HERA

Human and Environmental Risk Assessment on Ingredients of Household Cleaning Products

Sodium carbonate

(CAS No. 497-19-8)

Edition 2.0

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Executive Summary

Sodium carbonate (soda ash) is used as a builder in detergent powders and tablets for water softening in the washing process. Sodium carbonate is also used in laundry additives, machine dishwashing products, surface cleaners, toilet cleaners and other household cleaning products. The product sodium carbonate is available for consumers and solutions of sodium carbonate in water have been used in the past for soaking of clothes, dishwashing, floor washing, degreasing operations and for personal care. These applications may still occur. The amount of sodium carbonate, which is used in household cleaning products in Europe, is estimated to be 550,000 tonnes per year.

After use of the household cleaning product, the water (containing the sodium carbonate) will be disposed via the drain. However, the carbonate will not be discharged to aquatic ecosystems but will be neutralized in the waste water treatment plant. Sodium has a low toxicity and the emitted amount of sodium is relatively low compared to background concentrations and therefore the emitted amount of sodium will not have an effect on the aquatic organisms of the receiving water.

The available acute ecotoxicity data with fish and water fleas revealed LC50 values which were higher than 68 mg/l. The increase in pH of the receiving water was used to obtain an idea of the acceptable amount of sodium carbonate which can be added to aquatic ecosystems. Depending on the buffer capacity of the aquatic ecosystem, an estimate of the acceptable amount ranges between 2 and 20 mg/l.

When humans are exposed to sodium carbonate, via the use of household cleaning products, the concentration of sodium in the blood and the pH of the blood will not be increased and therefore the exposure to sodium carbonate will not increase the normal physiological levels of sodium and carbonate/bicarbonate. Therefore there is no concern about a possible systemic toxic effect after short term or repeated exposure to the substance. No genotoxic effects in bacteria or teratogenic effects in rabbits, rats and mice have been reported. The only critical endpoint for sodium carbonate seems to be local irritation.

Consumers will be exposed to sodium carbonate due to direct skin contact with solutions which contain sodium carbonate, which can be laundry hand washing or use of a carbonate solution for personal care (e.g. skin treatment). However, the concentrations of sodium carbonate in these solutions are too low to cause local irritation.

Accidental or intentional overexposure to sodium carbonate may occur via oral uptake or via exposure of the eyes (e.g. due to splashing). The available animal data do not indicate severe adverse effects when accidental or intentional overexposure to sodium carbonate occurs. Although sodium carbonate is widely available for consumers and has been used for a long time, cases of oral poisoning or effects on human eyes have not been reported in the literature. Apparently accidental or intentional overexposure to sodium carbonate does not result in severe adverse effects on humans or does simply not occur.

Based on the available data, the use of sodium carbonate in household cleaning products has neither an adverse effect on the aquatic ecosystem nor an adverse effect on consumers.

Contents

This report has been prepared by Solvay S.A., Brussels, Belgium.

1. Introduction

1.1 Identity and physical/chemical properties

Sodium carbonate (soda ash) is a white, crystalline hygroscopic powder with a purity >99.5 % calculated on the anhydrous form. There are two forms of sodium carbonate available, light soda and dense soda. Impurities of sodium carbonate may include sodium chloride, sodium sulphate, calcium carbonate, magnesium carbonate, sodium bicarbonate and iron. The impurity profile depends on the production process and the composition of the raw materials.

Sodium carbonate has a melting point of 851°C (CRC Handbook, 1986; The Merck Index, 1983). When it is heated it decomposes and therefore a boiling point can not be determined. Its density is 2.532 (20°C) and its water solubility is 71 g/l water at 0°C, 215 g/l water at 20°C and 455 g/l water at 100°C (CRC Handbook, 1986). Determination of octanol water partition coefficient (log Pow) and vapour pressure are not applicable as sodium carbonate is an ionizable inorganic compound which has a melting point above 360°C and the boiling point cannot be measured due to decomposition. The average particle size diameter of light sodium carbonate is in the range of 90 to 120 µm and of dense sodium carbonate is in the range of 250 to 400 µm.

Table 1: Identity and physical/chemical properties of sodium carbonate

Property	**Results / Remarks**	**Reference**
Molecular formula	Na_2CO_3	
Molecular Weight	106	
CAS number	497-19-8	
EINECS number	207-838-8	
Particle size	Light sodium carbonate: 90 – 120 µm Dense sodium carbonate: 250 – 400 µm	
Melting Point	851 °C	CRC Handbook (1986), The Merck Index (1983)
Water Solubility	71 g/l at 0 °C 215 g/l at 20 °C 405 g/l at 100 °C	CRC Handbook (1986)
Density	2.532 kg/m^3 at 20 °C	CRC Handbook (1986)
pK_b	3.75 at 25 °C	Binas (1986)

Sodium carbonate is a strong alkaline compound with a pH of 11.6 for a 0.1M aqueous solution (The Merck Index, 1983; Johnson and Swanson, 1987). The pK_b of CO_3^{2-} is 3.75, which means that at a pH of 10.25 both carbonate and bicarbonate are present in equal amounts. The identity and several physical/chemical properties are summarized in Table 1.

1.2 Production

Sodium carbonate is produced at many different sites all over the world and there are about 12 production sites in Europe.

Sodium carbonate can be produced from minerals which contain sodium carbonate. It is present in large deposits in Africa and the United States as either carbonate or trona, a mixed ore of equal molar amounts of the carbonate and bicarbonate.

However, about 70 % of the world production capacity of sodium carbonate is manufactured by the Solvay (ammonia soda) process, whereby ammonia is added to a solution of sodium chloride. Carbon dioxide is then bubbled through to precipitate the bicarbonate, $NaHCO_3$. The sodium bicarbonate is decomposed by heat producing sodium carbonate. The traditional Solvay process is utilised in most parts of the world, with the exception of the U.S., where all production is based on the minerals which contain sodium carbonate.

Different qualities of the sodium carbonate are produced based on the final use of the substance (Morrin, 2000; Clayton and Clayton, 1993). Industrial, food and pharmaceutical grades are placed on the market.

1.3 Use

The main user of sodium carbonate is the glass industry, which used 51 % of the world sodium carbonate demand in 1999 (Morrin, 2000). About 10 % of the world sodium carbonate demand is used for detergents.

Sodium carbonate (soda ash) is used as a builder in detergent powders and tablets for water softening in the washing process. Sodium carbonate is also used in laundry additives, machine dishwashing products, surface cleaners, toilet cleaners and other household cleaning products. For most products the typical sodium carbonate content is less than 30 %, but maximum concentrations can be higher (up to 90 %).

The product sodium carbonate is also available for consumers. Solutions of sodium carbonate in water have been used in the past for soaking of clothes, dishwashing, floor washing, degreasing operations and for personal care. These, and other domestic applications, may still occur.

The amount of sodium carbonate, which was used in household cleaning products in Europe, was estimated to be 550,000 tonnes in 1999. This estimate was provided by the SBU Soda Ash of Solvay, which is a producer of sodium carbonate. This figure has been confirmed by the formulator companies (producers of household cleaning products).

2. Environmental Assessment

2.1 Environmental exposure assessment

The household cleaning products, which contain sodium carbonate, are normally diluted with tap water during use. For example a hard surface cleaner is diluted with water before the cleaning process starts and detergents are dissolved in water during the laundry washing process. After use the water will be disposed via the drain and finally it will be discharged to aquatic ecosystems (e.g. rivers, lakes, estuaries, sea) after a treatment.

2.1.1 Environmental Fate

In water, sodium carbonate dissociates into sodium and carbonate and both ions are not expected to adsorb significantly to sediment. Furthermore there is no distribution or transport to the atmosphere of these ions and therefore the environmental risk assessment can be focussed on the aquatic compartment. An emission of sodium carbonate to water will result in an increase in alkalinity and a tendency to raise the pH value. The carbonate ions will react with water, resulting in the formation of bicarbonate and hydroxide, until an equilibrium is established (McKee and Wolf, 1963).

Both sodium and inorganic carbon are ubiquitously present in the environment. The concentrations of CO_2, HCO_3^- and CO_3^{2-} in aquatic ecosystems are regulated by the following equilibria:

$HCO_3^- \leftrightarrow CO_3^{2-} + H^+$ pKa = 10.25 at 25ºC

$CO_2 + H_2O \leftrightarrow HCO_3^- + H^+$ pKa = 6.36 at 25ºC

Only a small fraction of the dissolved CO_2 is present as H_2CO_3, the major part is present as CO_2. The amount of CO_2 in water is in equilibrium with the partial pressure of CO_2 in the atmosphere. The CO_2 / HCO_3^- / CO_3^{2-} equilibria are the major buffer of the pH of freshwater throughout the world. Based on the above equations, CO_2 is the predominant species at a pH smaller than 6.36, while HCO_3^- is the predominant species at a pH in the range of 6.36-10.25 and CO_3^{2-} is the predominant species at a pH higher than 10.25.

The natural concentration of CO_2 / HCO_3^- / CO_3^{2-} in freshwater is influenced by geochemical and biological processes. Many minerals are deposited as salts of the carbonate ion and for this reason the dissolution of these minerals is a continuous source of carbonate in freshwater. Carbon dioxide is produced in aquatic ecosystems from microbial decay of organic matter. On the other hand plants utilise dissolved carbon dioxide for the synthesis of biomass (photosynthesis). Because many factors influence the natural concentration of CO_2 / HCO_3^- / CO_3^{2-} in freshwater, significant variations of the concentrations do occur.

As indicated before, the emission of sodium carbonate to the aquatic environment will increase the pH of the water. To underline the importance of the buffer capacity, a table is included with the concentration of sodium carbonate needed to increase the pH to a value of 9.0, 10.0 and 11.0 at different bicarbonate concentrations. The initial pH of a bicarbonate solution with a concentration of 15 – 233 mg/l is 8.3 (calculated). The data of Table 2 were based on calculations but they were confirmed by experimental titrations (De Groot et al., 2002).

Table 2: Concentration of sodium carbonate (mg/l) needed to increase the pH to values of 9.0, 10.0 and 11.0 (De Groot et al., 2002).

Buffer capacity[A]	Final pH[B]		
	9.0	10.0	11.0
0 mg/l HCO_3^- (distilled water)	1.1 (0.6)	16 (6.1)	603 (61)
15 mg/l HCO_3^- (10^{th} percentile of 21 European rivers)	2.3 (16)	28 (21)	725 (76)
128 mg/l HCO_3^- (mean value of 21 European rivers)	12 (129)	120 (134)	1646 (189)
233 mg/l HCO_3^- (90^{th} percentile of 21 European rivers)	20 (234)	206 (239)	2502 (294)

[A] The initial pH of a bicarbonate solution with a concentration of 15 – 233 mg/l is 8.3 (calculated).
[B] Between brackets the final concentration of bicarbonate is given.

2.1.2 Monitoring Data

Normally the pH in aquatic ecosystems is significantly less than 10.3 and therefore carbonate is present in very low concentrations in aquatic ecosystems, which explains why monitoring data are not available for carbonate. However, for bicarbonate many monitoring data are available. An overview of the bicarbonate concentration in world river basins has been published by UNEP (1995). The concentration was measured in a total number of 21 rivers in Europe. These 21 rivers were representative for the large European rivers (e.g. Danube, Elbe, Rhine and Seine were included). The 10^{th} –percentile, mean and 90^{th}-percentile were 15, 128 and 233 mg/l, respectively. The concentrations of the bicarbonate ion were strongly related to Ca^{2+} concentrations which reflect the weathering of rocks. The distribution of bicarbonate followed therefore the same pattern as that of the Ca^{2+} ion.

The sodium ion is ubiquitously present in the environment and it has been measured extensively in aquatic ecosystems. Sodium and chloride concentrations in water are tightly linked. They both originate from natural weathering of rock and from atmospheric transport of oceanic inputs and from a wide variety of anthropogenic sources. The anthropogenic sources of sodium are so pervasive that the concentrations of sodium in water have risen by a factor of 10 to 20 in many rivers in the 20^{th} century. The sodium concentration was reported for a total number of 21 rivers in Europe, with a 10^{th} percentile of 1.9 mg/l, mean of 56 mg/l and 90^{th} percentile of 92 mg/l (UNEP, 1995).

2.1.3 Exposure assessment

To evaluate the potential effect of sodium carbonate on the aquatic organisms, the concentration of sodium carbonate in the receiving water (an aquatic ecoystem) must be determined. In other words, the Predicted Environmental Concentration (PEC) must be determined to know the exposure of the aquatic organisms to sodium carbonate.

To estimate the PEC, computer models can be used. In the European Union the model EUSES has been used to calculate the PEC of organic compounds (Vermeire et al., 1994). In some cases it can also be used for inorganic compounds to obtain a preliminary idea about the order of magnitude of the PEC. Within HERA the EUSES model has been adapted to develop a specific scenario for detergents (HERA, 2001). The HERA detergent scenario revealed a $PEC_{regional.added}$ and a $PEC_{local.added}$ of 1.7 and 5.7 mg sodium carbonate per liter, respectively. The PEC_{added} has been calculated using a tonnage of 550,000 t/y and assuming that the compound is inert. The PEC_{added} is the calculated concentration of sodium carbonate which is added to the background (ambient) concentration of the substance.

For transparency reasons three files have been included in this document:
1. NaCO3_1.exf: The EXF.file for a standard EU scenario (according to TGD),
2. NaCO3_2.exf: The EXF.file for the HERA detergent scenario and
3. Excel 2.x chart: the input file for the HERA detergent scenario.


naco3_1.exf


NaCO3_2.exf


Excel 2.x Chart

The previous calculations were reported to obtain a preliminary idea about the order of magnitude of the PEC_{added} when the substance would be discharged to aquatic ecosystems, without considering the fate of the compound, effluent treatments and other emission sources.

Carbonate

Based on the HERA detergent scenario the $PEC_{regional.added}$ and the $PEC_{local.added}$ of the carbonate anion are 1.0 and 3.2 mg/l, respectively. However, in reality the total domestic discharge of carbonate to aquatic ecosystems will be completely different because :

- The final discharge of carbonate/bicarbonate will depend very significantly on the domestic waste water treatment method. Normally the pH of the untreated waste water is measured and adapted when necessary (to neutral pH) to optimise the conditions for the domestic waste water treatment plant (WWTP). This means that carbonate is already neutralized to bicarbonate before the domestic WWTP.
- The discharge of organic and inorganic carbon via faeces and urine is much higher than the discharge via household cleaning products. Based on a total amount of 550 million kg of sodium carbonate used per year and based on 370 million inhabitants in the European Union, the daily use of inorganic carbon is 0.46 g per inhabitant per day. According to Directive 91/271/EEC the biodegradable organic load is 60 g oxygen per inhabitant per day in the EU. If this amount of oxygen is used for the formation of carbon dioxide then the discharge of organic carbon would be equal to 22.5 g per inhabitant per day. This shows that the amount of carbon, emitted via faeces/urine is much higher than the amount emitted via the use of sodium carbonate in household cleaning products. Due to the biodegradation of organic carbon to inorganic carbon in the waste water treatment plant, it is unlikely that the carbonate of the household cleaning products has an effect on the final concentration of inorganic carbon in the effluent.

These 2 factors show that the use carbonate in household cleaning products has a negligible effect on the carbon chemistry of the aquatic ecosystems. The domestic effluent treatment method and the discharges of organic carbon are more important for the carbon chemistry of the receiving water. Even the effect of these 2 factors is questionable. Eutrofication, acidification, deforestation and agricultural practices are known to have an important effect on the carbon chemistry of the aquatic ecosystems (Kempe, 1984).

Sodium
It is evident that effluent treatments do not affect the discharge of sodium. Therefore it can be assumed that the total quantity of sodium is emitted to the aquatic ecosystems. Based on the HERA detergents scenario this would result in a $PEC_{regional.added}$ and a $PEC_{local.added}$ for sodium of 0.7 and 2.5 mg/l, respectively.

Although the use of sodium carbonate in household cleaning products results in an emission of sodium to aquatic ecosytems it is clear that other anthropogenic activitities, e.g. mining and use of road salt, result also in an emission of sodium to aquatic ecosystems. According to UNEP (1995) the sodium and chloride concentrations in water are tightly linked for the major rivers of the world. Furthermore it should be realised that sodium is taken up via food, excreted and emitted to aquatic ecosystems. A normal uptake of sodium via food is 3.1-6.0 g per inhabitant per day according to Fodor et al. (1999) and a similar amount will be emitted to aquatic ecosystems. The daily discharge of sodium, based on the presence of sodium carbonate in household cleaning products, is equal to 1.8 g per inhabitant per day. This value is based on 370 million habitants and a total sodium carbonate use of 550,000 tonnes per year in household cleaning products.

2.2 Environmental effects assessment

2.2.1 Toxicity

Effects on fish
A toxicity test with 50 bluegill sunfish (*Lepomis macrochirus*) exposed to sodium carbonate and 10 control fish was performed by Cairns and Scheier (1959). After 24, 48, 72 and 96 hours the mortality was determined. The purpose of the work was to determine the tolerance of three distinct size ranges of the bluegill (small 3.88 cm and 0.96 g, medium 6.09 cm and 2.80 g, large 14.24 cm and 54.26 g). The TL_m or 96h.LC_{50}, which is the concentration at which 50 % of organism would be expected to survive, was equal to 300 mg/l for all three sizes.

Another 96 hr median tolerance limit test with sodium carbonate was performed with the mosquitofish (*Gambusia affinis*) by Wallen et al. (1957). The experiments were continued for at least 96 hours with observations after 24, 48, 72 and 96 hours. At 24 hours the TL_m (equal to LC_{50}) was 1200 mg/l, at 48 hours 840 mg/l and at 96 hours 740 mg/l.

The minimum lethal concentration of sodium carbonate to different species of minnows (Lake Emerald (*Notropis a. atherinoides*) and spotfin shiners (*Notropis spilopterus*) was determined by Van Horn et al. (1949). In this study the minimum lethal concentration was defined as the lowest concentration of a toxic material which would kill any of the test animals within a period of 120 hours, at a temperature of 18ºC. The minimum lethal concentration for sodium carbonate was determined to be 250 mg/l.

A short review of the acute toxicity for fish is described by the California State Water Resources Control Board (McKee and Wolf, 1976). Concentrations of 68-80 mg/l were reported to kill king salmon, silver salmon and cut-throat trout after 5 days of exposure, while other species, like carp, bass, shiners, sunfish and mosquito-fish, were killed at concentrations ranging between 200 and 1200 mg/l with an exposure duration ranging from hours up to five days. Furthermore, exposure for 5 days to concentrations of 33-58 mg/l to king salmon, silver

salmon and cut-throat trout has not been harmful. Exposure for 7 days to 100-200 mg/l to bass and sunfish and to 200-500 mg/l to goldfish was not harmful.

Effects on invertebrates
Recently toxicity tests with laundry detergent components and the freshwater cladoceran *Ceriodaphnia* cf. *dubia* were published by Warne et al. (1999). Two different tests were done with sodium carbonate and the reported EC_{50} values for 48 hr exposure were 200 and 227 mg/l, respectively. The study was well documented. For example the culture of organisms, the preparation of the test solutions, the test conditions and the data analysis were described adequately.

A short review of acute toxicity of sodium carbonate for *Daphnia magna* is described by the California State Water Resources Control Board (McKee and Wolf, 1976). The threshold concentration of sodium carbonate for immobilisation of *Daphnia magna* in Lake Erie water at 25°C was reported to be 424 mg/l, less than 424 mg/l and 300 mg/l. The minimum lethal concentration for *Daphnia* was also shown to be 300 mg/l at 17°C and at 800 mg/l all animals were killed.

An overview of the available acute toxicity data is presented in Table 3. To evaluate the reliability of the ecotoxicity studies a Code of Reliability (CoR) was assigned to each ecotoxicity test according to Klimisch et al. (1997). In general, mortality of the test organisms was found at concentrations higher than 100 mg/l but for salmon and trout lethal effects were already observed at 68-80 mg/l. Salmon and trout are fish species which are normally found in soft water and this could explain their sensitivity for sodium carbonate.

In general the available toxicity studies with sodium carbonate were not conducted according to current standard guidelines. In many cases pH, buffer capacity and/or medium composition were not discussed in the publications, although this is essential information for toxicity tests with sodium carbonate. For this reason many studies were assigned a Code of Reliability of 4 (see Table 3).

Mode of action
When sodium carbonate is added to water it results not only in an increase of the sodium and carbonate concentration but also to an increase of the bicarbonate and OH^- concentration (pH) of the water ($CO_3^- + H_2O \rightarrow HCO_3^- + OH^-$).
In theory all ions could contribute to the observed acute toxicity of sodium carbonate, which is found at concentrations of 68-1200 However, the addition of sodium can not explain the toxicity because the acute EC50 values of sodium chloride are an order of magnitude higher (> 1 g/l; Environment Canada, 2000). The concentrations of bicarbonate change only slightly at sodium carbonate additions of 100 – 1000 mg/l (see Table 2) and the acute toxicity of sodium bicarbonate is also too low to explain the effects. The acute EC50 of sodium bicarbonate for daphnids and bluegill sunfish was 4100 and 7100 mg/l, respectively (Church & Dwight, 2000). However, the increase of the pH can explain the observed acute toxicity of sodium carbonate. Additions of 100 – 1000 mg/l increase the pH to values of about 10 to 11 and these pH values have been shown to be toxic for aquatic organisms (Solvay, 2002).

Table 3: Summary of acute toxicity studies with sodium carbonate

Species	Endpoint	Result (mg/l)	Code of Reliability	Reference
Bluegill sunfish	EC_{50}.96h	300	3	Cairns et al. (1959)
Mosquitofish	EC_{50}.96h	740	4	Wallen et al. (1957)
Minnows and spotfin shiners	Minimum lethal concentration	250	4	Van Horn et al. (1949)
Salmon and trout	Lethal concentration	68-80	4	McKee et al. (1976)
Carp, bass, shiners, sunfish etc.	Lethal concentration	200-1200	4	McKee et al. (1976)
Cladoceran (*C.* cf. *dubia*)	EC_{50}.48h	200-227	2	Warne et al. (1999)
Cladoceran (*Daphnia*)	Minimum lethal concentration	300	4	McKee et al. (1976)

2.2.2 Derivation of PNEC

Normally the PNEC of chemicals is derived based on the available ecotoxicity data. In most cases the lowest NOEC of the available ecotoxicity tests is used, in combination with an assessment factor, to determine the PNEC. This procedure is not valid for sodium carbonate because the carbonate re-equilibrates in water and because all ions, which are released/formed (sodium, carbonate, bicarbonate and OH^-), are naturally present.

In theory a maximum value of the pH/alkalinity could be used to calculate a PNEC or a $PNEC_{added}$ of sodium carbonate. However, individual aquatic ecosytems are characterized by specific alkalinity/pH and the organisms of the ecosystem are adapted to these specific natural conditions. Based on the natural alkalinity/pH of waters, organisms will have different optimum conditions, ranging from poorly buffered waters with a pH of 6 or less to very hard waters with pH values up to 9. A lot of information is available about the relationship between alkalinity/pH and ecosystem structure and also natural variations in alkalinity/pH of aquatic ecosystems have been quantified and reported extensively in ecological publications and handbooks. For this reason it is not considered useful to derive a PNEC or a $PNEC_{added}$ for sodium carbonate because:

- The natural alkalinity/pH of aquatic ecosystems can vary significantly between aquatic ecosystems and
- Also the sensitivity of the aquatic ecosystems to a change of the alkalinity/pH can vary significantly between aquatic ecosystems.

Although a PNEC or a $PNEC_{added}$ was not calculated for sodium carbonate there is a need to assess the environmental effect of a sodium carbonate (alkaline) discharge. The most appropriate parameter to assess the environmental effect of such a discharge is to determine the change in pH. Based on the pH and buffer capacity of effluent and receiving water and the dilution factor of the effluent, the pH of the receiving water after the discharge can be calculated. Of course the pH change can be measured also very easily via a laboratory experiment or by conducting field measurements. The change in pH should be compared with the natural variation in pH of the receiving water and based on this comparison it should be assessed if the pH change is acceptable.

To illustrate the procedure and to get an idea about the order of magnitude for acceptable anthropogenic additions, the acceptable sodium carbonate addition will be calculated for 2 representative cases. According to Directive 78/659/EEC (CEE, 1978), the pH of surface water for the protection of fish should be between 6 and 9. In section 2.1 it has been mentioned that the 10^{th} percentile and the 90^{th} percentile of the bicarbonate concentrations of 21 European rivers were 15 and 233 mg/l, respectively. If it is assumed that only bicarbonate is responsible for the buffer capacity of the ecosystem and if it is assumed that an increase of the pH to a value of 9.0 would be the maximum accepted value then the acceptable anthropogenic addition of sodium carbonate would be 2.3 and 20 mg/l for bicarbonate concentrations of 15 and 233 mg/l, respectively. This gives an indication of the acceptable amount of sodium carbonate which could be discharged to an aquatic ecosystem if there was an emission of a pure sodium carbonate solution.

Sodium carbonate concentrations of 2.3 and 20 mg/l are equivalent with the sodium concentrations of 1.0 and 8.7 mg/l. Sodium concentrations of 1.0 to 8.7 have no effect on aquatic organisms because sodium has a low toxicity for aquatic organisms. Reconstituted water of toxicity tests contains for example sodium concentrations which range between 3.3 and 105 mg/l (ASTM, 1996).

2.3 Environmental Risk Characterisation

The previous paragraph indicates that the acceptable amount of sodium carbonate which can be discharged to an ecosystem would be 2.3 to 20 mg/l (order of magnitude). This was based on the change of the pH.

The amount of sodium carbonate which is used in the EU in household cleaning products was estimated to be 550,000 tonnes per year. Based on the EUSES HERA detergent scenario the $PEC_{regional.added}$ and $PEC_{local.added}$ of sodium carbonate were 1.7 and 5.7 mg/l, respectively.

However, the use of sodium carbonate in household cleaning products will not result in an emission of carbonate to aquatic ecosystems because the domestic effluents are treated and because the organic carbon from faeces and urine have a more important effect on the carbon chemistry of the aquatic ecosystems. Eutrofication, acidification, deforestation and agricultural practices are also known to have an important effect on the carbon chemistry of the aquatic ecosystems (Kempe, 1984). For this reason it can be concluded that the use of sodium carbonate in household cleaning products has a negligible effect on the carbon chemistry of aquatic ecosystems.

The $PEC_{regional.added}$ and $PEC_{local.added}$ of sodium carbonate were 1.7 and 5.7 mg/l, respectively, which means that the $PEC_{regional.added}$ and $PEC_{local.added}$ of sodium are 0.7 and 2.5 mg/l, respectively. These concentrations are expected to have no effect on aquatic organisms because reconstituted water for acute and chronic toxicity tests contain sodium concentrations which range between 3.3 and 105 mg/l (ASTM, 1996). These PEC_{added} values of sodium are also relatively low compared to measured concentrations of sodium in aquatic ecosystems. The sodium concentration was reported for a total number of 21 rivers in Europe, with a 10^{th} percentile of 1.9 mg/l, mean of 56 mg/l and 90^{th} percentile of 92 mg/l (UNEP, 1995). Other anthropogenic activities have most likely a more important effect on the sodium content of aquatic ecosystems. For this reason it can be concluded that the sodium, which originates from the use of sodium carbonate in household cleaning products, has a negligible effect on the aquatic ecoystems.

2.4 Discussion and Conclusions

Sodium carbonate dissociates into sodium and carbonate and both ions do not adsorb to sediment. Furthermore there is no distribution or transport to the atmosphere and therefore the environmental risk assessment can be focussed on the aquatic compartment.

An initial environmental exposure assessment was done with EUSES using the HERA detergent scenario. Due to effluent treatments the carbonate, present in the household cleaning products, will not be discharged to the aquatic ecosystems but will be neutralized. Furthermore there is evidence that the carbon chemistry of aquatic ecosystems is influenced by other anthropogenic activities. Sodium has a low toxicity and the emitted amount of sodium is relatively low compared to background concentrations and therefore the emitted amount of sodium will not have an effect on the aquatic organisms of the receiving water.

Based on the available data, the use of sodium carbonate in household cleaning products has no adverse effect on the aquatic ecosystem.

3. Human Health Assessment

3.1 Consumer Exposure

The product sodium carbonate is available for consumers and solutions of sodium carbonate in water have been used in the past for soaking of clothes, dishwashing, floor washing, degreasing operations and for personal care (e.g. skin treatment). These applications may still occur.

Much larger quantities of sodium carbonate are used for the production of detergents, laundry additives, machine dishwashing products, surface cleaners, toilet cleaners and other household cleaning products. For most products the typical sodium carbonate content is less than 30 %, but maximum concentrations can be higher (up to 90 %).

As relevant consumer contact scenarios, skin contact, inhalation and oral uptake of sodium carbonate were identified and assessed.

3.1.1 Direct skin contact with sodium carbonate via solutions

Consumers may be exposed to sodium carbonate via skin contact with solutions which contain sodium carbonate. A common exposure scenario seems to be laundry hand washing with a detergent and therefore this scenario will be discussed below. An exposure scenario with a relatively high exposure seems to be the use of a sodium carbonate solution for personal care and for this reason this scenario will be evaluated also. Other applications, resulting in exposure to solutions of sodium carbonate, do exist but they are probably less common or the exposure will be lower.

Laundry hand washing

There is a consolidated overview concerning habits and uses of detergents and surface cleaners in Western Europe issued by A.I.S.E. (2002). This list reflects the consumer`s use of detergents in g/cup, tasks/week, duration of task and other uses of products. This overview of A.I.S.E. (2002) has been used to calculate the exposure.

The concentration of laundry detergent in the hand washing solution is approximately 1 % (10 g/l). The highest concentration of sodium carbonates in laundry detergents amounts to 30 %. For this reason in a worst case assumption, the hands and forearms of the consumer are exposed to an estimated sodium carbonate concentration of 3.0 g/l (= mg/ml). The estimated surface of the hands and the forearms, exposed to the washing solution, is 1980 cm² (EPA, 1997).

Assuming a film thickness of 100 µm (Lally, Ch., 2001) on the hands and a percutaneous absorption of 0.1 % for a ionic substances (absorption of ionic salts via the skin is essentially negligible; Schaefer et al., 1996) in 24 hr exposure time, the following amount of sodium carbonate absorbed via skin can be calculated:
1980 cm² (surface of hands and forearms) x 0.01 cm (film thickness) x 0.001 (fraction absorbed) x 3.0 mg/cm^3 (sodium carbonate concentration) = 0.059 mg. As this is calculated for a 24 h exposure and the exposure time is normally 10 minutes (A.I.S.E., 2002) this has to

be corrected by a factor of (24x60/10) yielding an assumed absorption of 4.1 x 10^{-4} mg per event.

Based on a body weight of 60 kg the estimated systemic dose of sodium carbonate would be equal to 4.1 x 10^{-4} / 60 = 6.8 x 10^{-6} mg/kg body weight per event which can be regarded as negligible.

Use of sodium carbonate solution for personal care

Solutions of sodium carbonate have been and can still be used for personal care (e.g. skin treatment). It is difficult to define a standardized exposure scenario or a typical exposure scenario for such an application of sodium carbonate. A concentration of 1 % sodium carbonate in water could be a realistic concentration. Exposure of feet would result in an exposure surface are of 1,120 cm^2 (TGD, Part I, Annex VI). Using a film thickness of 100 µm (Lally, Ch., 2001) and a percutaneous absorption of 0.1 %, the amount of sodium carbonate absorbed via skin would be 0.1 mg. Assuming an exposure time of 20 min this would amount to 0.0014 mg per event. The estimated systemic dose of sodium carbonate would be equal to 0.0014/60 = 2.3 x 10^{-5} mg/kg body weight.

3.1.2 Direct skin contact with solid sodium carbonate

The absorption of inorganic solids via skin contact is very low. Furthermore the contact time is low and area of contact with skin is so small that the amount absorbed percutaneously by consumers is considered negligible.

3.1.3 Inhalation

The dust formation from the product sodium carbonate, and also from products containing sodium carbonate, is so small that the amount is considered negligible for consumers. The negligible inhalation has been confirmed for the laundry washing scenario. According to Van de Plassche et al. (1998) studies indicate an average exposure of about 0.27 µg dust per cup of product used for machine laundering, of which up to 30 % or 0.08 µg is sodium carbonate.

3.1.4 Oral uptake

Oral uptake of sodium carbonate by consumers via the use of household cleaning products is considered negligible under normal handling and use conditions.

3.1.5 Accidental or intentional overexposure

Accidental or intentional overexposure to sodium carbonates may potentially occur via:

- oral exposure to the product sodium carbonate or to products which contain sodium carbonate,
- oral exposure to solutions of these products in water,
- exposure of the eyes to the product sodium carbonate or to products which contain sodium carbonate,
- exposure of the eyes to solutions of these products in water (e.g. due to splashing) and
- inhalation exposure to the product sodium carbonate or to products which contain sodium carbonate.

No fatal cases arising from oral uptake of sodium carbonate (solutions) have been reported. Furthermore case reports related with high exposure to sodium carbonate (solutions) have not been reported in the medical literature. The German Federal Institute for Health Protection of Consumers and Veterinary Medicine (BgVV, 1999) published recently a report on products involved in poisoning cases. No fatal case of poisoning with detergents was reported in this report. Detergent products were not mentioned as dangerous products with a high incidence of poisoning.

Accidental spillage may cause eye contact of sodium carbonate. Cases of eye irritation, which were caused by sodium carbonate (solutions), have not been reported in the literature.

Cases of accidental inhalation exposure to the product sodium carbonate have not been found in the literature. However, sodium carbonate has a large particle size (average particle size diameter > 90 µm, see section 1.1). Inhalation of laundry detergent powder by children has been reported in the Unites States (Einhorn et al., 1989). The predominant symptoms were stridor, drooling and respiratory distress. It is unknown if similar cases of accidental inhalation exposure have occurred in Europe.

3.2 Hazard Assessment

3.2.1 Toxicokinetics, metabolism and mechanism of action

When sodium carbonate comes into contact with body fluids it will dissociate into carbonate and sodium. The carbonate could potentially increase the pH of the blood. The major extracellular buffer in the blood and the interstitial fluid of vertebrates is the bicarbonate buffer system, described by the following equation:

$$H_2O + CO_2 \leftrightarrow H_2CO_3 \leftrightarrow H^+ + HCO_3^-$$

Carbon dioxide from the tissues diffuses rapidly into red blood cells, where it is hydrated with water to form carbonic acid. This reaction is accelerated by carbonic anhydrase, an enzyme present in high concentrations in red blood cells. The carbonic acid formed dissociates into bicarbonate and hydrogen ions. Most of the bicarbonate ions diffuse into the plasma. Since the ratio of H_2CO_3 to dissolved CO_2 is constant at equilibrium, pH may be expressed in terms of bicarbonate ion concentration and partial pressure of CO_2 by means of the Henderson-Hasselbalch equation:

$$pH = pKa + \log [HCO_3^-]/\alpha PCO_2$$

The blood plasma of man normally has a pH of 7.4. Should the pH fall below 7.0 or rise above 7.8, irreversible damage may occur. Compensatory mechanisms for acid-base disturbances function to alter the ratio of HCO_3^- to PCO_2 , returning the pH of the blood to normal. Thus, metabolic acidosis may be compensated for by hyperventilation and increased renal absorption of HCO_3^-. Metabolic alkalosis may be compensated for by hypoventilation and the increased excretion of HCO_3^- in the urine (Johnson and Swanson, 1987). Therefore, if carbonate is absorbed its concentration will be regulated by these physiological mechanisms

and therefore elevated amounts of carbonate are not expected to be available in the body. In other words, carbonate is not expected to be systemically available in the body.

Furthermore it should be realised that an oral uptake of sodium carbonate results in a neutralisation of carbonate in the stomach by the gastric acids. Significant amounts of gastric acids are present in the stomach (pH about 2) which will result in a formation of bicarbonate and/or carbon dioxide. Therefore it is very unlikely that an oral uptake of sodium carbonate will result in a pH increase of the blood. Acute effects after oral exposure to high amounts of sodium carbonate can be due to CO_2 gas formation in the stomach.

Sodium is an essential element in the diet but a high intake of sodium has been accociated with cardio-vascular diseases. Sodium is readily absorbed throughout the small intestine and is subject to rapid exchange by the large majority of cells in the body. The main regulation of the body concentrations of sodium takes place in the kidney. The consumer exposure to household cleaning products results in a relatively low exposure to sodium (compared to dietary uptake) and therefore elevated amounts of sodium are not expected to be available in the body. In other words, sodium is not expected to be systemically available in the body.

3.2.2 Acute Toxicity

An acute oral toxicity study with rats revealed an LD_{50} of 4090 mg/kg (Lewis, 1996; Richardson, 1994). An acute dermal toxicity study with mouse resulted in an LD_{50} of 2210 mg/kg (Lewis, 1996; Richardson, 1994). No further information was reported. Both these studies were reported in secondary literature but the original reference could not be found (Code of Reliability = 4). While the value for the acute oral toxicity seems to be plausible given the considerations above, the anticipated low availability by the dermal route causes some doubts about the reliability of the dermal LD50 reported.

In an attempt to establish a LC_{50} for sodium carbonate, a series of whole-body inhalation exposures of rats (Sprague-Dawley and Wistar strains), mice (Swiss-Webster) and guinea pigs (Hartley-albino) to varying concentrations were performed (Busch et al., 1983). The animals exhibited respiratory impairment when exposed for 2 hours to aerosols of sodium combustion products (1 µm aerodynamic equivalent diameter), the major constituent of which was shown to be sodium carbonate (rats 91 % Na_2CO_3, dose range 800-4600 mg/m^3, mice 95 % Na_2CO_3, dose range 600-3000 mg/m^3, guinea pigs 95 % Na_2CO_3, dose range 500-3000 mg/m^3). The LC_{50}s for guinea pigs, mice and rats were calculated to be 800, 1200 and 2300 mg/m^3, respectively. Lesions in animals which died were limited to the posterior pharynx, larynx, anterior trachea, and in approximately 3 % of the animals, lungs. It should be taken into consideration that the particles from fumes of combustion products are very small and not comparable to particles that could be obtained by preparing an aerosol of crystalline sodium carbonate. The majoritiy of the particles would then be rather in the range around 10 µm which would result in a lower exposure of the lower respiratory tract. Therefore the data obtained from fumes are of limited relevance for an evaluation of the acute inhalation toxicity of sodium carbonate dust.

The *in vitro* and *in vivo* toxicity of sodium carbonate for the rat nasal cavity has been studied by Kilgour et al. (2000). Rats were exposed to a solution of sodium carbonate in high purity water for 4h at target chamber concentrations of 250 and 750 µg/l. The aerosols contained particles of less than 2 µm mass mean aerodynamic diameter. No morphological changes

were observed within any region of the nasal cavity following exposure of the rats to sodium carbonate.

A transformed epithelial cell line derived from normal human bronchial epithelium was used to assess the *in vitro* toxicity of sodium carbonate (Westmoreland et al., 1999). The IC_{50} of neutral red uptake and MTT reduction was 3840 and 4770 µg/ml, respectively.

Reliable acute oral or dermal toxicity studies are not available. However, the substance has been used for a long time and very extensively but medical cases have not been reported in the literature and for this reason it is unlikely that the substance is toxic. Furthermore, sodium carbonate is not expected to be systemically available in the body due to neutralisation by the gastric acids and blood, and thus there seems to be no need for further testing on acute oral or dermal toxicity with animals.

3.2.3 Skin Irritation

Animal data

A skin irritation study was performed with six New Zealand White rabbits (Chibanguza, 1985a). An amount of 0.5 g sodium carbonate was applied to intact and abraded skin (6.25 cm^2) and covered with an occlusive bandage for 4 hours. After this period the skin was washed. Sixty min, 24, 48 and 72 hours after exposure no signs of erythema or oedema were observed. The method used in this study was comparable to OECD guideline 404.

An aqueous solution of sodium carbonate (50 % w/v) was applied to the skin (intact and abraded) of six rabbits and six guinea pigs for 4 hours (Nixon et al., 1975). The animals were examined at 4, 24 and 48 hours after application of the solution for erythema and oedema. Categorisation of irritancy to rabbit and guinea pig skin was based on the primary irritation index (PII), which includes the response of abraded skin. The abraded skin of the rabbits had slight erythema and oedema, and those of the guinea pigs were negligibly affected. There were no signs of erythema or oedema in the intact skins (mean PII 0.8 for rabbits and 0.1 for guinea pigs). The test procedure was the revised FHSA procedure that had been proposed by the FDA in 1972, except for minor deviations. The study was performed before OECD 404 came into force.

Human data

A human patch (skin irritation) test with 98 % sodium carbonate was performed using 26 human volunteers and exposing them for 15, 30 or 60 minutes through to 1, 2, 3 and 4 hours (York et al., 1996). The patch test involved the application of 0.2 g on to a plain Hill Top Chamber and treated sites were assessed 24, 48 and 72 hours after patch removal. The results showed no reactivity among the volunteers and therefore sodium carbonate was not classified as irritant based on the human patch test.

An aqueous solution of sodium carbonate (50 % w/v) was applied to the skin (intact and abraded) of six human volunteers for 4 hours (Nixon et al., 1975). The volunteers were examined at 4, 24 and 48 hours after application of the solution for erythema and oedema. Categorisation of irritancy to human skin was based on the PII. The abraded skin had erythema and oedema (mean score 2) with one or more of the subjects having the maximum grade of 4. There were no signs of erythema or oedema in the intact skins (mean PII >1.0).

3.2.4 Eye Irritation

An eye irritation study was performed with six New Zealand White rabbits (Chibanguza, 1985b). Ocular irritancy was tested by instilling 0.1 g sodium carbonate into the left eye (conjunctival sac) of each animal, the right eye served as the untreated control. After 1 hour, 24, 48 and 72 hours the eyes were examined for observations of the conjunctivae, cornea and iris. Ocular irritation was scored according to the scale by Draize. The mean Draize intensity score was for conjunctival redness 1.67, for conjunctival chemosis 1.38 and for the cornea 0.25. It was concluded by the authors that the test substance could not be classified as an ocular irritant. The method used in this study was comparable to OECD guideline 405.

Ocular irritation of sodium carbonate was evaluated in two groups of at least six New Zealand albino rabbits (male and female) based on the methodology of Draize (Murphy's et al., 1982). This study was performed to show ocular irritancy responses to various pHs of acids and bases with and without irrigation. Sodium carbonate (0.1 ml) was administered to the right eye directly on the central portion of the cornea, the left eye served as the untreated control. The eyes of the first group of rabbits were rinsed for 2 minutes, 30 seconds after instillation (rinsed eyes), the eyes tested in the second group were not rinsed after instillation (unrinsed eyes). Control and treated eyes were scored at 1 h and 1, 2, 3 and 7 days after exposure according to the scale of Draize. Corneal opacities were produced in unrinsed eyes within 1 h after exposure to sodium carbonate and the severest effect was noted by day 3 (mean Draize intensity score 3.8), the severity was maintained through day 7. In rinsed eyes, corneal opacities were observed on day 2 (mean Draize intensity score 0.8) and had disappeared by day 7. Iritis (inflammation of the iris) was observed in unrinsed eyes at 1 h after exposure to sodium carbonate and a mean draize score of 2 was reported on days 1, 2, 3 and 7. In rinsed eyes, iritis was observed at 1 hr after exposure (mean Draize intensity score 1.0) and had disappeared by day 3 after exposure. Sodium carbonate produced conjunctivitis which lasted through day 7 in all animals tested. It also produced pannus (a vascular superficial opacity of the cornea) in 6/12 unrinsed eyes and keratoconus (abnormal cone-shaped protrusion of the cornea of the eye) in 2/12 unrinsed eyes. The method used in this study was mainly comparable to OECD guideline 405, but the duration of the study was not long enough to evaluate reversibility of the effects. Furthermore as the test substance was not applied into the conjunctival sac, as required by the OECD guideline, the normal clearance by tear-fluid could not take place

Results of eye irritation tests with non-phosphate detergents, which contained 65-80 % sodium carbonate, and albino rabbits were reported by Scharpf et al. (1972). Exactly 0.1 ml of solid was instilled into the conjunctival sac of the right eye of six rabbits. The detergents caused considerable ocular irritation followed by opacity and corrosion of the cornea. Little healing of the eye was apparent after a single contact with these detergents. Detergents with such a high concentration of sodium carbonate (65-80 %) are not used in Europe anymore.

3.2.5 Sensitisation

Since 1863, when sodium carbonate production started, millions of people have been exposed to sodium carbonate, products which contain sodium carbonate or to solutions of sodium carbonate. Skin sensitisation has never been reported. Apparently sodium carbonate is not a skin sensitizer. This was confirmed by workers data of sodium carbonate production sites.

3.2.6 Repeated Dose Toxicity

Oral and dermal toxicity
No animal data are available on repeated dose toxicity studies by oral or dermal routes for sodium carbonate.

Inhalation toxicity
Male rats were exposed to a 2 % aqueous sodium carbonate aerosol for 4 h/day, 5 days/week for 3.5 months (Reshetyuk and Shevchenko, 1966). The final concentration was reported to be 70 ± 2.9 mg/m^3, whereas particle size was reported not to exceed 5 µm (no further details given). When compared to controls there were no changes in body weight gain, organ weights, body temperature, or several blood parameters. Pulmonary ascorbic acid levels were decreased. Histopathological examination revealed hyperplasia and desquamation of bronchiolar epithelium, and perivascular oedema. The upper respiratory tract was not examined. Other pulmonary changes included thickening of alveolar walls, hyperaemia and lymphoid infiltration but these changes were also observed in about 50 % of the controls. A preliminary study of unknown duration at a concentration of 10-20 mg/m^3, did not induce toxic effects (Reshetyuk and Shevchenko, 1966).

Although this was a limited reported study, the histopathological changes observed in the lungs are not unexpected, in view of the alkaline nature of the solution (0.1 M (ca. 1 %), pH = 11.6). However, as similar effects were also observed in the control animals it cannot be judged if the observed changes were treatment related or rather due to an infection of the animals which was not uncommon at the time the study was performed.

Although a reliable repeated dose study, done according to current guidelines and reported in sufficient detail, is not available an additional repeated dose toxicity study in rats with sodium carbonate does not seem to be necessary. The effect of repeated exposure of humans to sodium has been studied extensively and has mainly focussed on the effects of sodium on the prevention and control of hypertension. Recommendations on daily dietary sodium intake were reported to be 2.0-3.0 g for a moderately restricted intake and 3.1-6.0 g was considered to be a normal intake (Fodor et al., 1999). An oral uptake of carbonate will result in a neutralisation in the stomach by the low pH of the gastric juice and therefore systemic effects are not expected after oral exposure. Also via other exposure routes (inhalation, dermal exposure) carbonate is not expected to be systemically available in the body due to the limited uptake compared to the neutralisation capacity of the blood. Therefore, additional testing for repeated dose toxicity is considered unnecessary for sodium carbonate.

3.2.7 Genetic Toxicity

Olivier and Marzin (1987) examined sodium carbonate for its potential to induce primary DNA damage in the *Escherichia coli* Chromotest. Different concentrations of sodium carbonate were incubated with samples of an exponentially growing culture of *Escherichia coli* PQ37 for 2 h without metabolic activation. The concentrations tested ranged from 1 to 100,000 nM/ml (appr. 0.11 – 11000 µg/ml). Each dose was studied at least in triplicate. The test was not conducted with addition of a metabolic activation system. There were no indications of a positive response. Toxicity was observed at 10,000 nM/ml (1100 µg/ml). It was concluded that sodium carbonate did not induce primary DNA damage in *Escherichia coli* Chromotest without metabolic activation.

The reporting of the results in the publication of Olivier and Marzin (1987) was rather limited. However, the *Escherichia coli* Chromotest is a well known test of high quality and generally used as a pre-screen, although a negative test result can not be used to rule out possible mutagenicity. An Ames test with sodium bicarbonate and sodium sesquicarbonate with and without metabolic activation has been performed and a negative result was noted (Johnson and Swanson, 1987). When the pH will be kept below 8 to have a good functioning biological test system, mainly bicarbonate will be available. Furthermore, the structural properties of sodium carbonate do not show a reason to evaluate the potential genotoxicity of sodium carbonate further. Data on the structural analog potassium carbonate, did not show any genotoxic activity in the Ames assay with *S. typhimurium* TA92, TA94, TA98, TA100, TA1535, TA1537 at concentrations up to 10 mg/plate with and without metabolic activation and in a cytogenetic assay in Chinese Hamster fibroblasts up to 1 mg/ml without metabolic activation (Ishidate et al., 1984). Therefore there is no concern with regard to a possible genotoxicity of sodium carbonate.

3.2.8 Carcinogenicity

Carcinogenicity studies with animals and sodium carbonate are not available. The substance sodium carbonate is not systemically available and therefore a systemic carcinogenic effect can be excluded. The substance has no structural alerts for a possible genotoxicity or reactivity with DNA or proteins. A possible local carcinogenic effect is therefore also highly unlikely.

3.2.9 Toxicity to Reproduction

An animal reproduction study with sodium carbonate is not available. The substance will usually not reach the male and female reproductive organs when exposed orally, dermally or by inhalation, as it does not become available systemically (see 3.2.1). For this reason the substance is not considered toxic to reproduction and it is considered not useful to perform a reproduction study with animals.

3.2.10 Developmental Toxicity / Teratogenicity

Aqueous solutions of sodium carbonate were administered via oral intubation to pregnant mice at doses ranging from 3.4 to 340 mg/kg during days 6-15 of gestation. The test substance did not affect implantation nor the survival of dams and foetuses. The number of soft tissue or skeletal abnormalities of the test group did not differ from that of controls. Similar negative results were reported for rats and rabbits at doses up to 245 mg/kg and 179 mg/kg, respectively (Johnson and Swanson, 1987; FDA, 1974). Although it is not a developmental toxicity study, done according to current guidelines and GLP, it is reported in sufficient detail, and it confirms in three species that there is no concern with regard to developmental toxicity up to the tested dose levels. This supports the general consideration that the substance will usually not reach the foetus when exposed orally, dermally or by inhalation, as it does not become systemically available (see section 3.2.1).

3.2.11 Identification of critical endpoints

Sodium carbonate is an eye irritating substance and may have an irritant effect on the abraded skin. These local effects are probably related to the alkaline properties of the substance.

The carbon dioxide/bicarbonate/carbonate buffer system is one of the main physiological buffer systems that regulates the cellular and blood pH. The concentration of sodium in the blood and the pH of the blood will not be increased due to sodium carbonate exposure and therefore the substance is not expected to become systemically available. Furthermore, the gastric acids will neutralise the carbonate in the stomach when sodium carbonate is taken up orally. No genotoxic effects in bacteria and in bacteria and mammalian cells with the structural analogon potassium carbonate were reported. Neither a developmental toxicity nor teratogenic effects were observed in studies in rabbits, rats and mice, receiving high oral doses of sodium carbonate. There are no concerns with regard to carcinogenicity or possible reproductive effects and for this reason the substance is not expected to have any adverse long term effects. Implicitly this has been recognised in the past, because sodium carbonate is considered 'GRAS' (Generally Recognised As Safe) in food with no limitation other than current good manufacturing practice (CFR, 1999).

The only critical endpoint for sodium carbonate seems to be local irritation in particular to mucous membranes of the eye, which is confirmed by the human experience with this substance. Sodium carbonate has been used widely and for a long time and until now there is no evidence for a high acute toxicity, sensitisation, carcinogenicity or a chronic toxicity.

3.3 Consumer Risk Characterization

Based on normal habits and uses, the consumer exposure to sodium carbonate by inhalation, oral uptake and skin contact to solid sodium carbonate is negligible and therefore the associated risk is also negligible. However, two relevant exposure scenario's were identified and the potential risks will be characterized for both scenario's.

3.3.1 Direct skin contact with sodium carbonate via solutions

The estimated absorbed dose of sodium carbonate due to laundry hand washing was 4.1×10^{-4} mg per event, while the estimated absorbed dose due to use of a sodium carbonate solution for personal care was 1.4×10^{-3} mg per event. This results in a total absorbed dose of 1.8 µg sodium carbonate, which is equivalent with 0.8 µg sodium and 1.0 µg carbonate, respectively. The amount of 1.0 µg carbonate will not affect the pH of the blood, while the amount of 0.8 µg sodium is negligible compared to the normal daily dietary uptake of sodium of 3.1- 6.0 g (Fodor et al., 1999). For this reason it can be concluded that the exposure to sodium carbonate via solutions has no systemic effect on the consumers.

Based on studies with rabbits and human volunteers a concentration of sodium carbonate of 50 % has no or a low skin irritancy. The estimated concentration of sodium carbonate in solutions is 1 % or less and therefore there is no risk for irritation. Therefore it can be concluded that local effects on the skin are not expected when consumers are exposed to sodium carbonate via solutions (laundry hand washing, personal care).

3.3.2 Accidental or intentional overexposure

Accidental or intentional overexposure to sodium carbonate may occur via the oral route, via exposure of the eyes (e.g. due to splashing) or via inhalation.

An acute oral toxicity study, although unreliable, did not indicate a high toxicity. Furthermore a high oral toxicity of sodium carbonate is not expected due to the neutralisation of carbonate in the stomach to bicarbonate and/or carbon dioxide. For this reason the LD50 is not expected

to be lower than 2000 mg/kg bodyweight. Based on such an LD50 value of 2000 mg/kg the uptake of sodium carbonate by humans must be very high (> 100 g) to reach acute lethal effects. This has been confirmed by the human experience with this substance. Although sodium carbonate is widely available for consumers and has been used for a long time, acute cases of oral poisoning have not been reported in the literature. Apparently accidental or intentional overexposure to sodium carbonate via the oral route does not result in severe adverse effects on humans or does simply not occur.

Sodium carbonate is not a corrosive substance but it has an eye irritation potential probably due to the alkaline properties. Eye irritation tests with rabbits showed for example corneal opacities, iritis and conjunctivitis after exposure to sodium carbonate. These effects were found after exposure to 0.1 ml or 0.1 g, which is a relatively large amount compared to accidental eye exposure of humans. Effects on human eyes, due to exposure to sodium carbonate, were not found in the literature, notwithstanding the extensive use of the substance by consumers. Apparently accidental or intentional overexposure of the eyes does not result in severe adverse effects on humans or does simply not occur.

Although accidental oral, eye or inhalation exposure to the product sodium carbonate has not been reported in the literature, ingestion and inhalation of laundry detergent powder by children has been reported in the Unites States (Einhorn et al., 1989). The predominant symptoms were stridor, drooling and respiratory distress. It is unknown if similar cases of accidental inhalation exposure have occurred in Europe.

3.4 Discussion and conclusions

The concentration of sodium in the blood and the pH of the blood will not be increased due to sodium carbonate exposure and therefore the substance is not expected to become systemically available. No genotoxic effects in bacteria or teratogenic effects in rabbits, rats and mice have been reported. Therefore, sodium carbonate will not reach the organs or the foetus and there is no risk for systemic, developmental or reproductive toxicity or genotoxicity. It is also very unlikely to have sensitising properties. The only critical endpoint for sodium carbonate seems to be local irritation.

Consumers will be exposed to sodium carbonate due to direct skin contact with solutions which contain sodium carbonate, which can be laundry hand washing or use of a carbonate solution for personal care (e.g. skin treatment). However, the estimated concentrations of sodium carbonate in these solutions are too low to cause local irritation.

Accidental or intentional acute overexposure to sodium carbonate may occur via the oral route, via exposure of the eyes (e.g. due to splashing) or via inhalation. The available animal data do not indicate severe adverse effects when accidental or intentional overexposure to sodium carbonate occurs. Although sodium carbonate is widely available for consumers and has been used for a long time, acute cases of oral poisoning or effects on human eyes have not been reported in the literature. Apparently accidental or intentional overexposure to sodium carbonate does not result in severe adverse effects on humans or does simply not occur.

Based on the available data, the use of sodium carbonate in household cleaning products has no adverse effect on consumers.

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