The MAK Collection for Occupational Health and Safety

2-Ethylhexyl acetate

MAK Value Documentation – Translation of the German version from 2014

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2-Ethylhexyl acetate

MAK value (2013)	**10 ml/m³ (ppm) ≙ 71 mg/m³**
Peak limitation (2013)	**Category I, excursion factor 1**
Absorption through the skin	**–**
Sensitization	**–**
Carcinogenicity	**–**
Prenatal toxicity (2013)	**Pregnancy Risk Group C**
Germ cell mutagenicity	**–**
BAT value	**–**
Chemical name	2-ethyl-1-hexyl acetate
Synonyms	acetic acid, 2-ethylhexyl ester 2-ethyl-1-hexanol acetate beta-ethylhexyl acetate
CAS number	103-09-3
Structural formula	$CH_3COO–CH_2–CH(C_2H_5)(CH_2)_3–CH_3$
Molecular formula	$C_{10}H_{20}O_2$
Molecular weight	172.27
Melting point	–80°C (ECHA 2012)
Boiling point at 1000 hPa	198.7°C (ECHA 2012)
Density at 20°C	0.87 g/cm³ (ECHA 2012)
Vapour pressure at 25°C	0.31 hPa (ECHA 2012; OECD 2010)
log K_{OW}[1)]	4.13 (Tanii et al. 1994); 4.2 (ECHA 2012)
Solubility	3.9 mg/l water at 20°C (ECHA 2012; OECD 2010)
1 ml/m³ (ppm) ≙ 7.148 mg/m³	**1 mg/m³ ≙ 0.139 ml/m³ (ppm)**

1) octanol/water partition coefficient

2-Ethylhexyl acetate is produced with acetic acid via the esterification of 2-ethylhexanol. 2-Ethylhexyl acetate is used mainly as solvent in the production of paints and coatings. A small amount is used as fragrance. 2-Ethylhexyl acetate has a sweet smell and an odour threshold of about 0.007 ml/m^3 (OECD 2010).

After systemic absorption, 2-ethylhexyl acetate is rapidly hydrolyzed to 2-ethylhexanol and acetic acid. The rapid and complete hydrolysis has been demonstrated in vitro in blood and in vivo (see Section 3). Studies with 2-ethylhexanol can therefore be used for the evaluation of 2-ethylhexyl acetate (EFSA 2008; OECD 2010). Local irritation is the critical effect of 2-ethylhexanol and acetic acid (documentation "2-Ethylhexanol" 2003, supplement "Acetic acid" 2010 b, a translation of the 2008 German documentation); the same should also apply for 2-ethylhexyl acetate.

1 Toxic Effects and Mode of Action

2-Ethylhexyl acetate is rapidly hydrolyzed to 2-ethylhexanol and acetic acid after absorption.

2-Ethylhexyl acetate has mildly irritating effects on the skin and eyes of rabbits; there are no studies available of the irritating effects on the airways. Local irritation of the eyes is the critical effect of the hydrolysis products 2-ethylhexanol and acetic acid; the same should apply also for 2-ethylhexyl acetate, although the effects of the hydrolysis products themselves are stronger.

2-Ethylhexyl acetate causes only mild systemic toxicity after short-term inhalation, oral and dermal exposure. There are no valid studies available with repeated exposure to 2-ethylhexyl acetate. In a 90-day inhalation study with rats, neither effects on the nose nor systemic effects occurred up to the highest 2-ethylhexanol concentration tested of 120 ml/m^3. In long-term studies with 2-ethylhexanol, reduced body weight gains, a poor general condition, laboured breathing, piloerection or a urine-coated genital region were observed in rats at doses of 150 mg/kg body weight and day and above. In the mouse, mortality, reduced body weight gains and food consumption, and hyperplasia of the epithelium of the forestomach were found at 750 mg/kg body weight and day and above. Acetic acid is a product of intermediary metabolism. In rats, the maximum tolerable dose (MTD) was found to be acetic acid doses of 1800 mg/kg body weight and day administered for the duration of two weeks.

In studies with humans, 2-ethylhexyl acetate did not cause sensitization of the skin; there are no studies with animals available.

2-Ethylhexyl acetate yielded negative results in mutagenicity tests with bacteria and UDS (test for unscheduled DNA synthesis) tests with primary rat hepatocytes. There are no in vivo genotoxicity studies with 2-ethylhexyl acetate. 2-Ethylhexanol is not genotoxic in vivo, and therefore genotoxic effects are not expected for 2-ethylhexyl acetate in vivo.

There are no carcinogenicity studies with 2-ethylhexyl acetate available. In a long-term study, the hydrolysis product 2-ethylhexanol caused liver tumours only in female mice after oral doses of 750 mg/kg body weight and day, which exceeded the maximum tolerable dose. In rats and male mice, 2-ethylhexanol did not induce an increase in the incidence of tumours.

Studies with 2-ethylhexyl acetate of prenatal toxicity are not available. As 2-ethylhexanol does not have toxic effects on development after either inhalation or dermal application, and effects on the offspring occur after oral administration only at maternally toxic doses, developmental toxicity is not to be expected after exposure to 2-ethylhexyl acetate.

2 Mechanism of Action

There are no data available for 2-ethylhexyl acetate.

The metabolite 2-ethylhexanol leads to peroxisome proliferation (see documentation "2-Ethylhexanol" 2003).

3 Toxicokinetics and Metabolism

There are no data available for dermal or oral absorption, or the inhalation of 2-ethylhexyl acetate.

Assuming the exposure of a surface area of skin of 2000 cm^2 for one hour to an aqueous solution saturated with 2-ethylhexyl acetate, amounts of 68, 1.1 and 0.4 mg are absorbed according to the models of Fiserova-Bergerova et al. (1990), Guy and Potts (1993) and Wilschut et al. (1995).

In vitro (blood) and in vivo experiments with rodents demonstrated the rapid hydrolysis of 2-ethylhexyl acetate to 2-ethylhexanol and acetic acid. The half-life in vitro in blood was 2.3 minutes (OECD 2010).

The hydrolysis of 2-ethylhexyl acetate (highest purity) was investigated in an in vitro study with S9 mix from nasal turbinates. The S9 homogenates were produced from the tissue of male F344/N rats (15–20 weeks old). The esterase activity in nasal turbinates is high compared with that in other tissues, including the liver. Incubation for 10 minutes at 37°C yielded a hydrolysis rate for 2-ethylhexyl acetate of 47 nmol/mg S9 protein/minute (Dahl et al. 1987).

It is not known, however, which of the numerous esterases are involved in the hydrolysis of 2-ethylhexyl acetate. Polymorphisms of the esterases may also be present, as with pseudo-cholinesterase (for example, butyrylcholinesterase or serum cholinesterase), which can cleave various esters which contain an alcohol component other than choline. Homozygous persons with the atypical variant of the enzyme (around 1 : 2000) hydrolyze the corresponding substrates much slower (Allebrand 2005; Lockridge 1990; Novelli and Biancolella 2011).

The metabolism of acetic acid in the organism corresponds to the acetate ion formed during intermediary metabolism, which is used on the one hand in the production of numerous endogenous substances, and on the other hand can be degraded to form CO_2 (documentation "Acetic acid" 2010 a, a translation at the 2002 German documentation).

It is assumed that after intravenous injection, dermal application and oral administration, 2-ethylhexanol is metabolized via 2-ethylhexaldehyde to 2-ethylhexanoic acid. Further metabolites found in animal experiments are 5-hydroxy-2-ethylhexanoic acid, 2-ethyl-1,6-hexanedioic acid and 6-hydroxy-2-ethylhexanoic acid. Elimination takes place rapidly and mainly with the urine (OECD 2010; documentation "2-Ethylhexanol" 2003, supplement "2-Ethylhexanol" 2006, available in German only).

4 Effects in Humans

There are no data available for the end points repeated exposure, reproductive toxicity, genotoxicity and carcinogenicity.

Single exposures

There are no studies with humans after single exposures to 2-ethylhexyl acetate.

In volunteers, the metabolite 2-ethylhexanol did not cause local irritation up to concentrations of 15 ml/m^3 (supplement "2-Ethylhexanol" 2012, available in German only).

The irritation threshold for acetic acid is 40 ml/m^3. The NOAEC (no observed adverse effect concentration) for subjective sensory effects and objective local effects is 10 ml/m^3 (supplement "Acetic acid" 2010 b, a translation of the 2008 German documentation).

Local effects on skin and mucous membranes

In patch tests, 2-ethylhexyl acetate was not found to cause skin irritation (no other details; ECHA 2012).

Allergenic effects

In a maximization test with 29 volunteers, 4% 2-ethylhexyl acetate in petrolatum did not cause sensitization of the skin (no other details; Opdyke 1979).

5 Animal Experiments and in vitro Studies

5.1 Acute toxicity

5.1.1 Inhalation

The exposure of 3 rats to 2-ethylhexyl acetate concentrations of 7800 mg/m³ (1106 ml/m³) for 6 hours was not lethal. The effects on the respiratory tract were not reported (OECD 2010).

5.1.2 Oral administration

The oral LD_{50} for 2-ethylhexyl acetate in rats and mice is more than 3200 mg/kg body weight. At 3200 mg/kg body weight, weakness and ataxia occurred (no other details; OECD 2010).

2-Ethylhexyl acetate (technical grade) was administered to groups of 10 female rats (doses not specified) with a recovery period of 4 weeks. The LD_{50} was 5140 mg/kg body weight. The substance was lethal for the animals mainly within the first 24 hours. The clinical symptoms of intoxication were slight agitation or narcosis. The surviving animals recovered sufficiently after severe weight loss (no other details). Gross-pathological examination of the animals that died revealed non-specific congestion in the internal organs; there were no gross-pathological findings in the surviving animals (ECHA 2012).

5.1.3 Dermal application

In a study from the 1940s, the dermal LD_{50} for 2-ethylhexyl acetate in guinea pigs was found to be more than 20 ml/kg body weight (17 436 mg/kg body weight). Undiluted substance was applied to the skin of 6 animals and left occlusively on the skin for 4 days; the animals were observed over a 10-day recovery period (ECHA 2012; OECD 2010).

5.2 Subacute, subchronic and chronic toxicity

5.2.1 Inhalation

There are no studies available with 2-ethylhexyl acetate or acetic acid.

In a 90-day study with rats, the NOAEC for the effects of 2-ethylhexanol on the nose and for systemic effects was 120 ml/m³. In the same report, a 14-day study was described in which likewise a NOAEC of 120 ml/m³ was found (BASF AG 1992 a). In both studies 120 ml/m³ was the highest concentration tested (vapour saturation concentration).

5.2.2 Oral administration

There are no studies with 2-ethylhexyl acetate available.

A 13-week gavage study with the metabolite 2-ethylhexanol yielded a NOAEL (no observed adverse effect level) of 125 mg/kg body weight and day (see documentation "2-Ethylhexanol" 2003). At 250 mg/kg body weight and day, the fat deposits in the liver of male rats were reduced, in the female animals the relative stomach weights were increased. In both sexes the absolute and relative liver weights were increased. Peroxisome proliferation was demonstrated by the increased activity of palmitoyl CoA oxidase. In a gavage study with mice with identical doses, the NOAEL was 125 mg/kg body weight and day for male mice and 250 mg/kg body weight and day for female mice. At the next-higher dose of 250 mg/kg body weight and day, increased relative stomach weights were found in the males, and at 500 mg/kg body weight and day and above slight acanthosis of the forestomach was observed in the female animals. The activity of palmitoyl CoA oxidase was not increased.

Other authors reported the induction of peroxisome proliferation in rats and mice after oral administration of the metabolite 2-ethylhexanol for 14 days (no other details; OECD 2010).

The carcinogenicity studies with the metabolite 2-ethylhexanol yielded a NOAEL of 50 mg/kg body weight and day in the rat and of 200 mg/kg body weight and day in the mouse. At the next-higher dose of 150 mg/kg body weight and day, reduced body weight gains and clinical symptoms such as a poor general condition, laboured breathing, piloerection or a urine-coated genital region were observed in the rat. In the mouse, reduced body weight gains (♂: –26%, ♀: –24%) and food consumption (♂: –9%, ♀: –12%), mortality (30%, see also Section 5.7), hyperplasia of the epithelium of the forestomach (5/50 ♂ and 4/50 ♀ compared with 1/50 ♂ and 1/50 ♀ in the control groups) and an increase in the relative liver weights (♀: 21%) and stomach weights (♂: 13%, ♀: 19%) were observed at doses of 750 mg/kg body weight and above (Astill et al. 1996; documentation "2-Ethylhexanol" 2003). The neoplastic findings are described in Section 5.7.

Daily acetic acid doses of 1800 mg/kg body weight over two weeks are regarded as the maximum tolerable dose for rats (see documentation "Acetic acid" 2010 a, a translation of the 2002 German documentation).

5.2.3 Dermal application

In a study from the 1960s, undiluted 2-ethylhexyl acetate (technical grade) doses of 1070 mg/kg body weight and day (corresponding to 535 mg/kg body weight/treatment) were applied non-occlusively to a 3 × 4 cm area of shaved skin of 12 female albino rats, twice a day, for 2 hours, on 10 of a total of 12 study days. The animals were immobilized during the exposure period and observed for a further 28 days. The skin and body weights of the treated animals and the six control animals were initially examined twice a week and later once a week. At the end of the study all

animals underwent gross-pathological examination. There were no significant differences between the treated animals and the controls. The NOAEL for systemic and local effects was found to be 1070 mg/kg body weight and day (no other details; ECHA 2012).

5.3 Local effects on skin and mucous membranes

5.3.1 Skin

2-Ethylhexyl acetate caused mild irritation of the skin in rabbits (no other details; OECD 2010).

In a study carried out according to OECD Test Guideline 404, 2-ethylhexyl acetate was found to cause skin irritation. Undiluted 2-ethylhexyl acetate (purity 99.1%, pH about 6) was applied semi-occlusively in amounts of 0.5 ml to the skin of 3 New Zealand White rabbits for 4 hours. The irritation index (determined after 24, 48 and 72 hours) was 2.11 for erythema and 0.0 for oedema on a scale up to a maximum of 4.0. The findings were not completely reversible within the 14-day recovery period (ECHA 2012).

Undiluted 2-ethylhexyl acetate (technical grade) doses of 1070 mg/kg body weight (corresponding to 535 mg/kg body weight/treatment) were applied ten times non-occlusively to the skin of rabbits (see Section 5.2.3), twice a day, for 2 hours; there were no gross-pathological changes to the skin. The NOAEL for local effects is 1070 mg/kg body weight and day (no other details; ECHA 2012).

2-Ethylhexanol caused irritation of the skin in rabbits with an irritation index of 6.75 on a scale with a maximum of 8.0 (documentation "2-Ethylhexanol" 2003).

The application of an 80% acetic acid solution to the skin of guinea pigs led to severe damage, 5% to 10% acetic acid solutions had no effect. In another study, 10% acetic acid applied to the intact and abraded skin of rabbits and guinea pigs caused mild irritation (documentation "Acetic acid" 2010 a, a translation of the 2002 German documentation).

To summarize, 2-ethylhexanol has somewhat stronger effects on the skin than 2-ethylhexyl acetate, while acetic acid causes much more severe skin irritation.

5.3.2 Eyes

2-Ethylhexyl acetate causes mild irritation of the eyes in rabbits (no other details; OECD 2010).

In a study carried out according to OECD Test Guideline 405, 2-ethylhexyl acetate did not cause eye irritation in 3 New Zealand White rabbits. Amounts of 0.1 ml 2-ethylhexyl acetate (purity 99.1%, pH about 6) were instilled into the conjunctival sac of the animals. The irritation index (determined after 24, 48 and 72 hours) was 0 for the cornea and chemosis on a scale up to a maximum of 4, 0 for the iris on a

scale up to 2, and 0.67 for the conjunctiva on a scale up to 3 (reversible within 72 hours) (ECHA 2012).

A Draize irritation index was reported for 2-ethylhexanol of 28.59 on a scale up to 110 (documentation "2-Ethylhexanol" 2003), which corresponds to moderate irritation.

Solutions with 10% acetic acid caused severe permanent eye damage in rabbits, 5% solutions caused damage which healed within 14 days (documentation "Acetic acid" 2010 a, a translation of the 2002 German documentation).

To summarize, 2-ethylhexanol causes somewhat stronger eye irritation than 2-ethylhexyl acetate, while acetic acid causes much more severe eye irritation.

5.4 Allergenic effects

There are no studies available with 2-ethylhexyl acetate, 2-ethylhexanol or acetic acid.

5.5 Reproductive and developmental toxicity

5.5.1 Fertility

There are no studies available with 2-ethylhexyl acetate, 2-ethylhexanol or acetic acid.

5.5.2 Developmental toxicity

There are no studies available with 2-ethylhexyl acetate.

As 2-ethylhexanol does not have toxic effects on development after either inhalation or dermal application, and effects on the offspring occur after oral administration only at maternally toxic doses, developmental toxicity is not to be expected after exposure to 2-ethylhexyl acetate (OECD 2010).

The inhalation of 2-ethylhexanol concentrations of 850 mg/m^3 (160 ml/m^3) from days 1 to 19 of gestation did not have any toxic effects on development in the foetuses of rats. 2-Ethylhexanol, with a MAK value of 10 ml/m^3, is classified in Pregnancy Risk Group C (supplement "2-Ethylhexanol" 2012, available in German only).

Acetic acid, with a MAK value of 10 ml/m^3, is likewise classified in Pregnancy Risk Group C (see supplement "Acetic acid" 2010 b, a translation of the 2008 German documentation).

5.6 Genotoxicity

5.6.1 In vitro

2-Ethylhexyl acetate did not induce any mutations in Salmonella typhimurium TA98, TA100, TA1535 and TA1537 or in Escherichia coli in concentrations of up to 5000 µg/plate either with or without the addition of a metabolic activation system (OECD 2010).

Also in a UDS test with primary rat hepatocytes, 2-ethylhexyl acetate (tested up to cytotoxic concentrations of 500 nl/ml) yielded negative results (ECHA 2012).

5.6.2 In vivo

There are no studies available with 2-ethylhexyl acetate.

In mice, 2-ethylhexanol did not induce an increase in the incidence of micronuclei in peripheral erythrocytes after single or two intraperitoneal doses of 456 mg/kg body weight (ECHA 2012; OECD 2010). In rats, no chromosomal aberrations in bone marrow cells were observed after oral 2-ethylhexanol doses of about 174 mg/kg body weight and day for 5 days (ECHA 2012).

2-Ethylhexanol was not mutagenic in dominant lethal tests with mice (no other details; OECD 2010).

Acetic acid did not cause somatic mutations and recombinations in Drosophila melanogaster in feeding studies (supplement "Acetic acid" 2010 b, a translation of the 2008 German documentation).

To summarize, 2-ethylhexanol is not genotoxic in vivo (see also documentation "2-Ethylhexanol" 2003), and therefore no genotoxic effects are expected for 2-ethylhexyl acetate in vivo, which yielded negative results in in vitro investigations. As a result of the few data available to date for the genotoxicity of acetic acid, it is not possible to make a conclusive evaluation; however, the only positive result in an in vitro chromosomal aberration test was attributed to the fact that the pH was not buffered.

5.7 Carcinogenicity

There are no studies available with 2-ethylhexyl acetate or acetic acid.

2-Ethylhexanol did not cause an increase in the incidence of tumours in long-term oral studies in male mice after doses of 750 mg/kg body weight and day or in rats after doses of 500 mg/kg body weight and day (documentation "2-Ethylhexanol" 2003).

In female mice, 2-ethylhexanol induced hepatocellular tumours at 750 mg/kg body weight and day. The incidence of hepatic basophilic foci was increased by 12% and that of hepatocellular carcinomas by 10%; compared with the incidence in the vehicle controls, but not with that in the water controls, the increase was statistically

significant. In addition to hyperplasia of the epithelium of the forestomach and the increase in relative liver and stomach weights (see Section 5.2.2), also reduced body weight gains (♂: –26%, ♀: –24%) and food consumption (♂: –9%, ♀: –12%), and increased mortality (30%) were observed at this dose, which indicates that the MTD was exceeded. The study was terminated in the high dose group after 78 weeks as a result of the early occurrence of mortality in order to guarantee the survival of more than 50% of the animals as stipulated by the test guidelines. The time-dependent and time-independent statistical analyses revealed an increasing trend in female and male mice for the incidence of hepatocellular carcinomas, which correlated with the toxicity (mortality) at the dose of 750 mg/kg body weight and day (Astill et al. 1996; documentation "2-Ethylhexanol" 2003).

6 Manifesto (MAK value/classification)

2-Ethylhexyl acetate is rapidly hydrolyzed to 2-ethylhexanol and acetic acid. Local irritation is the critical effect of 2-ethylhexanol and acetic acid, and the same should apply also for 2-ethylhexyl acetate.

MAK value. Studies with repeated inhalation or oral exposure to 2-ethylhexyl acetate are not available. Therefore, the data for the metabolites 2-ethylhexanol and acetic acid are used; a MAK value of 10 ml/m^3 has been established for both substances on the basis of studies with humans. 2-Ethylhexanol did not cause local irritation in volunteers up to concentrations of 15 ml/m^3. The irritation threshold for acetic acid is about 40 ml/m^3; the NOAEC for subjective sensory effects and objective local effects was found to be 10 ml/m^3.

There are no studies of local irritation after inhalation exposure to 2-ethylhexyl acetate; the data from a Draize test indicate that the local irritation is less pronounced that that after exposure to 2-ethylhexanol and acetic acid.

In analogy to its metabolites 2-ethylhexanol and acetic acid, a MAK value of 10 ml/m^3 has therefore been established also for 2-ethylhexyl acetate.

Peak limitation. 2-Ethylhexyl acetate has been classified in analogy to 2-ethylhexanol in Peak Limitation Category I with an excursion factor of 1. Acetic acid has a higher excursion factor of 2; as a result of insufficient data, this is, however, not justified for 2-ethylhexyl acetate.

Germ cell mutagenicity. 2-Ethylhexyl acetate was not found to be genotoxic in vitro; there are no data in vivo. The few data for genotoxicity available for acetic acid do not allow a conclusive evaluation to be made; however, the only positive result in an in vitro chromosomal aberration test was attributed to the fact that the pH was not buffered. As 2-ethylhexanol was not found to have genotoxic effects also in vivo (see documentation "2-Ethylhexanol" 2003), 2-ethylhexyl acetate is not

expected to have genotoxic effects. 2-Ethylhexyl acetate is therefore not classified in a category for germ cell mutagens.

Carcinogenicity. Studies of the carcinogenicity of 2-ethylhexyl acetate or acetic acid have not been carried out. The metabolite 2-ethylhexanol caused liver tumours in a long-term study with oral administration only at strongly toxic doses of 750 mg/kg body weight and day and only in female mice. No tumours were observed in the male mouse and in rats. 2-Ethylhexyl acetate does not have genotoxic effects in vitro. As 2-ethylhexanol was not found to be genotoxic also in vivo and the MTD was exceeded in the long-term study, liver toxicity seems to be responsible for the formation of tumours in female mice; if the MAK value, which protects against liver toxicity, is observed, liver tumours are excluded (see documentation "2-Ethylhexanol" 2003). A similar mechanism is to be expected for the carcinogenic effects of 2-ethylhexyl acetate. 2-Ethylhexyl acetate has therefore not been classified in a category for carcinogens.

Absorption through the skin. In comparison with the amount absorbed by inhalation after exposure for 8 hours at the level of the MAK value (710 mg, assuming 100% absorption), the maximum calculated amount absorbed through the skin of 68 mg is small, particularly in view of the fact that systemic toxicity is not the main effect. 2-Ethylhexyl acetate is therefore not designated with an "H" (for substances which can be absorbed through the skin).

Sensitization. There are no clinical findings for 2-ethylhexyl acetate available that indicate sensitizing effects on the skin or airways. There are also no studies with animals of the sensitizing effects of the substance. 2-Ethylhexyl acetate is therefore not designated with "Sa" or "Sh" (for substances which cause sensitization of the airways or skin).

Prenatal toxicity. There are no studies of the prenatal toxicity of 2-ethylhexyl acetate. Once again, the better investigated 2-ethylhexanol (see supplement "2-Ethylhexanol" 2012, available in German only) can be used for comparison, which, with a MAK value of 10 ml/m^3, is classified in Pregnancy Risk Group C. There are no relevant studies available for the evaluation of the prenatal toxicity of acetic acid. However, it can be expected that after inhalation exposure to acetic acid at the MAK value of 10 ml/m^3 (25 mg/m^3), prenatal toxicity does not occur. Acetic acid was classified in Pregnancy Risk Group C in analogy to the acetic acid esters methyl acetate, ethyl acetate, *n*-butyl acetate, octyl acetate and to ethanol, and assuming that the pH changes only very slightly with exposure at the level of the MAK value. In analogy to the metabolites 2-ethylhexanol and acetic acid, 2-ethylhexyl acetate is likewise classified in Pregnancy Risk Group C.

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