenol), which may increase CNS and pulmonary toxicity.[15] Pine bark extract contains a variety of phenolic acid glucosides with anti-inflammatory properties, catechin compounds, taxifolin and taxifolin derivatives, procyanidins, and lignan glucosides.[16]

## Physiochemical Properties

*d*-Limonene is an excellent solvent, but air oxidation of this substance produces potent sensitizing agents.[17] Turpentine also contains the strong sensitizer, Δ-3-carene.[18] These unsaturated cyclic hydrocarbons are very lipophilic. Turpentine is soluble in ether and alcohol, but insoluble in water. The water solubility of turpentine at 20 °C–25 °C (68 °F–77 °F) is 0.023% by weight and the vapor pressure at 25 °C (77 °F) is 5 mmHg.[19] The ingestion of turpentine produces a violet-like odor of the breath and urine. The viscosity of pine oil is greater than turpentine, and therefore the aspiration hazard of pine oil is less than the aspiration hazard of the turpentine. The terpene compounds present in turpentine are released into the air both from natural (bark, cortex of conifers) and anthropogenic (wood processing) sources. Monoterpenes (α-pinene, β-pinene, 3-carene) are highly photoreactive in the atmosphere with relatively short atmospheric half-lives.[20,21] Some evaporation of monoterpene compounds occurs during the use of cleaning products containing pine oil, particularly α-phellandrene, terpinolene, γ-terpinene, *d*-limonene, camphene, and α-pinene.[22]

**TABLE 104.1.** Most Common Constituents as a Percentage of the Total Essential Oil from Several *Pinus* Species

| Compound | *Pinus peuce*[a] | *Pinus peuce*[b] | *Pinus strobes*[b] | *Pinus ponderosa*[b] | *Pinus resinosa*[b] |
|---|---|---|---|---|---|
| β-Phellandrene | 26.93 | 6.78 | 3.0[+] | 2.4[+] | 2.5[+] |
| β-Pinene | 12.46 | 22.00 | 7.9 | 45.7 | 42.4 |
| Citronellol | 12.48 | 13.42 | NR | NR | NR |
| α-Pinene | 7.38 | 23.07 | 17.7 | 10.2 | 23.3 |
| β-Caryophyllene | 4.48 | 3.05 | 3.8 | 0.2 | 2.2 |
| Myrcene | 3.41 | 2.04 | 3.6 | 1.4 | 14.5 |
| 3-Carene | 2.58 | 0.46 | Trace | 8.4 | 0.5 |
| δ-Cadinene | 1.60 | 0.65 | 7.5 | 3.1 | 0.4 |
| Bornyl acetate | 0.57 | 9.76 | NR | NR | NR |
| Camphene | 0.24 | 5.52 | 3.2 | 0.5 | 1.6 |
| Terpinyl acetate | 0.56 | 2.02 | NR | NR | NR |
| Germacrene D | NR | NR | 12.2 | 0.3 | 4.9 |
| α-Cadinol | NR | NR | 5.7 | 2.7 | 0.3 |
| γ-Cadinene | 1.16 | 0.36 | 2.8 | 0.9 | 0.1 |
| α-Humulene | 0.97 | 0.53 | 0.9 | 0.3 | 0.4 |

Source: Data from Refs 13 and 14.
[a]Twig oil.
[b]Needle oil.
[+]Includes limonene.
NR, not reported.

## Mechanism of Toxicity

*In vivo* animal experiments indicate that the central nervous system is the primary organ of toxicity, whereas alveolar damage results from the aspiration of less volatile gums and resins.[23] Hence, lower volatility means aspiration risk is much lower for turpentine than with more volatile petroleum distillates. Colophony is also a potent skin sensitizer that previously contaminated turpentine. Older preparations of turpentine contained irritants (formic acid, aldehydes) and relatively high concentrations of skin sensitizers (δ-3-carene, α-pinene).[24,25]

# DOSE RESPONSE

## Ingestion

The toxic dose of turpentine or pine oil is not well defined because of the effects of aspiration, the variable composition of essential oils, and the presence of other toxic compounds in most turpentine and pine oil products. Most accidental exposures to turpentine or pine oil produce mild symptoms, if any, provided pulmonary aspiration does not occur. Ingestions exceeding 2 mL/kg probably are potentially serious.[26] The ingestion of the equivalent of about 200 mL pure pine oil by an adult was associated with coma, whereas the ingestion of the equivalent of approximately 30 mL by an 18-month-old child caused lethargy and ataxia.[27] Both patients recovered without sequelae. A 14-month-old child developed tachypnea, lethargy, and seizures after reportedly ingesting 120 mL turpentine.[28] Within 24 hours, the child was asymptomatic.

## Inhalation

Volunteer studies indicate that eye, nose, and throat irritation occurs following exposure to turpentine concentrations in the range of 125–175 ppm.[29] Following exposure of volunteers to turpentine vapors at 75 ppm, several subjects developed mild nasal and throat irritation. Although 175 ppm was intolerable, most subjects could tolerate daily exposure to 100 ppm. The American Conference on Governmental and Industrial Hygienists (ACGIH) recommends a threshold limit value–time weighted average (TLV-TWA) of 100 ppm to minimize the potential for upper respiratory tract irritation. There is no short-term exposure limit (STEL) because of limited data. Both the US Occupational Safety and Health Administration permissible exposure limit (OSHA PEL) and the National Institute for Occupational Safety and Health recommended exposure limits (NIOSH REL) are consistent with the TLV-TWA. The NIOSH immediately dangerous to life or health (IDLH) value is 1500 ppm. There are no well-recognized chronic effects from inhalational exposure to turpentine other than contact dermatitis.

## TOXICOKINETICS

The main components of turpentine are monoterpenes (e.g., α-pinene, β-pinene, 3-carene, *d*-limonene). The high solubility of monoterpenes in blood indicates that these compounds are rapidly absorbed from the lungs and stored in adipose tissues.[30] Based on volunteer studies, the respiratory uptake of these monoterpenes ranges from about 60–70%. In a study of eight volunteers exposed to turpentine at 450 mg/m$^3$ for 2 hours, the mean relative uptakes of α-pinene, β-pinene, and 3-carene were 62%, 66%, and 68%, respectively.[31] Metabolism accounts for most of the elimination of the ingested monoterpenes with relatively small amounts of unchanged monoterpenes appearing in the urine and in exhaled air. During experimental studies, the amount of unchanged α-pinene,[32] 3-carene,[33] and *d*-limonene[36] excreted in expired air were 8%, 3%, and 1%, respectively, whereas the amount of unchanged monoterpene excreted in the urine was <0.001%. Following the ingestion of pine oil, the metabolism and renal elimination of monoterpenes is relatively slow. The peak urinary excretion of the main metabolite, bornyl acetate, occurred approximately 5–6 days after the ingestion of pine oil.[34] Following exposure of volunteers to α-pinene, the kidneys eliminated about 4% of the dose as *cis*- and *trans*-verbenol.[35] Although a slow terminal elimination phase exists, volunteer studies indicate that most of a single, ingested dose of these monoterpenes is eliminated within 3–4 days.[36] α-Terpineol glucuronide disappeared rapidly from the urine of an 18-month-old child who was surreptitiously administered pine oil, and this compound was not detectable in urine samples obtained 12 days after admission.[37]

## CLINICAL RESPONSE

### Adverse Effects

Turpentine oil is both an irritant and a sensitizer. As reported to the German Information Network of Departments of Dermatology Multicentre Project, the rate of sensitization to oil of turpentine was approximately 2% in tested patients with a slight decline in recent years.[38] Although relatively rare, allergic contact dermatitis results from exposure to turpentine oil, particularly in atopic individuals working as painters, potters, perfumery workers, or violinists (fiddler's neck).[39,40] A painter with prior sensitization to colophony developed hand eczema after handling lottery tickets that contained colophony.[41] Cross-reaction occurs between turpentine and a variety of other essential oils including tea tree oil, peppermint, balsam of Peru, pyrethrum, chrysanthemum, and ragweed.[42,43]

### Acute Toxicity

Most internal human exposures to turpentine or pine oil involve the accidental ingestion of turpentine-containing products by children. Turpentine oil is a marked gastrointestinal irritant that produces more gastrointestinal and central nervous system symptoms than a similar ingestion of a petroleum distillate.[37] However, the ingestion of turpentine products produces a significantly lower incidence of pneumonitis compared with petroleum distillates.[44] Clinical features following the ingestion of mixtures containing *Pinus* distillates (turpentine oil, pine oil) include mucosal irritation (nausea, vomiting, abdominal pain, sore throat, chest pain), mild respiratory tract irritation (cough, transient shortness of breath) and central nervous symptoms (headache, elation, confusion, ataxia, lethargy).[27] A violet-like odor to the breath and urine suggests ingestion of pine oil, although this odor is not always present. Coma, seizures, and death can result from large, intentional ingestions of turpentine or pine oil.[45] Symptoms usually develop within 2–3 hours following ingestion, and the toxic effects typically resolve within 24 hours.[26] Rare complications of turpentine or pine oil aspiration include acute respiratory distress syndrome (ARDS), bronchopleural fistula, pneumatoceles, lung necrosis, empyema,[46] pneumothorax, sepsis, and multiorgan failure.[47,48] Urinary tract irritation (dysuria, hematuria, frequency) may also occur.[49,50] The parenteral injection of approximately 5 mL of turpentine was associated with the development of hypoxemia and noncardiogenic pulmonary edema, followed by cellulitis and a sterile abscess at the injection site.[51] Similar respiratory abnormalities occurred in horses injected with pine oil for malicious purposes.[52]

### Chronic Toxicity

Preliminary animal studies indicate that turpentine oil has weak tumor promoting properties.[53] Neither the International Agency for research on Cancer (IARC) nor the US Toxicology Program list turpentine oil or major ingredients in turpentine oil as suspected carcinogens.

## DIAGNOSTIC TESTING

### Analytical Methods

Turpentine oil contains many compounds. Terpenoid compounds increased the flexibility of the oil binders in oil paintings.[54] Analysis of these compounds in pine resins (e.g., Venice turpentine from *Larix decidua* Mill.) by gas chromatography (GC) and mass spectrometry

(MS) helps determine the best compounds for restorations of old paintings.[55]

## Biomarkers

The blood turpentine concentration in an 85-year-old woman as measured by GC with flame ionization detector was 28 μg/mL, when compared with a standard consisting of the material involved with the poisoning.[56] At the time the sample was drawn, she was comatose, hypotensive, and apneic. 1-α-Terpineol is a major constituent of pine oil and a biomarker for ingestion of substances containing pine oil.[52] Postmortem blood from a 89-year-old Alzheimer's patient, who was found at home in cardiac arrest after drinking about 100 mL Pine-Sol® (Uline, Waukegan, IL), contained 25 mg/dL 1-α-terpineol as measured by gas chromatography/mass spectrometry.[57] Pine oil composed about 17–22% of this product, and about 50% of the pine oil consisted of 1-α-terpineol.

## Abnormalities

Complications of the ingestion of turpentine include hematuria, thrombocytopenia,[58] and pneumatoceles.[59] The typical course of the pneumatocele is slow, progressive resolution of the radiographic changes over a period of weeks. Laboratory abnormalities following the ingestion of mixtures of monoterpene compounds can also include leukocytosis and mild elevation of hepatic aminotransferases.[12]

# TREATMENT

The treatment of accidental exposures to turpentine is similar to the treatment of petroleum distillates. Patients should be observed for the development of signs of aspiration because the ingestion of turpentine frequently causes vomiting. Decontamination measures are not recommended because of the lack of documented efficacy and the risk of pulmonary aspiration. These patients should be admitted for observation for the development of respiratory depression and seizure activity. Asymptomatic children may be discharged after 3–4 hours of observation if the 2-hour postingestion chest x-ray is normal. Because monoterpenes are lipophilic compounds with relatively low blood concentration, the use of hemodialysis and hemoperfusion probably does not alter clinical outcome.[34]

## References


1. Haller JS Jr. Sampson of the terebinthinates: medical history of turpentine. South Med J 1984;77:750–754.
2. Hasegawa GR. Quinine substitutes in the confederate army. Mil Med 2007;172:650–655.
3. Doyle D. Per rectum: a history enemata. J R Coll Physicians Edinb 2005;35:367–370.
4. Cronin E. Oil of turpentine—a disappearing allergen. Contact Dermatitis 1979;5:308–311.
5. Quander MF, Moseley JE. Abortion, chemical peritonitis and pulmonary edema following intrauterine injection of turpentine. Obstet Gynecol 1964;24:572–574.
6. Lanski SL, Greenwald M, Perkins A, Simon HK. Herbal therapy use in a pediatric emergency department population: expect the unexpected. Pediatrics 2003;111:981–985.
7. Ansari MA, Mittal PK, Razdan RK, Sreehari U. Larvicidal and mosquito repellent activities of pine (*Pinus longifolia*, Family: Pinaceae) oil. J Vect Borne Dis 2005;42:95–99.
8. Shaunik A. Pelvic organ myiasis. Obstet Gynecol 2006;107: 501–503.
9. Mirov NT, Zavarin E, Bicho JG. Composition of gum turpentine of pines *Pinus nelsonii* and *Pinus occidentalis*. J Pharm Sci 1962;51:1131–1135.
10. Williams AL, Bannister MH. Composition of gum turpentines from twenty-two species of pines grown in New Zealand. J Pharm Sci 1962;51;970–975.
11. Fehsenfeld F, Calvert J, Fall R, Goldan P, Guenther AB, Hewitt CN, et al. Emissions of volatile organic compounds from vegetation and the implications for atmospheric chemistry. Global Biogeochem Cycles 1992;6:389–430.
12. Koppel C, Tenczer J, Tonnesmann U, Schirop TH, Ibe K. Acute poisoning with pine oil– metabolism of monoterpenes. Arch Toxicol 1981;49:73–78.
13. Koukos PK, Papadopoulou KI, Patiaka DT, Papagiannopoulos AD. Chemical composition of essential oils from needles and twigs of Balkan pine (*Pinus peuce* Grisebach) grown in Northern Greece. J Agric Food Chem 2000;48:1266–1268.
14. Krauze-Baranowska M, Maradarowicz M, Wiwart M, Poblocka L, Dynowska M. Antifungal activity of the essential oils from some species of the genus *Pinus*. Z Naturforsch [C] 2002;57:478–482.
15. Chan TY, Critchley AJ, Lau JT. The risk of aspiration in Dettol poisoning: a retrospective cohort study. Hum Exp Toxicol 1995;14:190–191.
16. Karonen M, Hamalainen M, Nieminen R, Klika KD, Loponen J, Ovcharenko VV, et al. Phenolic extractives from the bark of *Pinus sylvestris* L. and their effects on inflammatory mediators nitric oxide and prostaglandin E2. J Agric Food Chem 2004;52:7532–7540.
17. Karlberg A-T, Magnusson K, Nilsson U. Air oxidation of *d*-limonene (the citrus solvent) creates potent allergens. Contact Dermatitis 1992;26:332–340.
18. Dooms-Goossens A, Degreef H, Holvoet C, Maertens M. Turpentine-induced hypersensitivity to peppermint oil. Contact Dermatitis 1977;3:304–308.
19. Anonymous. Turpentine. Am Ind Hyg Assoc J 1967;28: 297–300.

20. Stromvall A-M, Petersson G. Monoterpenes emitted to air from industrial barking of Scandinavian conifers. Environ Poll 1993;79;215–218.
21. Stromvall A-M, Petersson G. Photooxidant-forming monoterpenes in air plumes from Kraft pulp industries. Environ Poll 1993;79:219–223.
22. Singer BC, Destaillats H, Hodgson AT, Nazaroff WW. Cleaning products and air fresheners: emissions and resulting concentrations of glycol ethers and terpenoids. Indoor Air 2006;16:179–191.
23. Sperlin F. *In vivo* and *in vitro* toxicology of turpentine. Clin Toxicol 1969;2:21–35.
24. Lear JT, Heagerty AH, Tan BB, Smith AG, English JS. Transient re-emergence of oil of turpentine allergy in the pottery industry. Contact Dermatitis 1996;35:169–172.
25. Pirila V, Kilpio O, Olkkonen A, Pirila L, Siltanen E. On the chemical nature of the eczematogens in oil of turpentine. Dermatologica 1969;139:183–194.
26. Jacobziner H, Raybin HW. Turpentine poisoning. Arch Pediatr 1961;78:357–364.
27. Brook MP, McCarron MM, Mueller JA. Pine oil cleaner ingestion. Ann Emerg Med 1989;18:391–395.
28. Craig JO. Poisoning by the volatile oils in childhood. Arch Dis Child 1953;28:475–483.
29. Nelson KW, Ege JF Jr , Ross M, Woodman LE, Silverman L. Sensory response to certain industrial solvent vapors. J Ind Hyg Toxicol 1943;25:282–285.
30. Falk A, Gullstrand E, Lof A, Wigaeus-Hjelm E. Liquid/air partition coefficients of four terpenes. Br J Ind Med 1990;47:62–64.
31. Filipsson AJ. Short term inhalation exposure to turpentine: toxicokinetics and acute effects in men. Occup Environ Med 1996;53:100–105.
32. Falk AA, Hagberg MT, Lof AE, Wigaeus-Hjelm EM, Wang ZP. Uptake, distribution and elimination of alpha-pinene in man after exposure by inhalation. Scand J Work Environ Health 1990;16:372–378.
33. Falk AA, Lof AE, Hagberg MT, Wigaeus-Hjelm EM, Wang ZP. Human exposure to 3-carene by inhalation: toxicokinetics, effects on pulmonary function and occurrence of irritative and CNS symptoms. Toxicol Appl Pharmacol 1991;110:198–205.
34. Koppel C, Tenczer J, Tonnesmann U, Schirop T, Ibe K. Acute poisoning with pine oil: metabolism of monoterpenes. Arch Toxicol 1981;49:73–78.
35. Levin J-O, Eriksson K, Falk A, Lof A. Renal elimination of verbenols in man following experimental α-pinene inhalation exposure. Int Arch Occup Environ Health 1992;63:571–573.
36. Falk-Filipsson A, Lof A, Hagberg M, Wigaeus-Hjelm E, Wang Z. *d*-Limonene exposure to humans by inhalation: uptake, distribution, elimination, and effects on the pulmonary function. J Toxicol Environ Health 1993;38: 77–88.
37. Hill RM, Barer J, Hill LL, Butler CM, Harvey DJ, Horning MG. An investigation of recurrent pine oil poisoning in an infant by the use of gas chromatographic-mass spectrometric methods. J Pediatrics 1975;87:115–118.
38. Schnuch A, Lessmann H, Geier J, Frosch PJ, Uter W. Contact allergy to fragrances: frequencies of sensitization from 1996 to 2002. Results of the IVDK. Contact Dermatitis 2004;50:65–76.
39. Kuner N, Jappe U. Allergic contact dermatitis from colophonium, turpentine and ebony in a violinist presenting as fiddler's neck. Contact Dermatitis 2004;50:258–259.
40. Vente C, Fuchs T. Contact dermatitis due to oil of turpentine in a porcelain painter. Contact Dermatitis 1997;37: 187.
41. Pereira F, Manuel R, Gafvert E, Lacerda MH. Relapse of colophony dermatitis from lottery tickers. Contact Dermatitis 1997;37:43.
42. Heine G, Schnuch A, Uter W, Worm M. Frequency of contact allergy in German children and adolescents path tested between 1995 and 2002: results from the Information Network of Department of Dermatology and the German Contact Dermatitis Research Group. Contact Dermatitis 2004;51:111–117.
43. Laube S, Tan BB. Contact dermatitis from turpentine in a painter. Contact Dermatitis 2004;51:41–42.
44. Beamon RF, Siegel CJ, Landers G, Green V. Hydrocarbon ingestion in children: A six year retrospective study. JACEP 1976;5:771–775.
45. Pande TK, Pani S, Hiran S, Rao VV, Shah H, Vishwanathan KA. Turpentine poisoning: a case report. Forensic Sci Int 1994;65:47–49.
46. Khan AJ, Akhtar RP, Faruqui ZS. Turpentine oil inhalation leading to lung necrosis and empyema in a toddler. Pediatr Emerg Care 2006;22:355–357.
47. Welker JA, Zaloga GP. Pine oil ingestion a common cause of poisoning. Chest 1999;116:1822–1826.
48. Rodricks A, Satyanarayana M, D'Souza GA, Ramachandran P. Turpentine-induced chemical pneumonitis with broncho-pleural fistula. J Assoc Physicians India 2003;51: 729–730.
49. Chapman EM. Observations of the effect of paint on the kidneys with particular reference to the role of turpentine. J Ind Hyg Toxicol 1941;23:277–289.
50. Klein FA, Hackler RH. Hemorrhagic cystitis associated with turpentine ingestion. Urology 1980;16:187.
51. Wason S, Greiner PT. Intravenous hydrocarbon abuse. Am J Emerg Med 1986;4:543–544.
52. Tobin T, Swerczek TW, Blake JW. Pine oil toxicity in the horse: drug detection, residues and pathological changes. Res Commun Chem Pathol Pharmacol 1976;15:291–301.
53. Roe FJ, Field WE. Chronic toxicity of essential oils and certain other products of natural origin. Food Cosmet Toxicol 1965;3:311–324.
54. Cartoni G, Russo MV, Spinelli F, Talarico F. GC-MS characterisation and identification of natural terpenic resins employed in works of art. Ann Chim 2004;94:767–782
55. Osete-Cortina L, Domenech-Carbo MT, Mateo-Castro R, Gimeno-Adelantado JV, Bosch-Reig F. Identification

of diterpenes in canvas painting varnishes by gas chromatography-mas spectrometry with combined derivatisation. J Chromatogr A 2004;1024:187–194.

56. Troulakis G, Tsatsakis AM, Tzatzarakis M, Astrakianakis A, Dolapsakis G, Kotas R. Acute intoxication and recovery following massive turpentine ingestion: clinical and toxicological data. Vet Hum Toxicol 1997;28:155–157.
57. Cording DJ, Vallaro GM, DeLuca R, Camporese, Spratt E. A fatality due to accidental PineSol™ ingestion. J Anal Toxicol 2000;24:664–667.
58. Wahlberg P, Nyman D. Turpentine and thrombocytopenic purpura. Lancet 1969;2:215–216.
59. Bray A, Pirronti T, Marano P. Pneumatoceles following hydrocarbon aspiration. Eur Radiol 1998;8:262–263.