**n-Butyl acetate**

| Property                              | Value |
|---------------------------------------|-------|
| MAK value (1997)                      | 100 ml/m³ (ppm) ≈ 480 mg/m³ |
| Peak limitation (2000)                | Category I, excursion factor 2 |
| Absorption through the skin           | –     |
| Sensitization                         | –     |
| Carcinogenicity                       | –     |
| Prenatal toxicity (1999)              | Pregnancy risk group C |
| Germ cell mutagenicity                | –     |
| BAT value                             | –     |
| Synonyms                              | –     |
| Chemical name (CAS)                   | acetic acid butyl ester |
| CAS number                            | 123-86-4 |
| Structural formula                    | CH₃COO–CH₂(CH₂)₂–CH₃ |
| Molecular formula                     | C₆H₁₂O₂ |
| Molecular weight                      | 116.16 |
| Melting point                         | –77°C |
| Boiling point                         | 126°C |
| Density at 20°C                       | 0.88 g/cm³ |
| Vapour pressure at 20°C               | 13.3 hPa |
| log P<sub>ow</sub>                     | 1.82 |
| 1 ml/m³ (ppm) ≈ 4.82 mg/m³            | 1 mg/m³ ≈ 0.207 ml/m³ (ppm) |

The present documentation is based on reviews of the toxicological data for n-butyl acetate (ACGIH 1997, ECB 1995).

* *n*-octanol/water distribution coefficient
1 Toxic Effects and Mode of Action

Once absorbed by the organism or in contact with mucous membranes, \( n \)-butyl acetate is hydrolysed to \( n \)-butyl alcohol and acetic acid. The acute toxicity of \( n \)-butyl acetate is low after oral and dermal absorption and inhalation exposure to the vapour. After high concentrations, irritation of the eyes and respiratory passages and central nervous and narcotic effects are observed. The substance does not affect rabbit skin, but has weak irritative effects on the rabbit eye. \( n \)-Butyl acetate does not cause sensitization in man or animals.

The critical effect for man is irritation of the eyes and upper respiratory tract which begins from concentrations of about 200 ml/m\(^3\). The few data available from experiments with animals suggest that the toxicity of the substance after repeated exposure is also low; again, irritation is the main effect. \( n \)-Butyl acetate is not mutagenic in prokaryotes and does not induce chromosomal aberrations in mammalian cells. Studies of the carcinogenic effects of the substance are not available.

Exposure of pregnant rats to the maternally toxic concentration of 1500 ml/m\(^3\) leads in the offspring to reduced body sizes and in some cases to skeletal variations. Malformations do not occur. In rabbits this treatment has no effects on the parent animals, but in the offspring the incidence of morphological variations is increased.

2 Mechanism of Action

There are no data available for the mechanism of action of \( n \)-butyl acetate. The irritative effects may be caused by the acetic acid formed during hydrolysis.

3 Toxicokinetics and Metabolism

The rate of absorption of ingested or inhaled \( n \)-butyl acetate is not known. \( n \)-Butyl acetate does not readily penetrate the skin. With isolated human skin, a permeability constant of 1.6 ± 0.1 g/m\(^2\) and hour was determined (Ursin et al. 1995). From the distribution coefficients liver/air, muscle/air and fat/air for \( n \)-butyl acetate of 281, 157 and 900 determined at 37°C with rat tissue (Poulin and Krishnan 1996) it can be concluded that the absorbed substance is distributed in the whole body with the greatest accumulation in adipose tissue. In persons exposed to \( n \)-butyl acetate concentrations of 200 mg/m\(^3\), the \( n \)-butyl acetate concentration in the exhaled air was found to be 100 mg/m\(^3\) (ACGIH 1997).

After contact with mucous membranes and absorption by the organism, butyl acetates may be assumed to be hydrolysed to the corresponding butyl alcohols and acetic acid in
reactions catalysed by various esterases; this was demonstrated with isolated nasal tissue from the rat. The rate of hydrolysis decreased with increasing branching of the butyl residue and was between 42 and 77 nmol/mg protein and minute. This can be explained by the increasing steric hindrance (Dahl et al. 1987). Exposure of rats for 5 hours to n-butyl acetate concentrations of 1000 ml/m$^3$ via tracheal cannula led to rapid equilibration at a blood level of 25 µM. During the first 30 minutes of exposure the level of n-butyl alcohol in the blood increased to 52 µM and then remained constant (Groth and Freundt 1991, 1994). In male Sprague-Dawley rats given intravenous injections of $^{14}$C-labelled n-butyl acetate (about 30 mg/kg body weight) the elimination half-time from the blood was 0.4 minutes. During the first 2.5 minutes after administration the substance could also be detected in the brain. The maximum level of n-butyl alcohol could be detected in the blood and brain 2.5 minutes after administration. Somewhat later, but only in the blood, also butyraldehyde and polar metabolites (probably conjugates of n-butyl alcohol with sulfuric acid or glucuronic acid and intermediates from the citric acid cycle) were found. The elimination half-time for n-butyl alcohol (1–1.2 minutes) was also short. 20 minutes after the administration of n-butyl acetate no more n-butyl alcohol could be detected (CMA 1997). In an earlier study, 4-hydroxy-3-methoxymandelic acid was shown to be the main metabolite in the urine of mammals (no other details). It was shown later, however, that this substance is excreted with the urine also under normal or pathological conditions, which is the reason it is not clear whether 4-hydroxy-3-methoxymandelic acid is a true butyl acetate metabolite (ACGIH 1997, ECB 1995).

4 Effects in Man

Workers repeatedly exposed to n-butyl acetate and other substances complained of irritation of the eyes, nose and throat, and tightness of the chest. Very high concentrations (no other details) led also to weakness, dizziness and unconsciousness. As the exposure was always to a mixture of solvents, the symptoms cannot be attributed exclusively to n-butyl acetate. Repeated contact with the liquid substance led to defatting and slight irritation of the affected area of skin (ACGIH 1997, ECB 1995).

In a controlled study, 10 volunteers were exposed to n-butyl acetate for 2 to 5 minutes. At concentrations of 200 ml/m$^3$ most of the persons complained of throat irritation, at 300 ml/m$^3$ the symptoms given were irritation of the eyes and nose and severe throat irritation (Nelson et al. 1943). Exposure for 5 minutes in a room in which 3 minutes previously n-butyl acetate had been sprayed and then distributed evenly, led at the low concentration of 1000 mg/m$^3$ (210 ml/m$^3$) to slight eye, nose and throat irritation, and to very mild irritation of the airways. At the high concentration of 10000 mg/m$^3$ (2100 ml/m$^3$), a moderate irritation score was given for all symptoms (Flury and Wirth 1933). Persons who were exposed for a short time (no other details) to concentrations of 3300, 7000 and 14000 ml/m$^3$, reported a strong, unpleasant odour and irritation of the eyes and nasal mucosa which was more severe with increasing concentration. The odour
A threshold for \( n \)-butyl acetate was given as 0.39 ml/m\(^3\) (ACGIH 1997). In another inhalation study with controlled exposure in a chamber, 24 healthy non-smokers were exposed to \( n \)-butyl acetate according to 3 different exposure patterns. The irritative effects were evaluated on the basis of the subjective feeling of irritation, clinical examination of eye irritation and the lung function. In the first experiment the persons were exposed for 20 minutes to concentrations of 74, 147, 221 and 295 ml/m\(^3\). Evaluation of the irritative effects by the exposed group and the not exposed control group revealed no significant differences. In the second experiment one group was exposed for 20 minutes to \( n \)-butyl acetate concentrations of 15 ml/m\(^3\), the other group to 295 ml/m\(^3\). The group exposed to the low concentration was regarded as the control group. The authors later judged this concentration, however, to be too high for a control exposure. The persons from the high concentration group gave their symptoms of irritation a significantly higher score than did the control group, although the irritation was not felt to be severe. An unpleasant odour was described only by the control group. In the third experiment the persons were exposed for 4 hours to concentrations of 15 ml/m\(^3\) (controls) and 147 ml/m\(^3\). In the high concentration group, throat irritation, breathing difficulties and the perception of an unpleasant odour were significantly more marked than in the control group. Red eyes were observed in 50% of the persons exposed to the high concentration and in 17% of the control persons. Nevertheless, the authors concluded from these investigations that \( n \)-butyl acetate has only a low irritative potential (Iregren \textit{et al.} 1993). For exposure to \( n \)-butyl acetate for about 2 seconds a threshold value of 3650 ml/m\(^3\) was given for nasal irritation in anosmic persons (Abraham \textit{et al.} 1996).

Unexposed persons and persons with solvent-induced toxic encephalopathy were exposed for 2 hours to increasing \( n \)-butyl acetate concentrations (3–50 ml/m\(^3\)). The persons of the second collective were mostly significantly more sensitive to the odour, taste, unpleasant effects, irritation and tiredness associated with the exposure than were the healthy persons (Ørbæk \textit{et al.} 1998). As there was no group exposed only to air, it is not possible to make a statement about the irritation threshold of \( n \)-butyl acetate.

A 4% \( n \)-butyl acetate preparation in petrolatum did not cause skin irritation after occlusive application for 48 hours. Nail varnish containing 25.5% \( n \)-butyl acetate was not found to have irritative effects after repeated occlusive application to the skin. Also after repeated application of the nail varnish to the nails, no irritative effects were seen on the nails, cuticle or skin. In an accident at work, butyl acetate caused corneal corrosion. After removal of the damaged epithelium down to Bowman’s membrane, the cornea healed within 48 hours (no other details) (ACGIH 1997, ECB 1995).

Several maximization tests with \( n \)-butyl acetate did not lead to sensitization. Induction was carried out by repeated occlusive application of 0.5 ml of the pure substance, of 4% \( n \)-butyl acetate in petrolatum or of nail varnish containing 25.5% \( n \)-butyl acetate. Provocation took place 10 days after the last induction treatment. A patch test with 5% \( n \)-butyl acetate in olive oil produced positive results in one worker who had occupational contact with \( n \)-butyl acetate and other substances and suffered from dermatitis on the hands, arms and face. In 36 control persons the results were negative. Of 149 patients with dermatitis, one produced a positive reaction to \( n \)-butyl acetate (ACGIH 1997, ECB 1995).
5 Animal Experiments and *in vitro* Studies

5.1 Acute toxicity

5.1.1 Inhalation

The symptoms observed after single exposures to *n*-butyl acetate were irritation of the eyes and respiratory tract; with high concentrations severe lung damage was observed (haemorrhage, congestion, oedema) and was regarded as the cause of death. In addition, there were central nervous and narcotic effects.

In two studies with rats, inhaled *n*-butyl acetate was found to be of unexpectedly high toxicity with 4-hour LC$_{50}$ values of 160 and 391 ml/m$^3$. This was attributed to the high fraction of the substance present as an aerosol in the test atmosphere, but could not be reproduced in other studies with *n*-butyl acetate aerosol. The acute inhalation toxicity of *n*-butyl acetate vapour is low. For rats exposed for 4 hours, an LC$_{50}$ value of 2000 ml/m$^3$ was determined. In other studies carried out according to OECD guideline 403 the 4-hour LC$_{50}$ values were all over 4000 ml/m$^3$. All rats survived exposure for 4 hours to an atmosphere more or less saturated with *n*-butyl acetate (about 10000 ml/m$^3$) and exposure for 6 hours to 1850 ml/m$^3$. All animals died after exposure for 8 hours to an atmosphere saturated with *n*-butyl acetate and exposure for 6 hours to 14050 ml/m$^3$ (ACGIH 1997, ECB 1995). After exposure of rats for 6 hours to *n*-butyl acetate concentrations of 1500, 3000 and 6000 ml/m$^3$ no deaths occurred. The clinical and gross pathological examinations after autopsy did not yield unusual findings. Particular consideration was given to possible neurotoxic effects. In the two high concentration groups the average motor activity was reduced and some behavioural parameters changed on the day of exposure. On days 1, 7 and 14 after the end of exposure such anomalies could no longer be detected. The animals of all concentration groups seemed to be slightly sedated during the exposure and reacted more slowly to external stimuli (CMA 1994).

Mice survived exposure to 5900 ml/m$^3$ for 6 hours and to 7350 ml/m$^3$ for 3.6 hours. In an earlier investigation an LC$_{50}$ value of 1260 ml/m$^3$ was found for mice (ACGIH 1997, ECB 1995). Exposure of mice for 20 minutes to *n*-butyl acetate led from concentrations of 8000 ml/m$^3$ to reduced motor activity, and numerous parameters investigated in a behavioural test battery were changed relative to the control values. All symptoms were reversible within a few minutes after the end of exposure (Bowen and Blaster 1997). In Swiss-OF1 mice, the concentration which reduced the respiration rate by 50% (RD$_{50}$ value) after exposure for several minutes was found to be 730 ml/m$^3$. The concentration–effect relationship was not given (Muller and Greff 1984).

The lowest lethal concentration for guinea pigs after exposure for 4 hours was 14000 ml/m$^3$ and for cats after exposure for 1.2 hours was 14300 ml/m$^3$ (ACGIH 1997, ECB 1995).
5.1.2 Ingestion and dermal absorption

The acute oral toxicity of \( n \)-butyl acetate is also low. For the rat, the \( LD_{50} \) values were in the range from 10700 to 14130 mg/kg body weight. A value of 7060 mg/kg body weight was determined for the mouse. For the rabbit, in two different studies \( LD_{50} \) values of 7440 and 3200 mg/kg body weight were obtained. The dermal \( LD_{50} \) value for the rabbit was found to be over 17600 mg/kg body weight. In guinea pigs, occlusive application of \( n \)-butyl acetate doses of up to 10 ml/kg body weight (8800 mg/kg body weight) for 24 hours was not lethal (ACGIH 1997, ECB 1995).

5.2 Subacute, subchronic and chronic toxicity

Groups of 10 male and 10 female Sprague-Dawley rats were exposed to \( n \)-butyl acetate concentrations of 500, 1500 or 3000 ml/m\(^3\) for 6 hours a day over 14 weeks for a total of 65 days. Specific investigations to evaluate the neurotoxic potential of the substance were carried out. The results of tests for functional behaviour, motor activity and operant behaviour were not unusual at the lowest concentration. At concentrations of 1500 and 3000 ml/m\(^3\) there were transient signs of sedation and hypoactivity. Motor activity was significantly increased in the male animals of the high concentration group only in week 4 of the study, but not in weeks 8 or 13. Histological examination of the central and peripheral nervous systems did not reveal any differences between the control group and the 3000 ml/m\(^3\) group. Other organs were not investigated. The only sign of systemic toxicity was reduced body weights from concentrations of 1500 ml/m\(^3\) in the female animals and from 3000 ml/m\(^3\) in the male animals (David et al. 1998). From this study, a NOEL (no observed effect level) of 500 ml/m\(^3\) can be deduced for rats, at least for neurotoxic effects.

The following data for toxicity after repeated exposure are from studies carried out over 60 years ago.

Mice tolerated without symptoms exposure for 4 days (17 hours/day) to an atmosphere saturated with \( n \)-butyl acetate. Cats were exposed to concentrations of 1600, 3100 or 4200 ml/m\(^3\), 6 hours/day for 6 days. At 1600 ml/m\(^3\) increased salivation and slight eye irritation were observed. At 3100 ml/m\(^3\) morphological changes in the blood cells were detected, and after 4200 ml/m\(^3\) the animals appeared weak, body weights were reduced and slight irritation of the airways was observed. In guinea pigs exposed to \( n \)-butyl acetate concentrations of 1000 ml/m\(^3\), 6 hours daily on 6 days weekly for 5 weeks, neither urine and blood analyses nor autopsy revealed pathological findings (ACGIH 1997, ECB 1995).

5.3 Local effects on skin and mucous membranes

0.01 ml \( n \)-butyl acetate applied for 24 hours to the rabbit skin produced effects which were given an irritation index of 1 (maximum value 10), and thus the substance was classified as not irritative. Also after occlusive application of 0.5 ml \( n \)-butyl acetate to rabbit
skin for 4 hours no signs of irritation were observed. Occlusive application of \( n \)-butyl acetate doses of up to 10 ml/kg body weight for 24 hours to the depilated abdominal skin of guinea pigs was not found to have irritative effects. The observation period was 2 weeks (ACGIH 1997, ECB 1995).

In an earlier study of the irritative effects of \( n \)-butyl acetate on the rabbit eye, severe, yet reversible corrosion of the cornea was described after the instillation of 0.005 ml into the conjunctival sac. The irritative effects were given a score of 5 on a scale with a maximum value of 10. On the other hand, in several more recent studies the substance had only weak irritative effects or was not irritating to the eye when applied for 24 hours. The effects of 0.1 ml \( n \)-butyl acetate were given an irritation index of 8 on a scale with a maximum value of 110. With a 30 % solution an irritation index of 11 was obtained, with a 10 % solution an irritation index of 19 (ACGIH 1997, ECB 1995).

5.4 Allergenic effects

In the maximization test with guinea pigs and the mouse ear swelling test, \( n \)-butyl acetate was not found to have sensitizing effects (ACGIH 1997, ECB 1995).

5.5 Reproductive and developmental toxicity

The reproductive toxicity of \( n \)-butyl acetate was investigated with Sprague-Dawley rats (37–43 animals/group) exposed to 1500 ml/m\(^3\) for 7 hours daily. The animals from group 1 served as controls and were exposed to filtered air according to the exposure pattern of group 4 (see below). The animals from group 2 were exposed from days 7 to 16 of gestation, those from group 3 from days 1 to 16. The animals from group 4 were exposed during the 3 weeks before mating on 5 days per week, then mated with untreated male animals and exposed from days 1 to 16 of gestation. In all groups food consumption, and as a result body weights, were reduced at the end of the study, an indication of maternal toxicity. The histological examination of the dams did not reveal any pathological findings. The course and success of gestation were not affected in any group. The length of the foetuses was reduced in all groups. Whether this is the result of effects of \( n \)-butyl acetate on the dams or a direct effect on the foetuses cannot be determined. The incidence of abnormally formed ribs (wavy, fused, branched) was significantly increased in the foetuses of groups 2 and 3, but not in the foetuses of group 4. Extended urethras were found in an increased number only in groups 3 and 4, but not in group 2. As these findings are generally regarded as variations and not as malformations and they did not occur equally in all groups, they are not to be seen as teratogenic effects (NIOSH 1982).

In another test, artificially inseminated New Zealand White rabbits (21–25 animals per group) were investigated. The rabbits were exposed to \( n \)-butyl acetate concentrations of 1500 ml/m\(^3\) from days 7 to 19 of gestation (group 2) or from days 1 to 19 (group 3) for 7 hours daily. The control animals (group 1) were exposed to filtered air according to the exposure pattern of group 3. Food consumption was reduced in the treated groups; body weights, however, corresponded with those of the controls up to the end of the
study. The course and success of gestation were not affected in any group. The histological examination of the dams did not reveal any pathological findings. In the offspring of the two treated groups no malformations were detected. The increase in the incidence of morphological variations of the gall bladder and of other anomalies (retinal folds, unsymmetrically fused breastbone) was statistically significant only in the offspring of group 3. In the opinion of the authors n-butyl acetate had no significant influence on the development of the foetuses (NIOSH 1982).

In rats, the n-butyl acetate metabolite n-butyl alcohol had no effects on the offspring and dams up to concentrations of 3500 ml/m³ (see “n-Butyl alcohol” in this volume). For the other metabolite, acetic acid, the effective principle of any prenatal toxicity could be acidosis. However, exposure of rabbits for over 2 hours even to very high concentrations of various alkyl acetate esters (e.g. n-propyl acetate concentrations of 54000 mg/m³, corresponding to 13000 ml/m³) did not produce such effects (Flury and Wirth 1933), so that in the range of the MAK value for n-butyl acetate germ cell mutagenicity resulting from acetic acid is not to be expected. This is confirmed by a study with rabbits given cider vinegar in doses of 1600 mg/kg body weight from days 6 to 18 of gestation. Assuming an acetic acid content of 5 %, this corresponds to acetic acid doses of 80 mg/kg body weight. Neither maternal toxicity nor foetal toxicity, nor an increased incidence of malformations in the foetuses were observed (FDA 1977).

5.6 Genotoxicity

n-Butyl acetate was not found to be mutagenic in the Salmonella typhimurium strains TA92, TA94, TA98, TA100, TA1535, TA1537 or TA1538, or in Echerichia coli WP2 uvr either with or without an added metabolic activation system. The highest tested concentration was 10000 µg/plate. With Saccharomyces cerevisiae D61.M, the substance did not induce mitotic aneuploidy up to the highest concentration tested of 4 mg/ml.

Tests for chromosomal aberrations in CHL cells (a cell line derived from Chinese hamster liver) yielded negative results up to the highest concentration tested of 2 mg/ml (ACGIH 1997, ECB 1995).

There are no data available for the carcinogenicity of n-butyl acetate.
6 Manifesto (MAK value/classification)

For the workplace situation, the critical effects of \( n \)-butyl acetate are the irritative effects on the eyes, nasal mucosa and throat. The studies of the threshold concentration in man do not give consistent results. Symptoms are described by 2 authors after a maximum of 5 minutes exposure to 200 ml/m\(^3\). At 300 ml/m\(^3\) throat irritation was described as severe. On the other hand, the symptoms were not evaluated differently by the persons exposed for 20 minutes to concentrations of 74 to 295 ml/m\(^3\) than by the control persons. Yet in the same study, volunteers exposed for 4 hours to 147 ml/m\(^3\) reported symptoms of irritation, in particular throat irritation, significantly more often.

It was concluded from these findings that the MAK value for \( n \)-butyl acetate which applied until 1999 (200 ml/m\(^3\)), and in particular the peak exposure value of 400 ml/m\(^3\), did not provide adequate protection against the irritative effects of the substance. The MAK value was therefore reduced to 100 ml/m\(^3\). In the study of Iregren et al. (1993), important for determining the excursion factor, no irritative effects occurred at concentrations of 221 ml/m\(^3\) for 20 minutes. \( n \)-Butyl acetate is therefore classified in Peak limitation category I with an excursion factor of 2.

Systemic-toxic effects are not to be expected if the MAK value is observed. A medium-term inhalation study with rats yielded a NOEL of 500 ml/m\(^3\). It must, however, be pointed out that mainly neurotoxic parameters were investigated. The systemic toxicity of \( n \)-butyl acetate can also be estimated by evaluating the systemic toxicity of the metabolites. For \( n \)-butyl alcohol, the MAK value is also 100 ml/m\(^3\), a concentration at which no systemic effects occur (see “\( n \)-Butyl alcohol” in this volume). The amount of acetic acid taken up daily during exposure at the level of the MAK value, at most 2.5 g, is of the order of magnitude of the average amount taken up with food (Guest et al. 1982). In addition, acetic acid is formed endogenously and is produced in numerous pathways of intermediary metabolism, so that no adverse effects are expected as a result of the additional occupational exposure.

The maternally toxic concentration of 1500 ml/m\(^3\) was also foetotoxic in rats, but not teratogenic. Concentrations not toxic for the dams were not tested; it is therefore not possible to use this study to evaluate the potential reproductive toxicity of \( n \)-butyl acetate. However, the metabolite \( n \)-butyl alcohol also did not have effects on the offspring of rats exposed at the concentration 3500 ml/m\(^3\), which was not toxic for the dams. Given observation of the MAK value for \( n \)-butyl acetate, germ cell mutagenicity is not to be expected from acetic acid, the other metabolite (see Section 5.5). In rabbits, \( n \)-butyl acetate concentrations of 1500 ml/m\(^3\) were not maternally toxic and led in the offspring merely to an increase in the incidences of various variations not regarded as adverse. There is thus no reason to fear a risk of damage to the embryo or foetus when the MAK value is observed, and \( n \)-butyl acetate has been classified in Pregnancy risk group C.

Because of the high dermal LD\(_{50}\) value in rabbits and the poor skin penetration of \( n \)-butyl acetate, the substance is not designated with an “\( H \)”.

\( n \)-Butyl acetate was not found to have sensitizing effects in controlled investigations with man and animals, so that despite the two case reports with positive results the substance has not been designated with an “\( S \)”. 
7 References

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