100 gm water + 75 gm xylitol =
168 ml
75/168 = 45%

Xylitol

Cite this Record

STRUCTURE VENDORS DRUG INFO PHARMACOLOGY LITERATURE PATENTS BIOACTIVITIES

PubChem CID:	6912
Chemical Names:	Xylitol; Adonitol; Ribitol; D-Xylitol; Xylite; 488-81-3 More...
Molecular Formula:	$C_5H_{12}O_5$
Molecular Weight:	152.146 g/mol
InChI Key:	HEBKCHPVOIAQTA-NGQZWQHPSA-N
Drug Information:	Therapeutic Uses Clinical Trials FDA UNII

Xylitol is a five-carbon sugar alcohol derived from XYLOSE by reduction of the carbonyl group. It is as sweet as sucrose and used as a noncariogenic sweetener.

from MeSH

Xylitol is a five-carbon sugar alcohol that is obtained through the diet. It is not endogenously produced by humans. Xylitol is used as a diabetic sweetener which is roughly as sweet as sucrose with 33% fewer calories. Xylitol is naturally found in many fruits (strawberries, plums, raspberries) and vegetables (e. g. cauliflower). Because of fruit and vegetable consumption the human body naturally processes 15 grams of xylitol per day. Xylitol can be produced industrially starting from primary matters rich in xylan which is hydrolyzed to obtain xylose. It is extracted from hemicelluloses present in the corn raids, the almond hulls or the barks of birch (or of the by-products of wood: shavings hard, paper pulp). Of all polyols, it is the one that has the sweetest flavor (it borders that of saccharose). It gives a strong refreshing impression, making xylitol an ingredient of choice for the sugarless chewing gum industry. In addition to his use in confectionery, it is used in the pharmaceutical industry for certain mouthwashes and toothpastes and in cosmetics (creams, soaps, etc.). Xylitol is produced starting from xylose, the isomaltose, by enzymatic transposition of the saccharose (sugar). Xylitol is not metabolized by cariogenic (cavity-causing) bacteria and gum chewing stimulates the flow of saliva; as a result, chewing xylitol gum may prevent dental caries. Chewing xylitol gum for 4 to 14 days reduces the amount of dental plaque. The reduction in the amount of plaque following xylitol gum chewing within 2 weeks may be a transient phenomenon. Chewing xylitol gum for 6 months reduced mutans streptococci levels in saliva and plaque in adults (PMID: 17426399, 15964535). Studies have also shown xylitol chewing gum can help prevent acute otitis media (ear aches and infections) as the act of chewing and swallowing assists with the disposal of earwax and clearing the middle ear, while the presence of xylitol prevents the growth

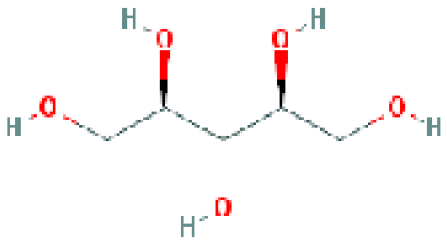
of bacteria in the eustachian tubes. Xylitol is well established as a life-threatening toxin to dogs. The number of reported cases of xylitol toxicosis in dogs has significantly increased since the first reports in 2002. Dogs that have ingested foods containing xylitol (greater than 100 milligrams of xylitol consumed per kilogram of bodyweight) have presented with low blood sugar (hypoglycemia), which can be life-threatening. Xylitol is found to be associated with ribose-5-phosphate isomerase deficiency, which is an inborn error of metabolism.

Metabolite Description from Human Metabolome Database (HMDB)

Modify Date: 2018-01-06; Create Date: 2004-09-16

1 2D Structure

Search Download Get Image

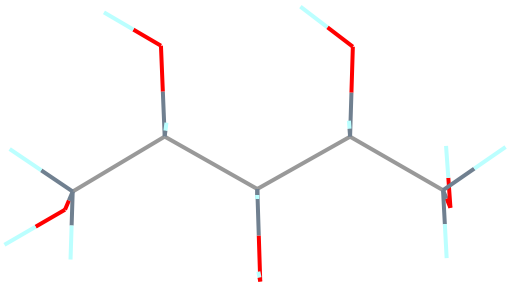


Magnify

from PubChem

2 3D Conformer

Search Download Get Image



Magnify

Show Hydrogens Show Atoms Animate

from PubChem

3 Names and Identifiers

3.1 Computed Descriptors

3.1.1 IUPAC Name

(2S,4R)-pentane-1,2,3,4,5-pentol

from PubChem

3.1.2 InChI

InChI=1S/C5H12O5/c6-1-3(8)5(10)4(9)2-7/h3-10H,1-2H2/t3-,4+,5?

from PubChem

3.1.3 InChI Key

HEBKCHPVOIAQTA-NGQZWQHPSA-N

from PubChem

3.1.4 Canonical SMILES

C(C(C(C(CO)O)O)O)O

from PubChem

3.1.5 Isomeric SMILES

C([C@H](C([C@H](CO)O)O)O)O

from PubChem

3.2 Molecular Formula

$C_5H_{12}O_5$

from EU Food Improvement Agents, PubChem

3.3 Other Identifiers

3.3.1 CAS

488-81-3

from ChemIDplus, DrugBank, European Chemicals Agency - ECHA

87-99-0

from ChemIDplus, DrugBank, EPA DSStox, European Chemicals Agency - ECHA, Human Metabolome Database (...

3.3.2 EC Number

201-788-0

from EU Food Improvement Agents

207-685-7

from European Chemicals Agency - ECHA

201-788-0

from European Chemicals Agency - ECHA

3.3.3 UNII

VCQ006KQ1E

from DrugBank, FDA/SPL Indexing Data

353ZQ9TVDA

from FDA/SPL Indexing Data

3.3.4 Wikipedia

Title	ribitol
Description	sugar alcohol
Title	xylitol
Description	chemical compound

from Wikipedia

3.4 Synonyms

3.4.1 MeSH Entry Terms

Xylitol

from MeSH

3.4.2 Depositor-Supplied Synonyms

1. xylitol
2. adonitol
11. Xylit
12. Xyliton
21. meso-xylitol
22. L-ribitol
31. CHEBI:15963
32. CHEBI:17151

3. ribitol
4. D-Xylitol
5. Xylite
6. 488-81-3
7. 87-99-0
8. Adonite
9. Adonit
10. D-ribitol
13. Eutrit
14. Klinit
15. Xylite (sugar)
16. Kannit
17. Newtol
18. 1,2,3,4,5-pentanepento
19. Xylisorb
20. meso-ribitol
23. L-xylitol
24. D-Adonitol
25. UNII-353ZQ9TVDA
26. UNII-VCQ006KQ1E
27. NSC 25283
28. (2R,3R,4S)-Pentane-1,2,3,4,5-pentaol
29. (2R,3S,4S)-Pentane-1,2,3,4,5-pentaol
30. BRN 1720523
33. EINECS 201-788-0
34. 353ZQ9TVDA
35. (2R,4S)-pentane-1,2,3,4,5-pen
36. HEBKCHPVOIAQTA-ZXFHETKH
37. MFCD00064291
38. MFCD00064292
39. (2S,4R)-pentane-1,2,3,4,5-pen
40. ST50411707

from PubChem

4 Chemical and Physical Properties

4.1 Computed Properties

Property Name	Property Value
Molecular Weight	152.146 g/mol
Hydrogen Bond Donor Count	5
Hydrogen Bond Acceptor Count	5
Rotatable Bond Count	4
Complexity	76.1
CACTVS Substructure Key Fingerprint	AAADccBgOAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAA AAAAAAAAAAAAGgAACAAACBSggAIAAAAAAgAAAAAAA AAAAAAAAAAAAAAAAAAABEAIAAAAAQAAFAAABAADAIA AAAAAAAAAAAAAAAAAAAAAAAAAAAAAAA==
Topological Polar Surface Area	101 A^2
Monoisotopic Mass	152.068 g/mol
Exact Mass	152.068 g/mol
XLogP3	-2.5
Compound Is Canonicalized	true
Formal Charge	0
Heavy Atom Count	10
Defined Atom Stereocenter Count	2
Undefined Atom Stereocenter Count	0
Defined Bond Stereocenter Count	0
Undefined Bond Stereocenter Count	0
Isotope Atom Count	0
Covalently-Bonded Unit Count	1

from PubChem

4.2 Experimental Properties

4.2.1 Physical Description

White, crystalline powder, practically odourless.

from EU Food Improvement Agents

Solid

from Human Metabolome Database (HMDB)

4.2.2 Color

Monoclinic crystals from alcohol

Lide, D.R. CRC Handbook of Chemistry and Physics 88TH Edition 2007-2008. CRC Press, Taylor & Francis, Boca Raton, FL 2007, p. 3-522

from HSDB

4.2.3 Taste

Relative sweetness equal to sucrose

O'Neil, M.J. (ed.). The Merck Index - An Encyclopedia of Chemicals, Drugs, and Biologicals. Whitehouse Station, NJ: Merck and Co., Inc., 2006., p. 1738

from HSDB

4.2.4 Boiling Point

216 deg C

Lide, D.R. CRC Handbook of Chemistry and Physics 88TH Edition 2007-2008. CRC Press, Taylor & Francis, Boca Raton, FL 2007, p. 3-522

from HSDB

4.2.5 Melting Point

104 °C

PhysProp

from DrugBank

92 to 96 °C

from EU Food Improvement Agents

93.5 deg C

Lide, D.R. CRC Handbook of Chemistry and Physics 88TH Edition 2007-2008. CRC Press, Taylor & Francis, Boca Raton, FL 2007, p. 3-522

from HSDB

93.5 °C

from Human Metabolome Database (HMDB)

4.2.6 Solubility

Very soluble in water, sparingly soluble in ethanol

from EU Food Improvement Agents

Very soluble in water, pyridene, ethanol

Lide, D.R. CRC Handbook of Chemistry and Physics 88TH Edition 2007-2008. CRC Press, Taylor & Francis, Boca Raton, FL 2007, p. 3-522

from HSDB

642 mg/mL

= 642 gm / L
= 64 gm / 100 gm

from Human Metabolome Database (HMDB)

4.2.7 Vapor Pressure

2.47X10-3 mm Hg at 25 deg C (est)

US EPA; Estimation Program Interface (EPI) Suite. Ver. 4.1. Jan, 2010. Available from, as of Jul 11, 2011: http://www.epa.gov/oppt/exposure/pubs/episuitedl.htm

from HSDB

4.2.8 LogP

log Kow = -2.56 (est)

US EPA; Estimation Program Interface (EPI) Suite. Ver. 4.1. Jan, 2010. Available from, as of Jul 11, 2011: http://www.epa.gov/oppt/exposure/pubs/episuitedl.htm

from HSDB

4.2.9 Stability

Xylitol is stable to heat but is marginally hygroscopic. Caramelization can occur only if it is heated for several minutes near ist boiling point. ... Milled and specialized granulated grades of xylitol have a tendency to cake and should therefore be used within 9 to 12 months. Aqueous xylitol solutions have been reported to be stable, even on prolonged heating and storage.

Rowe, R.C., Sheskey, P.J., Quinn, M.E.; (Eds.), Handbook of Pharmaceutical Excipients 6th edition Pharmaceutical Press, London, England 2009, p. 788

from HSDB

4.2.10 Decomposition

Since xylitol is not utilized by most microoragnisms, products made with xylitol are usually safe from fermentation and microbial spoilage.

Rowe, R.C., Sheskey, P.J., Quinn, M.E.; (Eds.), Handbook of Pharmaceutical Excipients 6th edition Pharmaceutical Press, London, England 2009, p. 788

from HSDB

4.2.11 pH

5 to 7 (10 % w/v aqueous solution)

from EU Food Improvement Agents

4.3 Spectral Properties

MASS: 63599 (NIST/EPA/MSDC Mass Spectral Database, 1990 version)

Lide, D.R., G.W.A. Milne (eds.). Handbook of Data on Organic Compounds. Volume I. 3rd ed. CRC Press, Inc. Boca Raton ,FL. 1994., p. V5: 5220

from HSDB

IR: 18012 (Sadtler Research Laboratories IR grating collection)

Lide, D.R., G.W.A. Milne (eds.). Handbook of Data on Organic Compounds. Volume I. 3rd ed. CRC Press, Inc. Boca Raton ,FL. 1994., p. V5: 5220

from HSDB

1H NMR: 8216 (Sadtler Research Laboratories spectral collection)

Lide, D.R., G.W.A. Milne (eds.). Handbook of Data on Organic Compounds. Volume I. 3rd ed. CRC Press, Inc. Boca Raton ,FL. 1994., p. V5: 5220

from HSDB

4.3.1 1D NMR Spectra

1D NMR Spectra: 1 of 5 (1H NMR Spectra)	
1H NMR Spectra	1D NMR Spectrum 1912 - Bruker 600 MHz 1H NMR

from Human Metabolome Database (HMDB)

1D NMR Spectra: 2 of 5 (13C NMR Spectra)	
Copyright	Copyright © 2006-2017 Bio-Rad Laboratories, Inc. Portions provided by BioMagResBank(BMRB) and the Board of Regents of the University of Wisconsin System. All Rights Reserved.
Thumbnail	CLICK TO LOAD...

from SpectraBase

1D NMR Spectra: 3 of 5 (1H NMR Spectra)	
Copyright	Copyright © 2006-2017 Bio-Rad Laboratories, Inc. Portions provided by BioMagResBank(BMRB) and the Board of Regents of the University of Wisconsin System. All Rights Reserved.

1D NMR Spectra: 3 of 5 (1H NMR Spectra)	
Thumbnail	CLICK TO LOAD...

from SpectraBase

View All 5 1D NMR Spectra

4.3.2 2D NMR Spectra

2D NMR Spectra: 1 of 1 (1H-13C NMR Spectra)	
1H-13C NMR Spectra	2D NMR Spectrum 1847 - Bruker 600 MHz 1H-13C HSQC

from Human Metabolome Database (HMDB)

4.3.3 Mass Spectrometry

4.3.3.1 GC-MS

1. GC-MS Spectrum 1369 - GC-MS (5 TMS)
2. GC-MS Spectrum 16309
3. GC-MS Spectrum 31433
4. GC-MS Spectrum 31901
5. GC-MS Spectrum 32322
6. GC-MS Spectrum 38496

from Human Metabolome Database (HMDB)

4.3.3.2 MS-MS

1. MS-MS Spectrum 2160 - Quattro_QQQ 10V Positive delivery=Flow_Injection analyzer=Triple_Quad
2. MS-MS Spectrum 2161 - Quattro_QQQ 25V Positive delivery=Flow_Injection analyzer=Triple_Quad
3. MS-MS Spectrum 2162 - Quattro_QQQ 40V Positive delivery=Flow_Injection analyzer=Triple_Quad
4. MS-MS Spectrum 178428
5. MS-MS Spectrum 178429
6. MS-MS Spectrum 178430

7. MS-MS Spectrum 180747
8. MS-MS Spectrum 180748
9. MS-MS Spectrum 180749

from Human Metabolome Database (HMDB)

1 of 2

NIST Number	1051903
Instrument Type	IT/ion trap
Spectrum Type	MS2
Precursor Type	[M+H]+
Precursor m/z	153.0758
Total Peaks	19
m/z Top Peak	135
m/z 2nd Highest	117.1
m/z 3rd Highest	125.1
Thumbnail	CLICK TO LOAD...

from NIST

5 Related Records

CLICK TO LOAD...

from NCBI

5.1 Related Compounds with Annotation

CLICK TO LOAD...

from PubChem

5.2 Related Compounds

Same Connectivity	8 records
Same Isotope	6 records
Same Parent, Connectivity	40 records
Same Parent, Isotope	38 records
Same Parent, Exact	23 records
Mixtures, Components, and Neutralized Forms	116 records
Similar Compounds	265 records
Similar Conformers	515 records

from PubChem

5.3 Substances

5.3.1 Related Substances

All	357 records
Same	219 records
Mixture	138 records

from PubChem

5.3.2 Substances by Category

CLICK TO LOAD...

from PubChem

5.4 Entrez Crosslinks

PubMed	1397 records
Protein Structures	39 records
Taxonomy	3 records
Gene	3 records

from PubChem

6 Chemical Vendors

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from PubChem

7 Drug and Medication Information

7.1 Clinical Trials

Download

1 to 5 of 5

Record ID	Title	Status	Phase
NCT03229551	Xylitol for Chronic Sinusitis	Not yet recruiting	3
NCT02036151	Impact of Maternal Xylitol Consumption on Mutans Sterptococci	Completed	4
NCT01355796	Inhaled Xylitol Versus Saline in Stable Subjects With Cystic Fibrosis	Completed	2
NCT00928135	Aerosolized Hypertonic Xylitol Versus Hypertonic Saline in Cystic Fibrosis (CF) Subjects	Active, not recruiting	2
NCT00924404	Xylitol Versus Saline in Chronic Sinusitis	Completed	

from ClinicalTrials.gov

7.2 Therapeutic Uses

Sweetening Agents

National Library of Medicine's Medical Subject Headings online file (MeSH, 2009)

from HSDB

8 Food Additives and Ingredients

8.1 Food Additive Classes

JECFA Functional Classes

Food Additives: HUMECTANT; SWEETENER

from FAO/WHO Food Additive Evaluations - JECFA

8.2 Evaluations of the Joint FAO/WHO Expert Committee on Food Additives - JECFA

Evaluations of the Joint FAO/WHO Expert Committee on Food Additives - JECFA: 1 of 1 (JECFA Chemical)	
Chemical Name	XYLITOL
ADI	NOT SPECIFIED
Evaluation Year	1983
Report	TRS 696-JECFA 27/23

from FAO/WHO Food Additive Evaluations - JECFA

9 Pharmacology and Biochemistry

9.1 MeSH Pharmacological Classification

Sweetening Agents

Substances that sweeten food, beverages, medications, etc., such as sugar, saccharine or other low-calorie synthetic products. (From Random House Unabridged Dictionary, 2d ed)
See a list of PubChem compounds matching this category.

from MeSH

9.2 Absorption, Distribution and Excretion

Five healthy human volunteers (two males and three females) received an orange flavored drink containing 30 g xylitol with breakfast. Before dosing, urine was collected for 24 hr and collections were continued 24 hr after the dose of xylitol. During the collection period no foods rich in oxalate were permitted. No significant changes in urinary oxalate excretion could be detected.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 18: Xylitol (87-99-0) (1983). Available from, as of July 11, 2011: http://www.inchem.org/pages/jecfa.html

from HSDB

Diets containing 20% of xylitol or one of the following carbohydrates: glucose, fructose, sucrose, xylose, sorbitol or mannitol were fed to groups of five Wistar rats for seven days. The rats were fasted for 12 hr and given a 5 uCi dose of U(14)C-oxalic acid mixed with 0.625 g/kg of xylitol or respective carbohydrate. Urine and feces were collected for 72 hr and counted for recovery of activity. Ten rats were gradually adapted to 20% xylitol diets. After a 12-hr fast these rats and 20 controls received 5 uCi of U(14)C-oxalic acid mixed with water only or together with 0.625 g/kg xylitol/body weight. Urine and feces were collected from five rats/group; tail vein blood from the other rats at intervals up to 24 hours. Urinary excretion of the label was virtually identical in all groups. The mean excretion of label in feces of control rats receiving oxalic acid was significantly lower ($P < 0.001$) than in control rats receiving oxalate alone, or xylitol adapted rats receiving oxalate with xylitol (fecal recoveries were 77.8 and 83% respectively). The urinary excretion of label was also significantly higher among control rats receiving oxalate with xylitol when compared to control rats receiving oxalate alone. However, xylitol adapted rats excreted a significantly smaller proportion of oxalate in urine compared to controls receiving oxalate alone. The mean plasma levels of radioactivity in control rats receiving oxalic acid with xylitol were significantly higher ($P < 0.05$) immediately after the start of the study when compared to controls receiving oxalic acid with water only or xylitol adapted rats. When samples of plasma, urine and feces were analyzed by use of thin layer chromatography, the major part of the radioactivity was recovered as oxalic acid.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 18: Xylitol (87-99-0) (1983). Available from, as of July 11, 2011: http://www.inchem.org/pages/jecfa.html

from HSDB

Male (30) and female (20) CD-1 mice were either gradually adapted to 20% xylitol diets or fed a control diet. After a 12-hour fast the mice received a single oral dose of 2 uCi of U(14)C-oxalic acid in water or in an xylitol solution (a total dose of 0.625 g/kg bw). For five xylitol adapted male mice the oxalic acid dose was given with sorbitol (0.625 g/kg bw) and for another group of five with mannitol (0.625 g/kg bw). Urine and feces were collected at intervals for 72 hours to monitor the excretion of the label. ... Adaptation of male mice to 20% dietary xylitol increased the urinary excretion of the label fourfold (4.5 versus 20%). No major changes were seen in fecal excretion. Both sorbitol and mannitol increased the urinary excretion of the label while only sorbitol also affected fecal excretion of the label. Urinary excretion of oxalic acid was significantly higher in xylitol adapted mice when compared to controls receiving oxalic acid only. Even greater urinary recovery of label was observed in control mice receiving oxalic acid with xylitol. In female mice xylitol appeared to induce an even more pronounced increase in oxalic acid excretion.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 18: Xylitol (87-99-0) (1983). Available from, as of July 11, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

Six groups of three Sprague-Dawley male weanling rats received semi-synthetic diets for 28 days; four of the groups were pyridoxine deficient. Diets contained either 50% glucose by weight or 30% glucose and 20% fructose. On day 28 the rats were injected three times at spaced intervals with either 15% glucose; 10% xylitol + 5% glucose; 15% fructose; or 10% xylitol + 5% fructose. Urine was collected on days 28 and 29 and rats were sacrificed and livers collected 30 minutes after a final injection on day 30. The final body weights for the rats show that the group with the best growth received fructose + xylitol injections and was pyridoxine adequate (mean weight 216.6 g) versus the group receiving fructose + fructose injection (15%) and were pyridoxine deficient (119.7 g). The poorest growing groups received glucose diets only and either xylitol or glucose and glucose injections, and were pyridoxine deficient (107.1 g, 103.1 g). The rats on the pyridoxine deficient diets tended to excrete more oxalate and have higher liver oxalate levels. Within the pyridoxine deficient group only, rats injected with xylitol tended to excrete more oxalate and have higher liver oxalate levels, but these differences were not significant. Fructose had no effect on oxalate excretion or liver oxalate levels in the rats injected with xylitol.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 18: Xylitol (87-99-0) (1983). Available from, as of July 11, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

Five groups of four BLV/HA male weanling mice (initial weight of 10 g) were adapted to 10 or 20% xylitol administered in a semi-synthetic diet with glucose beginning at 5% xylitol level with 45% glucose and increasing the xylitol level by 5%, while decreasing glucose by 5% stepwise every other day until the desired xylitol dietary level was achieved. Two control groups received glucose only. Three of the groups received similar diets which were deficient in pyridoxine. The diets were fed for 25 days to establish a pyridoxine deficient condition; urine and feces were collected for one week, and body weights were determined weekly. The mice were then sacrificed and the livers collected. The pyridoxine adequate group fed glucose only as a sugar source grew best (final mean weight 25 g). The three xylitol groups (10%, 20% dietary levels, pyridoxine deficient; 20% pyridoxine adequate) gained less weight and had approximately similar mean weights (20 g); the glucose, pyridoxine deficient group did not gain weight and had three deaths before the end of the study. The 20% xylitol, pyridoxine deficient group excreted the highest level of urinary oxalate, with the 10% xylitol deficient and 20% xylitol, pyridoxine adequate group excreting intermediate amounts and the glucose, pyridoxine adequate group the lowest levels, whereas the two groups fed 20% xylitol had higher urinary levels.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 18: Xylitol (87-99-0) (1983). Available from, as of July 11, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/In humans/ xylitol is absorbed from the intestine very slowly compared to glucose. After adaptation no significant rise of the absorption rate was observed and although the absorption increased with administrated dose of xylitol the percentage of absorption decreased. The absorption of xylitol ranged from 49 to 95% depending on the dose level. After both oral and intravenous administration of xylitol subjects showed a fast distribution in the extracellular compartment and the tissues. ... The excretion of (14)C-activity in urine is low after oral administration of (14)C-xylitol. An excretion of 1-3% in urine is recorded after oral administration and of 10% after intravenous infusion of xylitol. In feces also 1% of xylitol was excreted.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

Twelve groups of 8 BLU-HA male weanling mice received one of six different dietary levels of xylitol (0, 10, 12.5, 17.5 and 20%) with (20%) or without (O) fructose at each xylitol level. The mice were adapted to the xylitol diets by daily increment of 2.5% each day until the specified level was reached. The mice were sacrificed after 22 days of feeding, and liver, brain, bladder, right kidney and right thigh muscle were collected. The presence of xylitol in the diet resulted in a slight but significant increase in weight gain. Fructose had no effect on weight gain. There was a significant effect of xylitol on oxalate levels in brain and muscle but not in the liver. There was no consistent response of oxalate levels to xylitol dose. Fructose also had a significant effect on brain and muscle oxalate levels, but again there was no consistent trend. Less than half of the kidney samples had measurable levels of oxalate and none of the bladder samples had detectable levels of oxalate (1.0 ug/g wet weight).

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 18: Xylitol (87-99-0) (1983). Available from, as of July 11, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

The rate of expired xylitol in rats had a maximum value of 27 mg xylitol/hr/kg body weight for the adapted animals and 9 mg xylitol/hr/kg body weight for the unadapted animals ... The activity of the oxidized xylitol was present for the greatest part as (14)CO2 (68% of the administered dose) in the expired air, the urea present in the urine provided around 2% of the activity. The adaptive improvement in the rate of absorption had in all probabilities no effect on the retention of activity in the carcass. Approximately one quarter of the (14)C-dosage administered as xylitol was retained.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

Insulin stimulated the uptake of (14)C in the adipose tissue and the diaphragma of the rat, which indicates the transport action on the from (14)C-xylitol derived (14)C-glucose. ...

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

... In (14)C-xylitol unadapted /rats/ fed 250 mg xylitol/kg body weight excreted 3-8% of the activity in urine and 61-62% exhaled, while 6-10% excreted in feces and 20-25% found in the animal after a period of 16-24 hrs. Amounts of 0.3.-0.4% of the dose were found in the glycogen of the liver and muscle. ...

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

9.3 Metabolism/Metabolites

The biochemical pathways for formation of oxalate after intravenous injection of xylitol in humans were studied using enzymes derived from human liver. It was concluded that metabolic pathways based on a combination of the transketolase, fructokinase, and aldolase reactions can account for the production of glucose, lactate, tertronates (D-threonic and D-erythronic acids) and oxalate (precursors) during the metabolism of xylitol administered parenterally.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 18: Xylitol (87-99-0) (1983). Available from, as of July 11, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

After xylitol ingestion increase of serum lactate concentration and lactate-pyruvate ratio was observed, but to a degree less than after glucose. /Investigators/ also found a marked increase of alpha-dihydroxybutyrate. Complete metabolism of xylitol produces 35 equivalents of ATP compared to 32 from glucose.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/In humans/ exogenous xylitol enters the pathway by conversion to D-xylulose by a nonspecific cytoplasmic polyol dehydrogenase. Phosphorylation then yields D-xylulose phosphate, the link between the glucuronic acid and the pentose phosphate pathways; the latter leads to the formation of glyceraldehyde-3-phosphate and fructose-6-phosphate, intermediate metabolites of the Embden-Meyerhof (glycolytic) pathway. Thus xylitol can be metabolized via glucose-6-phosphate to glycogen and pyruvate or lactate via the citric acid cycle to CO2. Xylitol is mainly metabolized in the liver (80% to glucose only 20%) but a small amount also in kidney, myocardium, erythrocytes, adrenal, brain, lungs and adipose tissue. Exogenous xylitol can be metabolized in large quantities, intravenously 0.4 gm/kg/hour or 40 g/day orally raises the plasma level to a maximum of 1.5-16 mg/100 mL. The metabolic rate for xylitol is identical in both healthy and diabetic or uraemic patients and patients who suffered from liver diseases.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

In /human/ studies with (14)C-xylitol 90% of C-atoms taken up could be recovered in products and intermediates of the glycolytic and pentose phosphate pathway.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

The metabolism of xylitol in rats: ... Xylitol is first oxidized to D-xylulose by the NAD-xylitol dehydrogenase, causing the NADH/NAD ratio to increase. The next step is the phosphorylation of D-xylulose to D-xylulose-5-phosphate by D-xylulose-kinase. D-xylulose-5-phosphate is an intermediate of the pentose phosphate shunt and it is metabolized to fructose-6-phosphate and glyceraldehyde phosphate by this pathway. Three molecules xylitol yield two molecules of fructose-6-phosphate and one molecule of glyceraldehyde phosphate. Fructose-6-phosphate can readily be converted to glucose and glycogen; glyceraldehyde phosphate either to glucose, glycogen or lactate. Most of the xylitol is rapidly converted to glucose and only small quantities are converted to lactate. ...

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

In rats 80% of the xylitol is degraded in the liver, the remaining 20% is metabolized in extrahepatic organs, mainly in the kidneys.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

In rats xylitol is fast converted into glucose and the peripheral metabolism of xylitol follows only after the converting into glucose and by the influence of insulin. Five minutes after the injection of (14)C-xylitol more than 50% of the (14)C is circulating in the blood as glucose. Insulin is not needed for the uptake of (14)C-xylitol, but for the incorporation of (14)C-glucose in the extrahepatic organs. The erythrocytes and the adipose tissue are able to metabolize xylitol, although this is negligible in comparison to the metabolism in the liver.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

When incubating rat liver homogenate with [U- (14)C] glucose or [U-(14)C] xylitol as substrates in the absence of oxidants no oxalate was formed. Addition of NAD and phenazine methosulfate to the incubation medium led to an increased substrate uptake and CO2 production. Under these conditions oxalate formation was observed from both substrates the oxalate production from xylitol being 1.6 times higher than that from glucose.

Hauschildt S, Brand K; Biochemical Medicine 21 (1): 55-61 (1979)

▸ *from HSDB*

9.4 Biological Half-Life

/In humans/ the initial fast distribution phase /for xylitol/ had a half life of about four minutes, while the apparent half life of elimination was approximately 20 minutes.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

(14)C-xylitol disappeared extremely rapidly and the calculated half life is 165 seconds. ...

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

The half-life of exhalation of (14)CO2 after feeding with xylitol in unadapted /rats/ is 295 minutes, whereas rats which were adapted for 14 days to xylitol had a half-life of 237 minutes...

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

...Rats were administered (14)C-xylitol (250 mg) by intubation, the half-live of the resorption of xylitol was about 7 to 8 hr. The resorption rate is about 15-20% of that of glucose. After the animals had been fed xylitol for 14 days the half-life fell to 4.5 hr. ...

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

9.5 Human Metabolite Information

9.5.1 Metabolite Description

Xylitol is a five-carbon sugar alcohol that is obtained through the diet. It is not endogenously produced by humans. Xylitol is used as a diabetic sweetener which is roughly as sweet as sucrose with 33% fewer calories. Xylitol is naturally found in many fruits (strawberries, plums, raspberries) and vegetables (e. g. cauliflower). Because of fruit and vegetable consumption the human body naturally processes 15 grams of xylitol per day. Xylitol can be produced industrially starting from primary matters rich in xylan which is hydrolyzed to obtain xylose. It is extracted from hemicelluloses present in the corn raids, the almond hulls or the barks of birch (or of the by-products of wood: shavings hard, paper pulp). Of all polyols, it is the one that has the sweetest flavor (it borders that of saccharose). It gives a strong refreshing impression, making xylitol an ingredient of choice for the sugarless chewing gum industry. In addition to his use in confectionery, it is used in the pharmaceutical industry for certain mouthwashes and toothpastes and in cosmetics (creams, soaps, etc.). Xylitol is produced starting from xylose, the isomaltose, by enzymatic transposition of the saccharose (sugar). Xylitol is not metabolized by cariogenic (cavity-causing) bacteria and gum chewing stimulates the flow of saliva; as a result, chewing xylitol gum may prevent dental caries. Chewing xylitol gum for 4 to 14 days reduces the amount of dental plaque. The reduction in the amount of plaque following xylitol gum chewing within 2 weeks may be a transient phenomenon. Chewing xylitol gum for 6 months reduced mutans streptococci levels in saliva and plaque in adults (PMID: 17426399, 15964535). Studies have also shown xylitol chewing gum can help prevent acute otitis media (ear aches and infections) as the act of chewing and swallowing assists with the disposal of earwax and clearing the middle ear, while the presence of xylitol prevents the growth of bacteria in the eustachian tubes. Xylitol is well established as a life-threatening toxin to dogs. The number of reported cases of xylitol toxicosis in dogs has significantly increased since the first reports in 2002. Dogs that have ingested foods containing xylitol (greater than 100 milligrams of xylitol consumed per kilogram of bodyweight) have presented with low blood sugar (hypoglycemia), which can be life-threatening. Xylitol is found to be associated with ribose-5-phosphate isomerase deficiency, which is an inborn error of metabolism.

▸ *from Human Metabolome Database (HMDB)*

9.5.2 Biofluid Locations

1. Blood
2. Cerebrospinal Fluid (CSF)

3. Feces
4. Saliva
5. Urine

from Human Metabolome Database (HMDB)

9.5.3 Tissue Locations

Prostate

from Human Metabolome Database (HMDB)

9.5.4 Cellular Locations

Cytoplasm (predicted from logP)

from Human Metabolome Database (HMDB)

9.5.5 Associated Disorders and Diseases

Ribose-5-phosphate isomerase deficiency

Huck JH, Verhoeven NM, Struys EA, Salomons GS, Jakobs C, van der Knaap MS: Ribose-5-phosphate isomerase deficiency: new inborn error in the pentose phosphate pathway associated with a slowly progressive leukoencephalopathy. Am J Hum Genet. 2004 Apr;74(4):745-51. Epub 2004 Feb 25.[PMID:14988808]

from Human Metabolome Database (HMDB)

10 Use and Manufacturing

10.1 Uses

Food additives

from EU Food Improvement Agents

JECFA Functional Classes

Food Additives: HUMECTANT; SWEETENER

from FAO/WHO Food Additive Evaluations - JECFA

10.2 Methods of Manufacturing

... Produced on a commercial scale by catalytic reduction of D-xylose with hydrogen in the presence of Raney nickel. D-Xylose is obtained by acid-catalyzed hydrolysis of xylan-containing plant materials such as birch wood, corn cobs, and straw. Since these natural raw materials also contain other carbohydrates, xylitol must be separated from other polyols by chromatographic techniques.

Schiweck H et al; Ullmann's Encyclopedia of Industrial Chemistry 7th ed. (1999-2011). New York, NY: John Wiley & Sons; Sugar Alcohols. Online Posting Date: May 30, 2011

from HSDB

10.3 Formulations/Preparations

Trade Names: Eutrit; Kannit; Klinit; Kylit; Newtol; Torch; Xyliton.

O'Neil, M.J. (ed.). The Merck Index - An Encyclopedia of Chemicals, Drugs, and Biologicals. Whitehouse Station, NJ: Merck and Co., Inc., 2006., p. 1738

from HSDB

10.4 U.S. Production

Production volumes for non-confidential chemicals reported under the Inventory Update Rule.

Year	Production Range (pounds)
1986	No Reports
1990	No Reports
1994	10 thousand - 500 thousand
1998	No Reports
2002	10 thousand - 500 thousand

US EPA; Non-confidential Production Volume Information Submitted by Companies for Chemicals Under the 1986-2002 Inventory Update Rule (IUR). Xylitol (87-99-0). Available from, as of July 5, 2011: http://www.epa.gov/oppt/iur/tools/data/2002-vol.html

from HSDB

11 Identification

11.1 Analytic Laboratory Methods

... High-pressure liquid chromatography yields the most satisfactory results.

Schiweck H et al; Ullmann's Encyclopedia of Industrial Chemistry 7th ed. (2010). NY, NY: John Wiley & Sons; Sugar Alcohols. Online Posting Date: May 30, 2011

▸ *from HSDB*

11.2 Clinical Laboratory Methods

... High-pressure liquid chromatography yields the most satisfactory results.

Schiweck H et al; Ullmann's Encyclopedia of Industrial Chemistry 7th ed. (2010). NY, NY: John Wiley & Sons; Sugar Alcohols. Online Posting Date: May 30, 2011

▸ *from HSDB*

12 Safety and Hazards

12.1 Hazards Identification

12.1.1 Skin, Eye, and Respiratory Irritations

Xylitol may... also be an irritant to the eyes. Eye protection and gloves are recommended. Conventional dust-control practices should be employed.

Rowe, R.C., Sheskey, P.J., Quinn, M.E.; (Eds.), Handbook of Pharmaceutical Excipients 6th edition Pharmaceutical Press, London, England 2009, p. 788

from HSDB

12.2 Fire Fighting Measures

Water spray, dry chemical, carbon dioxide, or foam as appropriate for surrounding fire and materials.

United States Pharmacopeial Convention, Inc (USP); MSDS Database Online; Material Safety Data Sheet: Xylitol; Catalog Number: 1720600; (Revision Date: November 13, 2009)

from HSDB

As with all fires, evacuate personnel to a safe area. Firefighters should use self-contained breathing equipment and protective clothing.

United States Pharmacopeial Convention, Inc (USP); MSDS Database Online; Material Safety Data Sheet: Xylitol; Catalog Number: 1720600; (Revision Date: November 13, 2009)

from HSDB

12.3 Accidental Release Measures

12.3.1 Cleanup Methods

Wear approved respiratory protection, chemically compatible gloves, and protective clothing. Wipe up spillage or collect spillage using a high-efficiency vacuum cleaner. Avoid breathing dust. Place spillage in appropriately labeled container for disposal. Wash spill site.

United States Pharmacopeial Convention, Inc (USP); MSDS Database Online; Material Safety Data Sheet: Xylitol; Catalog Number: 1720600; (Revision Date: November 13, 2009)

from HSDB

12.3.2 Disposal Methods

SRP: Criteria for land treatment or burial (sanitary landfill) disposal practices are subject to significant revision. Prior to implementing land disposal of waste residue (including waste sludge), consult with environmental regulatory agencies for guidance on acceptable disposal practices.

from HSDB

12.3.3 Other Preventative Measures

SRP: The scientific literature for the use of contact lenses by industrial workers is inconsistent. The benefits or detrimental effects of wearing contact lenses depend not only upon the substance, but also on factors including the form of the substance, characteristics and duration of the exposure, the uses of other eye protection equipment, and the hygiene of the lenses. However, there may be individual substances whose irritating or corrosive properties are such that the wearing of contact lenses would be harmful to the eye. In those specific cases, contact lenses should not be worn. In any event, the usual eye protection equipment should be worn even when contact lenses are in place.

from HSDB

This material is assumed to be combustible. As with all dry powders, it is advisable to ground mechanical equipment in contact with dry material to dissipate the potential buildup of static electricity.

United States Pharmacopeial Convention, Inc (USP); MSDS Database Online; Material Safety Data Sheet: Xylitol; Catalog Number: 1720600; (Revision Date: November 13, 2009)

from HSDB

As a general rule, when handling USP Reference Standards, avoid all contact and inhalation of dust, mists, and/or vapors associated with this material. Clean equipment and work surfaces with suitable detergent or solvent after use. After removing gloves, wash hands and other exposed skin thoroughly.

United States Pharmacopeial Convention, Inc (USP); MSDS Database Online; Material Safety Data Sheet: Xylitol; Catalog Number: 1720600; (Revision Date: November 13, 2009)

from HSDB

Chemically compatible. For handling solutions, ensure that the glove material is protective against the solvent being used. Use handling practices that minimize direct hand contact. Employees who are sensitive to natural rubber (latex) should use nitrile or other synthetic nonlatex gloves. Use of powdered latex gloves should be avoided due to the risk of latex allergy.

United States Pharmacopeial Convention, Inc (USP); MSDS Database Online; Material Safety Data Sheet: Xylitol; Catalog Number: 1720600; (Revision Date: November 13, 2009)

from HSDB

Safety glasses with sideshields are recommended. Face shields or goggles may be required if splash potential exists or if corrosive materials are present. Approved eye protection (e.g., bearing the ANSI Z87 or CSA stamp) is preferred. Maintain eyewash facilities in the work area.

United States Pharmacopeial Convention, Inc (USP); MSDS Database Online; Material Safety Data Sheet: Xylitol; Catalog Number: 1720600; (Revision Date: November 13, 2009)

from HSDB

For handling of laboratory scale quantities, a cloth lab coat is recommended. Where significant quantities are handled, work clothing may be necessary to prevent take-home contamination.

United States Pharmacopeial Convention, Inc (USP); MSDS Database Online; Material Safety Data Sheet: Xylitol; Catalog Number: 1720600; (Revision Date: November 13, 2009)

from HSDB

12.4 Handling and Storage

12.4.1 Storage Conditions

Store in tight container as defined in the USP-NF. This material should be handled and stored per label instructions to ensure product integrity.

United States Pharmacopeial Convention, Inc (USP); MSDS Database Online; Material Safety Data Sheet: Xylitol; Catalog Number: 1720600; (Revision Date: November 13, 2009)

▸ *from HSDB*

Crystalline /xylitol/ is stable for at least 3 years if stored at less than 65% relative humidity and 25 deg C. ... Xylitol should be stored in a well-closed container in a cool, dry place.

Rowe, R.C., Sheskey, P.J., Quinn, M.E.; (Eds.), Handbook of Pharmaceutical Excipients 6th edition Pharmaceutical Press, London, England 2009, p. 788

▸ *from HSDB*

12.5 Exposure Control and Personal Protection

12.5.1 Protective Equipment and Clothing

Airborne exposure should be controlled primarily by engineering controls such as general dilution ventilation, local exhaust ventilation, or process enclosure. Local exhaust ventilation is generally preferred to general exhaust because it can control the contaminant at its source, preventing dispersion into the work area. An industrial hygiene survey involving air monitoring may be used to determine the effectiveness of engineering controls. Effectiveness of engineering controls intended for use with highly potent materials should be assessed by use of nontoxic surrogate materials.

United States Pharmacopeial Convention, Inc (USP); MSDS Database Online; Material Safety Data Sheet: Xylitol; Catalog Number: 1720600; (Revision Date: November 13, 2009)

▸ *from HSDB*

Where respirators are deemed necessary to reduce or control occupational exposures, use NIOSH-approved respiratory protection and have an effective respirator program in place (applicable U.S. regulation OSHA 29 CFR 1910.134).

United States Pharmacopeial Convention, Inc (USP); MSDS Database Online; Material Safety Data Sheet: Xylitol; Catalog Number: 1720600; (Revision Date: November 13, 2009)

▸ *from HSDB*

12.6 Regulatory Information

12.6.1 FDA Requirements

Xylitol is a food additive permitted for direct addition to food for human consumption, as long as 1) the quantity of the substance added to food does not exceed the amount reasonably required to accomplish its intended physical, nutritive, or other technical effect in food, and 2) any substance intended for use in or on food is of appropriate food grade and is prepared and handled as a food ingredient.

21 CFR 172.395 (USFDA); U.S. National Archives and Records Administration's Electronic Code of Federal Regulations. Available from, as of July 5, 2011: http://www.ecfr.gov

▸ *from HSDB*

Food labeling. Health claims: dietary noncariogenic carbohydrate sweeteners and dental caries. ... Eligible noncariogenic carbohydrate sweeteners are the sugar alcohols xylitol, sorbitol, mannitol, maltitol, isomalt, lactitol, hydrogenated starch hydrolysates, hydrogenated glucose syrups, and erythritol, or a combination of these.

21 CFR 101.80 (USFDA); U.S. National Archives and Records Administration's Electronic Code of Federal Regulations. Available from, as of July 11, 2011: http://www.ecfr.gov

from HSDB

13 Toxicity

13.1 Toxicological Information

13.1.1 Interactions

Porphyromonas gingivalis is one of the suspected periodontopathic bacteria. The lipopolysaccharide (LPS) of P. gingivalis is a key factor in the development of periodontitis. Inflammatory cytokines play important roles in the gingival tissue destruction that is a characteristic of periodontitis. Macrophages are prominent at chronic inflammatory sites and are considered to contribute to the pathogenesis of periodontitis. Xylitol stands out and is widely believed to possess anticaries properties. However, to date, little is known about the effect of xylitol on periodontitis. The aim of the present study was to determine tumor necrosis factor alpha (TNF-alpha) and interleukin-1beta (IL-1beta) expression when RAW 264.7 cells were stimulated with P. gingivalis LPS (hereafter, LPS refers to P. gingivalis LPS unless stated otherwise) and the effect of xylitol on the LPS-induced TNF-alpha and IL-1beta expression. The kinetics of TNF-alpha and IL-1beta levels in culture supernatant after LPS treatment showed peak values at 1 hr (TNF-alpha) and 2 to 4 hr (IL-1beta), respectively. NF-kappaB, a transcription factor, was also activated by LPS treatment. These cytokine expressions and NF-kappaB activation were suppressed by pretreatment with pyrrolidine dithiocarbamate (an inhibitor of NF-kappaB). Pretreatment with xylitol inhibited LPS-induced TNF-alpha and IL-1beta gene expression and protein synthesis. LPS-induced mobilization of NF-kappaB was also inhibited by pretreatment with xylitol in a dose-dependent manner. Xylitol also showed inhibitory effect on the growth of P. gingivalis. Taken together, these findings suggest that xylitol may have good clinical effect not only for caries but also for periodontitis by its inhibitory effect on the LPS-induced inflammatory cytokine expression.[Han SJ et al; Clin Diagn Lab Immunol 12 (11): 1285-1291 (2005)] Full text: PMC1287760
Abstract: PubMed

from HSDB

Oxalate levels in the plasma and urine fractions of fasted normal, oxythiamin treated (20 mg/kg) and 4-deoxypyridoxine treated (300 mg/kg) rabbits were determined following infusion with either xylitol or glucose at a dose of 2 g/kg body weight. Biochemical determinations showed that transient thiamin or pyridoxine deficient states had been induced in the antivitamin treated rabbits. In the first 24 hour following infusion with either carbohydrate, urinary oxalate levels remained within the normal range for all groups. Oxythiamin hastened the appearance of the transient, elevation in plasma oxalate concentrations seen in rabbits after infusion with glucose. After xylitol infusion, the elevation of plasma oxalate was not significantly above normal. 4-Deoxypyridoxine enhanced peak plasma oxalate levels above those of controls for both sugars. Glucose, at an equivalent dose to xylitol, resulted in higher plasma oxalate levels than xylitol for all groups. Infusions of [U-(14)C]xylitol and [U-(14)C]glucose solutions into 4-deoxypyridoxine treated rabbits demonstrated a conversion of the administered radioactive carbon into (14)C oxalate of 0.01% with a high dilution of the specific activity. The results suggest that oxalate production from xylitol is negligible; any toxicity related to xylitol administration is not a consequence of oxalate production.
Abstract: PubMed

Oshinsky RJ et al; J Nutr 107 (5): 792-804 (1977)

from HSDB

The absorption of (14)C -labelled oxalic acid was studied in Wistar rats, CD-1 mice and NMRI mice. Oxalic acid in solution was given to the animals by gavage either with water alone or with 0.625 g/kg b.w. of xylitol. Both xylitol-adapted animals and animals not previously exposed to xylitol were used. Adaption to xylitol diets enhanced the absorption and urinary excretion of the label (oxalic acid) in both strains of mice but not in rats. Earlier studies have indicated a high incidence of bladder calculi in mice but not in rats fed high amounts of xylitol. The results of the present study offer one likely explanation for the increased formation of bladder calculi as a result of oversaturation of urine with oxalate.

Salminen S et al; Toxicology Letters 44 (1-2): 113-120 (1988)

from HSDB

Xylitol was investigated for its ability to ameliorate hemolytic anemia induced by acetylphenylhydrazine in rabbits. Animal experiments were performed using two different concentrations of xylitol, a 5% and a 10% solution with a total dose of 2

g/kg body weight and infusion rates of 10 mg and 20 mg xylitol per kg body weight per minute respectively. Two doses of acetylphenylhydrazine (APH), 5 and 10 mg per kg, were injected intraperitoneally as hemolytic inducers in different groups of rabbits. All the rabbits infused with xylitol showed significantly less acute APH-induced hemolysis. The isotonic 5% xylitol solution was found to maintain and restore the hematological parameters (packed cell volume, hemoglobin concentration, reduced glutathione (GSH) content, and reticulocyte counts) better than the 10% xylitol solution. Increased (51)Cr-red cell survival confirmed the beneficial effect of xylitol. The survival of erythrocytes as represented by chromium-labeling in rabbits infused with 5% xylitol after treatment with 10 mg/kg APH increased from about 33% (the survival of red cells in rabbits injected with APH alone) to 67% of normal rabbits' red cell survival. Erythrocytes in APH-treated animals took up xylitol more readily than erythrocytes from control animals. Our results in rabbits suggest that (1) non-toxic dosage of xylitol is effective in ameliorating the hemolytic episode induced by APH, (2) there is a dose relationship between the hemolytic effect induced by APH and the preventive effect offered by xylitol, (3) drug-challenged cells effectively acquired two to three fold more xylitol to compensate for the cellular needs than that of the normal cells, and (4) sufficient xylitol (55 mg/dL) to act as substrate for xylitol dehydrogenase was recovered intracellularly in drug-challenged rabbit erythrocyte in vivo, in spite of a low plasma (<30 mg/dL) concentration of the substrate. This antihemolytic affect of xylitol is likely accomplished through NADPH generation, which maintains the level of GSH and protects the hemoglobin and other structural and functional proteins against peroxidative damage.

Ukab WA et al; Metabolism 30 (11): 1053-1059 (1981)

▸ *from HSDB*

The effects of oral administration of xylitol on the rate of ethanol elimination and on the ethanol-induced changes in blood concentrations of lactate and pyruvate were studied in seven healthy male subjects. Xylitol (1.0 g/kg body weight) was administered orally and ethanol (0.8 g/kg body weight) intravenously. In the control experiments glucose was given instead of xylitol. Xylitol had no significant effect on the rate of ethanol elimination or on the ethanol-induced increase in the blood lactate concentration. The ethanol-induced changes in the lactate/pyruvate ratio were not affected by xylitol. It is suggested that the ineffectiveness of xylitol is due to its low concentration in the liver after oral administration. Ethanol induced a 5-10-fold increase in the blood concentration of xylitol. This is most probably due to inhibition of xylitol oxidation in the liver by the ethanol-induced reduction in the hepatic redox state. The clinical significance of this finding is unknown.

Ylikahri RH, Leino T; Metabolism 28 (1): 25-29(1979)

▸ *from HSDB*

13.1.2 Antidote and Emergency Treatment

/SRP:/ Immediate first aid: Ensure that adequate decontamination has been carried out. If patient is not breathing, start artificial respiration, preferably with a demand valve resuscitator, bag-valve-mask device, or pocket mask, as trained. Perform CPR if necessary. Immediately flush contaminated eyes with gently flowing water. Do not induce vomiting. If vomiting occurs, lean patient forward or place on the left side (head-down position, if possible) to maintain an open airway and prevent aspiration. Keep patient quiet and maintain normal body temperature. Obtain medical attention. /Poisons A and B/

Currance, P.L. Clements, B., Bronstein, A.C. (Eds).; Emergency Care For Hazardous Materials Exposure. 3Rd edition, Elsevier Mosby, St. Louis, MO 2005, p. 160

▸ *from HSDB*

/SRP:/ Basic treatment: Establish a patent airway (oropharyngeal or nasopharyngeal airway, if needed). Suction if necessary. Watch for signs of respiratory insufficiency and assist ventilations if needed. Administer oxygen by nonrebreather mask at 10 to 15 L/min. Monitor for pulmonary edema and treat if necessary ... . Monitor for shock and treat if necessary ... . Anticipate seizures and treat if necessary ... . For eye contamination, flush eyes immediately with water. Irrigate each eye continuously with 0.9% saline (NS) during transport ... . Do not use emetics. For ingestion, rinse mouth and administer 5 mL/kg up to 200 mL of water for dilution if the patient can swallow, has a strong gag reflex, and does not drool ... . Cover skin burns with dry sterile dressings after decontamination ... . /Poisons A and B/

Currance, P.L. Clements, B., Bronstein, A.C. (Eds).; Emergency Care For Hazardous Materials Exposure. 3Rd edition, Elsevier Mosby, St. Louis, MO 2005, p. 160

▸ *from HSDB*

/SRP:/ Advanced treatment: Consider orotracheal or nasotracheal intubation for airway control in the patient who is unconscious, has severe pulmonary edema, or is in severe respiratory distress. Positive-pressure ventilation techniques with a bag valve mask device may be beneficial. Consider drug therapy for pulmonary edema ... . Consider administering a beta agonist such as albuterol for severe bronchospasm ... . Monitor cardiac rhythm and treat arrhythmias as necessary ... . Start IV administration of D5W /SRP: "To keep open", minimal flow rate/. Use 0.9% saline (NS) or lactated Ringer's if signs of hypovolemia are present. For hypotension with signs of hypovolemia, administer fluid cautiously. Watch for signs of fluid overload ... . Treat seizures with diazepam or lorazepam ... . Use proparacaine hydrochloride to assist eye irrigation ... . /Poisons A and B/

Currance, P.L. Clements, B., Bronstein, A.C. (Eds).; Emergency Care For Hazardous Materials Exposure. 3Rd edition, Elsevier Mosby, St. Louis, MO 2005, p. 160-1

▸ *from HSDB*

13.1.3 Human Toxicity Excerpts

/HUMAN EXPOSURE STUDIES/ ... The tolerance of increasing amounts of dietary xylitol in 13 healthy children, aged seven to 16 years was investigated. Xylitol was administered as a supplement in addition to the children's regular diet. The daily dose was increased during successive 10-day periods from 10 to 25, 45, 65 and 80 grams. Gastrointestinal symptoms (flatulence, occasional abdominal pain and diarrhea) were recorded daily throughout the study. Prior to xylitol supplementation and after 20-50 days of dietary supplement serum uric acid and total cholesterol were measured. Flatulence was the most common side effect occurring relatively infrequently in almost every other subject during the 45 g/day intake, and in most subjects with greater frequency at the 80 g/day intake. Transient diarrhea occurred in four children on 65 g xylitol/day and in one child at 80 g/day. After 50 days of xylitol consumption, there was an increase in serum uric acid and cholesterol. However, the values were within the normal ranges for children.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 18: Xylitol (87-99-0) (1983). Available from, as of July 11, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/HUMAN EXPOSURE STUDIES/ During fructose, sorbitol, and xylitol perfusions, carbohydrate utilization was studied by continuous indirect calorimetry and compared with glucose utilization during pharmacologic inhibition of endogenous insulin secretion. The experiment was performed in 28 normal volunteers divided into 5 groups (glucose, fructose, sorbitol, xylitol, and saline), each subject being its own control. Insulin suppression was obtained by means of a constant infusion of epinephrine (6 ug/min) and propranolol (0.08 mg/min). After 90 min, during plasma insulin steady state, each sugar or polyol was infused at a rate of 6 mg/kg/min for 120 min. In contrast with a rise in plasma glucose from 161 +/- 6 mg/dL to 291 +/- 14 mg/dL during glucose infusion, glucose levels remained unchanged during infusion of the glucose substitutes. Carbohydrate oxidation showed a rise of 24, 65, 76, and 44 mg/min during infusions of glucose, fructose, sorbitol, and xylitol, respectively. Lipid oxidation rates decreased by 7, 20, 33, and 23 mg/min during the same infusions. These results indicate that fructose, sorbitol, and xylitol are oxidized at a higher rate than glucose during suppression of endogenous insulin secretion, without any significant rise in glycemia.

de Kalbermatten N et al; Metabolism 299 (1): 62-67 (1980)

▸ *from HSDB*

/HUMAN EXPOSURE STUDIES/ A study was carried out on nine subjects who had consumed xylitol for 4.8 to 5.3 years. During the years 1972-1974 these individuals consumed amounts ranging from 376-2520 mg/kg/day, and at the end of 1977, from 46 to 354 mg/kg/day. In 1978 the diets of these individuals were loaded with 82.3 to 1400 mg/kg/day (females 70 g/day, males 100 g/day) for 14 days using a strictly controlled diet, and subsequently for seven days while on a normal diet. During these periods and also during period of normal diet + sucrose loading, the following plasma and urinary parameters were measured. For serum; alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, gamma-glutamyltranspeptidase, lactate dehydrogenase, amylase: blood acid base balance. For urine; Uric acid, oxalic acid, 3-methoxy-4-hydroxymandelic acid, catecholamines (adrenalin, noradrenalin), metanephrines (m-o-methylnorepinephrine and m-o-methylnorepinephrine) urine: deposits, sediments and microcrystals, specific gravity, pH, U.V. and visible spectrum, volume; acid excretion in urine; urinary electrolytes; as well as the usual hematological, plasma and urinary parameters. There were no significant changes in any of the serum or urinary parameters measured.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 18: Xylitol (87-99-0) (1983). Available from, as of July 11, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/HUMAN EXPOSURE STUDIES/ Eighteen diabetic children received 30 g dietary xylitol during four weeks. A significant elevation of the uric acid concentration in serum was observed, also significant increases of total protein content and of inorganic phosphorus. In these children no diarrhea was noticed.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/HUMAN EXPOSURE STUDIES/ During two to six weeks 30 g xylitol/day was given orally to 12 stable diabetics. The caloric intake and amount of carbohydrates was kept constant. Changes in fasting blood sugar, urine sugar, serum lipids, serum mucoprotein, SGOT and SGPT were studied before, during and after xylitol administration. In three of the 12 cases urinary sugar excretion disappeared. A slight impairment of glucose tolerance and a tendency to decreased SGOT and SGPT levels were observed. In some cases diarrhea was noticed, but no other effects of xylitol were noticed.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/HUMAN EXPOSURE STUDIES/ ... Xylitol was chronically intravenously infused to 12 mildly diabetic patients every morning for seven days. No changes were observed in fasting blood sugar, serum triglycerides, total cholesterol, serum electrolytes or ketone bodies. However, four cases indicated the marked decrease of urine sugar excretion during the experimental period. Xylitol was also given to two diabetic persons with ketosis and decreased the ketone bodies from 5.1 to 0.2 mg % within four hours.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/HUMAN EXPOSURE STUDIES/ /Researchers/ investigated the effects of acute intravenous infusion of 30 g/90 minutes on blood sugar, pyruvate and lactate, and on plasma free fatty acids and insulin levels in six normal and 11 diabetic persons. Xylitol infusion produced no change of blood sugar in normal and a slight increase in diabetic patients. Blood lactate showed a significant increase in the diabetic group but only a slight increase in the normal group. Blood ketone bodies and plasma free fatty acids decreased in both groups after xylitol administration. Plasma insulin increased slightly in five of the normal subjects and in four of the nine diabetic subjects.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/HUMAN EXPOSURE STUDIES/ Xylitol in concentration varying from 5 to 20 /mg/ in food was administered to 23 diabetic persons. Except for diarrhea in some cases no harmful side effects were observed. No negative influence on the diabetic metabolism was observed. Xylitol (25 g) dissolved in tea caused more frequent diarrhea and other abdominal side effects in the subjects.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/HUMAN EXPOSURE STUDIES/ Acute tests with xylitol were conducted on 13 recruited adult diabetics and the following parameters were determined: aceto-acetate, beta-hydroxy-butyrate, unesterized fatty acid, free glycerine and total lipids. A significant drop in concentrations of aceto-acetate and beta-hydroxybutyrate occurred under administration of xylitol as compared with the control experiments. Unesterized fatty acid behaved similarly. The concentration of free glycerine also clearly dropped at the end of the experiment. It can be assumed from the results that the diminished lipolyserate is the cause of reduced formation of ketone bodies. A drop in total lipids indicated that hyperlipidemia might possibly also be favorably influenced by xylitol.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/HUMAN EXPOSURE STUDIES/ ... 100 human young adult subjects were divided into S-(sucrose) group, and X-(xylitol) group. Consumption was 4.0 chewing gums per subject per day in the S-group and 4.5 in the X-group. Frequency of sucrose intake was 4.2 times per day in the S-group, and 4.9 in the X-group. The caries incidence was 2.92 in the S-group and -1.04 in the X-group. The results show a profound difference in the caries increment rate between the two experimental groups.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/HUMAN EXPOSURE STUDIES/ In a two year tolerance study three groups of volunteers (35, 38 and 52) remained on strict diet containing respectively, fructose, sucrose and xylitol. The average monthly amounts consumed in a varied assortment of foods were respectively 2.1, 2.2 and 1.5 kg. The highest daily doses of fructose and xylitol were 200-400 g. Serum samples were analyzed for sodium, potassium, calcium, magnesium, inorganic phosphates, ascorbate, bilirubin, amylase, alkaline phosphatase, amino-acids, immunoglobulin A, immunoglobulin G and immunoglobulin M. In addition saliva analyses of immunoglobulin A, immunoglobulin G and immunoglobulin M and amylase were carried out. The number of occurrences of diarrhea and flatulence-like conditions were also scored. Body weights of the volunteers were recorded weekly. The numbers of pregnancies in the groups were as follows; eight in the sucrose, six in the fructose and eight in the xylitol group. No significant changes of clinico-chemical parameters in serum and saliva were observed in the xylitol group. A significant rise in the occurrence of diarrhea and flatulence-like conditions was noted in the xylitol group. All pregnancies, deliveries and infants were normal.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/HUMAN EXPOSURE STUDIES/ The tolerance of xylitol was studied in 18 male and one female non-diabetic students aged 21-27 years. The students were given xylitol for 21 days in increasing dose levels from five up to a maximum of 75 g/day. After one month of interruption the same group received xylitol in increasing dose levels from 40 g up to 220 g again during 21 days (19 students received during the first week 40-100 g/day, 18 during the second week 100-150 g, and six during the third week 150-220 g). The subjects themselves recorded quantity, daily division of xylitol intake, number and consistency of bowel movements as well as general condition and obvious side effects. Body weights were estimated weekly. At the third day of each experimental period and seven days after termination, fasting blood sugar analyses and urinalyses on presence of reducing sugar were carried out. From a 130 g/day dose level diarrhea was observed when the single doses were poorly distributed over the day. No other significant effects were noticed. In a similar study 23 men and three women were given xylitol or sorbitol. The initial dose was 5 g which was increased to 75 g/day after 14 days. In addition to the parameters investigated in the first experiment, xylitol and glucose analyses in 24 hour urine were carried out. Identically to the first experiment diarrhea was the only effect observed.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/HUMAN EXPOSURE STUDIES/ In an oral tolerance test with five persons adapted to xylitol up to 120 g/day. The initial dose level was 30 g/day and this was raised by 30 g/day at three day intervals. Liver function tests were normal throughout the experiment, while there was a transient increase in plasma lactate and urate. No diarrhea below 90 g/day was noticed.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/HUMAN EXPOSURE STUDIES/ In a short-term study 5, 15 and 30 g xylitol were administered respectively to healthy men during two and three weeks. Blood chemistry, bilirubin, SGOT, alkaline phosphatase, uric acid and blood sugar were

normal. No diarrhea was observed.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/HUMAN EXPOSURE STUDIES/ After parenteral administration postoperatively of 10% xylitol solution (1.5 g/kg body weight) /investigators/ found a significant increase of lactic acid, uric acid, bilirubin and alkaline phosphatase in two diabetic and two non-diabetic patients.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/HUMAN EXPOSURE STUDIES/ In a short-term experiment carried out with two normal volunteers a dosage of 4.5 g/kg during five days produced significant increased levels of urine uric acid, SGOT, serum alkaline phosphatase, bilirubin, lactic acid, and inorganic phosphate in serum. The levels returned to normal 10 days after cessation of infusion. No effects were found in the BUN, calcium, cholesterol, glucose, amino-acids and insulin analyses, urinalyses and hematology were found.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/HUMAN EXPOSURE STUDIES/ In a short-term experiment with eight normal subjects (21-27 years) receiving orally xylitol over two weeks the fat and uric acid metabolism was studied. After an initial adaptation phase the xylitol level was raised from 5 g up to 50 g during seven days. No significant changes in the serum concentration of triglycerides, free fatty acid, free glycerol, alpha-lipoproteins, total cholesterol, phosphatides, aceto-acetate and beta-hydroxybutyrate were observed during xylitol administration. The serum inorganic phosphate concentration was elevated during the experiment and decreased again afterwards. After an initial rise a significant decrease of the serum pyruvate level and decrease of lactate level from the beginning were observed. The serum uric acid level was not influenced by a xylitol intake.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/SIGNS AND SYMPTOMS/ If excessive amounts of bulk sweeteners (polyols) are consumed, laxative effects may occur.

European Food Safety Authority (EFSA); EFSA Panel on Dietetic Products, Nutrition and Allergies (NDA): Scientific Opinion on the substantiation of health claims related to the sugar replacers xylitol, sorbitol, mannitol, maltitol, lactitol, isomalt, erythritol, D-tagatose, isomaltulose, sucralose and polydextrose and maintenance of tooth mineralisation by decreasing tooth demineralisation and reduction of post-prandial glycaemic responses (April 2011). Available from, as of July 28, 2011: http://www.efsa.europa.eu/en/publications.htm

▸ *from HSDB*

/OTHER TOXICITY INFORMATION/ The food constituents xylitol, sorbitol, mannitol, maltitol, lactitol, isomalt, erythritol, D-tagatose, isomaltulose, sucralose or polydextrose resulted in reduced post-prandial blood glucose (or insulinemic) responses compared with sugars on a weight by weight basis owing to their reduced/delayed digestion/absorption and/or to a decrease in the amount of available carbohydrates, and that the consumption of foods/drinks in which xylitol, sorbitol, mannitol, maltitol, lactitol, isomalt, erythritol, D-tagatose, isomaltulose, sucralose or polydextrose replaced sugars induced lower post-prandial glycemic and insulinemic responses than sugar-containing foods/drinks. ... A cause and effect relationship has been established between the consumption of foods/drinks containing xylitol, sorbitol, mannitol, maltitol, lactitol, isomalt, erythritol, D-tagatose, isomaltulose, sucralose or polydextrose instead of sugar and reduction in post-prandial blood glucose responses (without disproportionally increasing post-prandial insulinemic responses) as compared to sugar-containing foods/drinks.

European Food Safety Authority (EFSA); EFSA Panel on Dietetic Products, Nutrition and Allergies (NDA): Scientific Opinion on the substantiation of health claims related to the sugar replacers xylitol, sorbitol, mannitol, maltitol, lactitol, isomalt, erythritol, D-tagatose, isomaltulose, sucralose and polydextrose and maintenance of tooth mineralisation by decreasing tooth demineralisation and reduction of post-prandial glycaemic responses (April 2011). Available from, as of July 28, 2011: http://www.efsa.europa.eu/en/publications.htm

▸ *from HSDB*

/OTHER TOXICITY INFORMATION/ ... Reports from authoritative bodies and reviews indicates that the decrease in pH in plaque as a consequence of metabolic acid production by saccharolytic bacteria when exposed to fermentable carbohydrates (i.e. sugars and starches) may promote demineralization and prevent remineralization of the hydroxyapatite crystals. Tooth hydroxyapatite crystals are very resistant to dissolution at neutral pH, but their solubility drastically increases as pH drops. Typically, the critical pH for dental enamel is around 5.5. ... Demineralization of tooth tissues can also occur as a result of consumption of dietary acids in foods or beverages, and that frequent consumption can lead to dental erosion. Xylitol, sorbitol, mannitol, maltitol, lactitol, isomalt, erythritol, D-tagatose, isomaltulose, sucralose and polydextrose are slowly metabolized by bacteria in the mouth. The rate and amount of acid production from these food constituents is significantly less than that from sucrose. ... Xylitol, sorbitol, mannitol, maltitol, lactitol, isomalt, erythritol, D-tagatose, isomaltulose, sucralose and polydextrose do not promote dental caries because they do not lower plaque pH to the level associated with enamel demineralization. ... A cause and effect relationship has been established between the consumption of sugar-containing foods/drinks at an exposure frequency of four times daily or more and an increased tooth demineralization, and that the consumption of foods/drinks containing xylitol, sorbitol, mannitol, maltitol, lactitol, isomalt, erythritol, D-tagatose, isomaltulose, sucralose or polydextrose, instead of sugar in sugar-containing foods/drinks, may maintain tooth mineralization by decreasing tooth demineralization compared with sugar-containing foods, provided that such foods/drinks do not lead to dental erosion.

European Food Safety Authority (EFSA); EFSA Panel on Dietetic Products, Nutrition and Allergies (NDA): Scientific Opinion on the substantiation of health claims related to the sugar replacers xylitol, sorbitol, mannitol, maltitol, lactitol, isomalt, erythritol, D-tagatose, isomaltulose, sucralose and polydextrose and maintenance of tooth mineralisation by decreasing tooth demineralisation and reduction of post-prandial glycaemic responses (April 2011). Available from, as of July 28, 2011: http://www.efsa.europa.eu/en/publications.htm

▸ *from HSDB*

/OTHER TOXICITY INFORMATION/ A series of studies in Turku, Finland showed that after one year on test in which xylitol replaced sucrose in the diet dental caries were reduced in the xylitol group by approximately 90%.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

13.1.4 Non-Human Toxicity Excerpts

/LABORATORY ANIMALS: Acute Exposure/ Groups of 35 male Wistar rats (about 200 g each) received by gavage 1.2 mL of a 50%-aqueous solution of maltitol, xylitol, sorbitol, or glucose, and their blood glucose and residual sugar alcohols in the digestive tract were determined hourly for 6 consecutive hrs. Animals given the sugar alcohols exhibited lower blood glucose values than animals receiving glucose itself. Maltitol disappeared from the digestive tract the most quickly of the sugars that were investigated.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 20: 603. Hydrogenated Glucose Syrups (1985). Available from, as of July 20, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/LABORATORY ANIMALS: Acute Exposure/ Glycogen production is stimulated by the administration of xylitol to guinea pigs, rats and perfused rat liver, even when diabetic animals are used. ... In the liver most of the xylitol administered to the animals was rapidly converted to glucose and to small quantities of lactate. /Investigators/ observed a stimulating influence of xylitol on the production of lactic acid in erythrocytes which was also noticed with glucose. In perfused rat liver xylitol stimulated the lactate production and caused an increased lactate to pyruvate ratio. Besides the increased ratio of lactate to pyruvate ... increased ratios of alpha-glycerophosphate to dihydroxy acetone-P and triosephosphate to 3-P-glycerate have been found. Significant decrease of blood pyruvic acid level /has also been noted/ in normal dogs.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/LABORATORY ANIMALS: Acute Exposure/ The acute oral toxicity of xylitol was determined in fasted NMRI mice in unadapted versus fully xylitol adapted mice (five mice/sex/dose group). Toxic signs consisted of staggering gait and a prone position. Slight diarrhea was noted in adapted mice as compared to extensive diarrhea in controls. ... Death

occurred in one to three hours. Necropsy revealed reddening of intestinal mucosa, swollen intestines and gas formation in cecum.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 18: Xylitol (87-99-0) (1983). Available from, as of July 11, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/LABORATORY ANIMALS: Acute Exposure/ To investigate the cause of xylitol induced dose-related diarrhea, cecal bacterial flora were investigated. Using a rapid and sensitive radioisotope bioassay, in which (14)CO2 production from i.v.-(14)C labeled xylitol was measured, it was possible to show that cecal micro-flora obtained from rats can metabolize xylitol. This activity was increased 10-, 15-, 30- and 40-fold in caecal flora taken from rats fed diets containing 2, 5, 5, 10 and 20% xylitol respectively.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 18: Xylitol (87-99-0) (1983). Available from, as of July 11, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/LABORATORY ANIMALS: Acute Exposure/ Sixty Wistar albino rats were gradually adapted to 20% dietary xylitol. Fully adapted and control rats were fasted overnight and dosed by oral intubation with 5 uCi of U-(14)C xylitol, U-(14)C sorbitol or 1-(14)C mannitol. Each isotope dose was mixed with corresponding ("cold") polyol to obtain a final dose of 0.625 g/kg bw. Tail vein blood samples were obtained and counted. In several experiments 0.5 mol of calcium were given together with xylitol. Xylitol adapted rats did not exhibit diarrhea following the dose administration. There was a significant increase in peak xylitol blood levels as determined by radioactivity in xylitol adapted rats as compared to controls. Xylitol adaptation also enhanced sorbitol and mannitol absorption when compared to controls. Calcium caused an initial rise in blood levels of radioactivity but the effect was not statistically significant.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 18: Xylitol (87-99-0) (1983). Available from, as of July 11, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/LABORATORY ANIMALS: Acute Exposure/ The acute toxicity in rabbits was determined by intravenous infusion of 87 mg xylitol/kg/minute. ... Striking increases of SGOT and serum LDH levels, along with an increase urine volume were observed.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/LABORATORY ANIMALS: Acute Exposure/ The Cases of xylitol poisoning in dogs are increasing as a result of ingestion of xylitol-containing products. Eighteen adult, clinically normal Pekingese dogs were orally dosed with 1 or 4 g/kg xylitol in aqueous solution. Blood samples were collected before and after dosing. Plasma insulin concentrations of both treated groups rose sharply from 20 min after xylitol dosing, peaking at 40 min. Hypoglycemia followed the increase in insulin concentration, with blood glucose values started to decrease 30 min after dosing. Other plasma biochemistry changes associated with xylitol administration were increased alanine aminotransferase and aspartate aminotransferase activities, hypophosphatemia, hypokalemia, and hypercalcemia. Plasma sodium and chloride concentrations remained normal. This study established a biochemical basis for diagnosis and treatment of xylitol poisoning in dogs.

Xia Z et al; J Vet Pharmacol Ther 32 (5): 465-469 (2009)

▸ *from HSDB*

/LABORATORY ANIMALS: Acute Exposure/ Xylitol toxicity was evaluated by intravenous infusion of varying doses of xylitol at different concentrations into rabbits. Acute toxicity was observed with the administration of 50% xylitol solutions. Striking increases of SGOT and serum LDH levels, along with an increase of urine volume were observed. The LD50 of a 50% solution infused at a rate of 87 mg/kg/min was estimated to be between 4-6 g xylitol/kg body wt. The toxicity correlated well with the concentration of the xylitol solution used or the infusion rate. Both determined serum xylitol concentration and serum osmolality. At a constant infusion rate, the total dose became the important factor in toxicity. The effects of xylitol were compared with those seen after the administration of isosmolar urea, mannitol, or glucose. The acute toxicity of xylitol appears to be mainly due to a hyperosmolar effect. A 5% xylitol solution, essentially isotonic, is nontoxic to the animal in a dose up to 1 g/kg body wt/12 hr even for multiple infusions.

Wang Y-M et al; Metabolism 22 (7): 885-894 (1973)

from HSDB

/LABORATORY ANIMALS: Subchronic or Prechronic Exposure/ Groups of Wistar rats (70 rats/group) were allocated to diets of either 20% xylitol (two groups, one group was gradually adapted beginning with a 5% diet to prevent diarrhea) or control diet (one group). Periodically rats were killed at intervals up to 150 days and subjected to gross necropsy. Sections of liver, kidney, adrenals, stomach, cecum and bladder were prepared for histopathological examination. Initially non-adapted rats showed a decrease in body weight, and decreased food consumption. Rats receiving xylitol without prior adaptation exhibited cecal enlargement. This was present to only a slight extent in adapted rats. There were small changes in the relative organ weights. The bladders of many rats showed one or more white precipitates. Histopathologically no changes were seen in liver, kidney, spleen, adrenals or stomach. Histopathologically many rats showed focal hyperplasia of the bladder wall associated with the precipitates (control 2/55, xylitol adapted 2/55, xylitol unadapted 9/55). In the non-adapted rats showing diarrhea inflammatory changes were observed on the bladder at the time of diarrhea.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 18: Xylitol (87-99-0) (1983). Available from, as of July 11, 2011: http://www.inchem.org/pages/jecfa.html

from HSDB

/LABORATORY ANIMALS: Subchronic or Prechronic Exposure/ Six male castrated pigs (39-75 kg weight range) were subjected to feeding trials based on a Latin square design with either a polyol mixture (a by-product of xylitol production containing xylane) or xylitol supplementations. The transition period between diets was five days, preliminary period seven days, and collection period seven days (seven days feeding per diet). The basic diet consisted of skim milk powder with minerals and vitamins, to which polyol mixture (levels of 5 or 2.5% dry matter) or xylitol (2.5 or 5% dry matter) were added. Wheat starch (5.0 or 2.5%) served as control supplement. Feces and urine were collected twice a day and frozen until analyzed. Venous blood samples were obtained one, two, or four hours after feeding. Glucose, plasma insulin and various clinical chemical parameters were determined. There was a slight decrease in the nitrogen balance of diets supplemented with 10% of polyol mixture or 5% of xylitol. There was no detectable xylitol or sugar alcohol in the feces; a small quantity of xylitol was found in the urine of pigs when fed polyol mixture but not when fed xylitol. There was a significant rise in plasma glucose levels in xylitol fed pigs. Urine nitrogen decreased slightly in polyol or xylitol fed animals. Albumin concentration was significantly raised. There were increases in plasma alanine and asparate transferases (transaminases) (ALAT and ASAT also called serum glutamic pyruvic and glutamic-oxaloacetic transaminases (SGPT and SGOT)). The SGPT and SGOT levels increased significantly in a dose-related manner and indicated possible liver toxicity. Only the high dose level was statistically significantly different from the control. There were increases in insulin concentrations following xylitol feeding, and values two hours after feeding were higher than control levels. This increase was also dose-related. The peak of insulin levels was between 40-60 minutes after feeding.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 18: Xylitol (87-99-0) (1983). Available from, as of July 11, 2011: http://www.inchem.org/pages/jecfa.html

from HSDB

/LABORATORY ANIMALS: Subchronic or Prechronic Exposure/ In a 13 weeks toxicity study two male and two female rhesus monkeys, weighing 2-3 kg, were dosed twice daily six days a week 0, 1.0, 3.0 and 5.0 g xylitol/kg/day by gastric intubation. The animals were observed daily for mortality, appearance, behavior, appetite, elimination and pharmacotoxic effects. Body weights were recorded weekly. At 0, 4, 8 and 13 weeks ophthalmoscopic and neurologic examinations, hematology, clinical chemistry and urinalyses were performed. Hematology included hematocrit, Hb, total and differential leucocyte counts, prothrombin time and coagulation time. Blood chemistry consisted of blood sugar, blood urea nitrogen, serum alkaline phosphatase, SGPT and SGOT estimations. In addition, free and total bilirubin and uric acid determinations were conducted at four, eight and 13 weeks. Urinalyses included specific gravity, pH, glucose, ketones, total protein, bilirubin and microscopic examination of sediment. Gross-necropsy was carried out on all animals. Spleen, brain, pituitary, thyroid, heart, lung, liver, kidneys, adrenals, testes, prostate, ovaries, uterus were weighed. Histopathology of these organs and thoracic spinal cord, eye, gall bladder, thymus, salivary gland, stomach, pancreas, small intestine, large intestine, mesenteric lymph nodes, sciatic nerve, skeletal muscle, urinary bladder, skin, rib, vertebra, femur, sternum were carried out at 13 weeks. Soft stool and/or diarrhea were noted in all treatment groups intermittently throughout the study and appeared to be dose related. In the treatment groups a tendency to decreased coagulation time (from 3.16 to 2.40 minutes) was noticed. Brain weight tended to a dose related decrease in the male animals receiving xylitol. No further dose related effects were observed in this study.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/LABORATORY ANIMALS: Subchronic or Prechronic Exposure/ A six weeks toxicity experiment with one female and one male dog (5.5-6 kg) per group was carried out. The concentration during the experiment was increased from 5% to 20%, after two weeks. Food consumption was recorded twice daily and body weights twice a week. At the end of the experiment gross pathology was carried out, and brain, heart, liver, lungs, pituitary, spleen, pancreas, thymus, prostate/uterus, kidneys, thyroids, adrenals and testes/ovaries were weighed. No histopathological investigation was carried out. Except for a tendency to increased liver weights in the xylitol groups no effects are observed. ...

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/LABORATORY ANIMALS: Subchronic or Prechronic Exposure/ In short-term study five dogs were dosed 10 g xylitol/kg/day by iv infusion during 7 weeks. Blood chemistry and urinalyses were investigated weekly. Plasma glucose content was reduced to 64 mg % after six weeks. A slight increase in plasma lactate and a significant increase of serum glutamic pyruvic transaminase and alkaline phosphatase was observed. Also an increased urinary loss was observed. No effect of xylitol on plasma insulin levels and no oxalate crystals in the kidneys were observed.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/LABORATORY ANIMALS: Subchronic or Prechronic Exposure/ In a short-term (13 weeks) study four groups of eight female and eight male Charles River CD rats weighing 120-150 g, were fed 0, 5, 10 and 20 g dietary xylitol/kg/day. Food and water consumption were recorded daily and body weight weekly. Hematology, blood glucose and urinalyses of five male and five female rats of each group were carried out at four, eight and 12 weeks. alkaline phosphatase, glutamic oxalic transaminase, bilirubin and uric acid in serum and blood urea nitrogen of five males and five females per group were determined at 13 weeks. The animals, particularly the male rats, showed reduced body weight gain and food consumption that were dose dependent. In the 20% xylitol group blood urea nitrogen was elevated. In treatment groups dose-related decrease of the absolute heart weight were observed. Except for transient diarrhea in a number of treated animals, no other significant changes were observed. No histopathological lesions related to xylitol administered were noticed.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/LABORATORY ANIMALS: Subchronic or Prechronic Exposure/ In a 15 days' experiment with rats, sucrose was replaced respectively by 10 and 20% xylitol. The protein of the diet consisted of casein, while their carbohydrate allowance consisted of starch and sugar. In the 10% xylitol dose group no histopathological changes were observed. However, in the 20% xylitol dose group adverse effects of the metabolism of the liver cells were noticed. Reductions of the glycogen and lipids concentrations were observed. The mitotic activity of the liver cells was sharply reduced to a level five times lower than in the control rats.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/LABORATORY ANIMALS: Subchronic or Prechronic Exposure/ In a subacute toxicity study xylitol was administered to 20 female and 20 male rats per group by daily gastric intubation for a period of 14 days. Dose levels were 1.25, 2.5, 5.0 and 10.0 g/kg. The animals receiving 1.25 g xylitol/kg during the first nine days, received 10.0 g xylitol/kg during the last seven days. In the control groups animals received 0, 2.5 and 5.0 g sucrose/kg/day. During treatment (two, five and 14 days) animals were submitted to careful clinical examinations and blood serum analyses related to hepatic functions. These analyses were glucose in blood and bilirubin, free fatty acid, total lipids, triglycerides, cholesterol, glucose-6-phosphate dehydrogenase and alkaline phosphatase, glutamic/glutamate pyruvic transaminase and glutamic oxalic transaminase in

serum. Also body weight and food consumption were recorded. The animals were sacrificed after two, five and 14 days. Heart, liver, spleen, kidneys, adrenals, gonads, stomach were weighed. These organs and jejunum, colon, pancreas and brain were histologically examined in two and five days treatment groups, while in 14 days treatment group this examination was only performed on liver. The histological study was conducted on five males and five females per group. Except for a decrease of the free fatty acid content in the blood at all xylitol dose levels, no effects are observed in this study. No evidence of hepatotoxicity was recorded.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/LABORATORY ANIMALS: Subchronic or Prechronic Exposure/ .../it has been reported/ that 6 days xylitol administration to rats results in hepatic dysfunction. In .../this/ study xylitol was administered to rats by daily gastric intubation for a period of 14 days. Dose levels were 1.25, 2.5, 5 and 10 g/kg. During treatment (2, 5 and 14 days), animals were submitted to careful clinical examinations and to blood serum analysis related to hepatic functions. They were sacrificed after 2, 5 and 14 days treatment. In the 2 and 5 days treatment groups, main organs were submitted to histological study. This examination was only performed on liver in the 14 days treatment group. No evidence of hepatotoxicity was recorded. Serum levels of all parameters measured were within normal limits (including bilirubin and serum alkaline phosphatase (SAP). No anomalies were shown histologically.
Abstract: PubMed

Truhaut R; Toxicology 8 (1): 79-85 (1977)

▸ *from HSDB*

/LABORATORY ANIMALS: Subchronic or Prechronic Exposure/... Male Fisher-344 rats were fed semipurified diets containing glucose without or with either 10% or 20% sorbitol or xylitol for 8 weeks following a 4-week adaptation period. The addition of sugar alcohols decreased food efficiency and total body weight gain regardless of the type fed. The addition of sugar alcohols to the diets decreased liver glucose-6-phosphate dehydrogenase (G6PD) and malic enzyme (ME). However, rats fed either 10% xylitol or sorbitol or 20% sorbitol had higher levels of epididymal fat tissue G6PD and ME. Plasma glucagon levels were decreased with the addition of sugar alcohols; whereas, in plasma insulin and glucose there was little effect. Diets containing sugar alcohols decreased levels of plasma triglyceride and cholesterol. The addition of sugar alcohols to the diets increased plasma alkaline phosphatase levels. Dietary treatments had no effect on urine or adrenal norephinephrine and epinephrine, and urine dihydroxyphenylacetic acid and dopamine.

Ellwood KC et al; Nutrition Research 19 (11): 1637-1648 (1999)

▸ *from HSDB*

/LABORATORY ANIMALS: Subchronic or Prechronic Exposure/ Xylitol was administered to rats by daily gastric intubation for a period of 14 days. Dose levels were 1.25, 2.5, 5 and 10 g/kg. During treatment (2, 5 and 14 days), animals were submitted to careful clinical examinations and to blood serum analysis related to hepatic functions. They were sacrificed after 2, 5, and 14 days treatment. In the 2 and 5 days treatment groups, main organs were submitted to histological study. This examination was only performed on liver in the 14 days treatment group. No evidence of hepatotoxicity was recorded. Serum levels of all parameters measured were within normal limits (including bilirubin and serum alkaline phosphatase (SAP). No anomalies were shown histologically.
Abstract: PubMed

Truhaut et al; Toxicology 8 (1): 79-85 (1977)

▸ *from HSDB*

/LABORATORY ANIMALS: Subchronic or Prechronic Exposure/ The acidification of urine during polyol feeding was investigated with 27 Long-Evans male rats (aged 12 weeks) which were fed a xylitol diet (X), a sorbitol diet (S), or a basal diet for 4 weeks. The amount of polyols in the diet was increased from 5% to the final 20% level within 3 weeks. The polyol-fed animals showed reduced weight gain, lowered urine pH (from 6.5 to 5.6), and a 4-fold increase in the titratable acid excretion. X and S increased the daily urine volumes by 49 and 63%, respectively, but did not affect the wet weight or the pH values of the feces. Gas chromatographic-mass spectrometric analyses of organic acids revealed highly increased amounts of methylmalonic acid (13- to 20-fold) and 2-oxoglutaric acid (4- to 5-fold) in the urine of polyol-fed rats. The urinary excretion of citric acid and malic acid was also increased significantly (2- to 4-fold). The acidity of urine was not reflected in the blood acid-base balance of the animals. The increases in the levels of urinary organic acids in the polyol-

fed rats were explained in terms of impaired mitochondrial oxidation of these acids and of impaired conversion of methylmalonic acid to succinic acid.

Hamalainen MM; Toxicology and Applied Pharmacology 90 (2): 217-226 (1987)

▸ *from HSDB*

/LABORATORY ANIMALS: Chronic Exposure or Carcinogenicity/ Xylitol was administered in the diet of pure-bred beagle dogs (eight male and eight female animals per group). Dietary concentration of 0, 2, 5, 10 and 20% xylitol were attained by a gradual increase of xylitol at the expense of starch. The 20% dietary level was reached by the fifth week. A further group received 20% sucrose for comparative purposes. Rice starch was included in the diets of groups receiving 0, 2, 5, and 10% xylitol, so that in each case the diet consisted of 80% normal diet and 20% carbohydrate. After week 42 diets were supplemented with 5 mL corn oil (during weeks 42-48 once/day, week 48 onward once/week). After 52 weeks there was an interim sacrifice of two males and two females per group. The study was terminated at 104 weeks. The 20% xylitol and 20% sucrose groups left significantly more food than the control group. The 2% xylitol group gained less weight than controls. All other groups gained more weight than controls. Hematological parameters and analysis were within normal limits during the course of the study. Clinical chemistry studies showed that from week 12 onwards slightly elevated total serum protein levels were recorded in 20% xylitol group. Particularly in the first year, but also in the second year, the 20% xylitol group had elevated SAP values. At almost every interval, SGPT for 20% xylitol group was elevated. In the second year this was also true for the 10% xylitol group. Total lactate dehydrogenase and alpha- hemoglobin dehydrogenase showed considerable variation. During weeks 89 and 100 the values for the 20% xylitol group were elevated. At autopsy there was a significant increase in liver weight in the 20% xylitol group. Changes in periportal hepatocytes were detected histologically in at least five of 12 dogs in this group. Electron micrographic analysis suggested that the changes in hepatocytes observed were consistent with alterations in glycogen storage. No evidence of degeneration or necrosis was noted. Similar histological changes were found in the hepatocytes of three and 12 dogs in the 10% sucrose group. No other compound-related changes were reported.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 13: Xylitol (87-99-0) (1978). Available from, as of July 8, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/LABORATORY ANIMALS: Chronic Exposure or Carcinogenicity/ A total of 75 male and 75 female Sprague-Dawley rats of the CD strain were included in each dosage group. Each group consisted of 50 male and 50 female rats for tumorigenic evaluation (104 weeks), 15 male and 15 female rats for laboratory investigation, and 10 male and 10 female for interim sacrifice of five male and female rats at 26 and 52 weeks. Xylitol was fed at 0, 2, 5, 10, and 20% of the diet. In addition, one group received 20% sucrose in the diet. All animals were derived from parents exposed to the respective test diets for 60 days prior to mating. Following parturition, the diet of all treated dams was reduced to 5% and gradually increased to the desired levels of 5, 10, 20% xylitol and 20% sucrose. The diet of all groups was maintained at 20% carbohydrate supplementation through the use of rice starch in the 0, 2, 5, and 10% groups. Protein was maintained constant in all groups through the addition of casein. For the 10 and 20% carbohydrate groups (xylitol and sucrose) the carbohydrate was increased 5% per week until the desired level was attained. Lower body weight gain was recorded through the first 78 weeks of the study for males and females receiving 5, 10, or 20% xylitol. This decreased weight gain was coupled with a dose-related impairment of food utilization efficiency for males and females in the 5, 10, and 20% xylitol groups. The 20% xylitol females had statistically higher water intake during weeks 26, 52, and 78. Urinalysis indicated increased urine volumes for the female 20% xylitol group. Twenty percent sucrose caused increased urinary protein levels during the first 26 weeks of treatment in some males and females. Hematologic examination indicated no xylitol-related effects through 78 weeks. At terminal sacrifice, the 20% xylitol males did, however, show lower PCV values and RBC counts. For up to 52 weeks, lower alpha-2-globulin was found in all xylitol-treated males. Lowered SGPT was recorded for 20% xylitol females at 13 weeks. Lower lactate levels were recorded for xylitol animals at 26 weeks but not at 52 weeks. Hematological and clinical chemistry values of serum were within normal limits with the exception that sucrose treatment increased cholesterol in males at 52 weeks and males and females at 78 weeks. No increased cholesterol was noted at terminal sacrifice for the sucrose group. Sucrose also increased insulin levels at 26 and 52 weeks but returned to normal at 78 weeks. Male sucrose animals had elevated insulin at terminal sacrifice. At autopsy macroscopic examination indicated that 20% xylitol treatment caused enlargement of the caecum. No other treatment-related gross pathological or ophthalmoscopic changes were noted. Organ weight analysis indicated higher liver weight ratios at 26 weeks for males receiving 5, 10, and 20% xylitol and females receiving 5 and 10% xylitol; such effects were not seen at 52 weeks or terminal sacrifice. At terminal sacrifice, lower absolute thyroid weights were recorded for males and females in all treated groups but was most marked in the 20% xylitol and sucrose groups. The relative thyroid weights were decreased only for the 20% xylitol males and 20% sucrose females. No other treatment-related effects were seen on organ weights.

Histological examination of the animals indicated no treatment-related effects on the major organ systems. However, the incidence of both unilateral and bilateral hyperplasia of the adrenal medulla was significantly increased in males treated with 10 and 20% xylitol and among females treated with 5, 10 and 20% xylitol. The adrenal medullary hyperplasia also was strongly dose related and was confined to only one gland in the control groups. Additionally, a significant increase in pheochromocytoma was found in the 20% xylitol males. For statistical comparison of pheochromocytoma the 2% xylitol and control groups were combined.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 13: Xylitol (87-99-0) (1978). Available from, as of July 8, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/LABORATORY ANIMALS: Chronic Exposure or Carcinogenicity/ Groups each of 100 male and 100 female CFLP mice per group were fed diets containing levels of 0, 2, 10, or 20% xylitol or 20% sucrose. The diet of all groups was maintained at 20% carbohydrate supplementation through the use of rice starch. Protein was maintained constant in all groups through the addition of casein. A group was terminated when 20% survival was reached. Initially the highest level of carbohydrate in any of the diets was 10%. Subsequently, desired levels of carbohydrate were obtained by increasing the amount in the diets by 5% each week. No diarrhea was observed with up to 10% xylitol or sucrose during the first three weeks. During the fourth week, mice receiving 15% xylitol (male and female) exhibited diarrhea. The males also had associated inflammation of the anogenital region. The 15% sucrose group also showed some evidence of diarrhea. These mice were returned to 10% xylitol and sucrose until week seven when they again received 15% xylitol and sucrose. The same signs as above were again seen. By week 11 the mice seemed normal and the 20% xylitol and sucrose levels were achieved by week 14. It therefore took as long as 16 weeks to achieve dietary accommodation to 20% xylitol and sucrose. An overall significant increase in mortality was seen for 20% xylitol males during the first year. Some increases in food intake was seen for males in the 10 and 20% xylitol groups between weeks one and 106 and for females in these groups between weeks one and 80. The 20% sucrose group showed increased food intake for the first year of the study. Males in the 10 and 20% xylitol groups as well as 20% xylitol females had decreased body weight gains. Water intake was increased for the 20% xylitol males. Macroscopic examination indicated a large increase in the incidence of urinary bladder calculi, urinary bladder nodules and masses, and urinary bladder distention for the 10 and 20% xylitol males. No similar effects were seen in the 2, 10, and 20% females, 2% xylitol males, or the 20% sucrose group. Histological examination of the 10 and 20% xylitol males revealed hyperplasia, metaplasia, and neoplasia of the transitional epithelium of the urinary bladder in male mice, associated with the macroscopically observed calculi. No metastasis was observed in the bladder tumors. The incidence of treatment-related tumors, metaplasias or neoplasias was not increased for the 2% xylitol males nor the 2, 10, and 20% xylitol females. Analysis of the bladder calculi indicates they are composed of calcium, phosphate, and oxalate. In the 20% sucrose-fed males there was an increase in kidney lesions described as cellular infiltration. Macroscopic examination of mice dying during the study revealed a statistically significant, dose-related reduction in the number of male mice bearing liver masses treated with xylitol, as compared to controls. Among mice killed at termination a lower prevalence of liver masses was recorded for males treated with 10 or 20% xylitol and attained a level of statistical significance ($P < 0.05$) when compared to controls. Histologically, the liver masses were mainly benign adenomas, a small proportion had a structure suggestive of carcinoma. The prevalence in the sucrose group was similar to controls. The differences were statistically significant when 10 and 20% xylitol males were compared to controls and when 2, 10 or 20% levels of xylitol compared to 20% sucrose group. The incidence of hepatocellular tumors (benign) was increased for the 20% sucrose females. Male 20% sucrose mice also showed an increase in fatty degeneration of hepatocytes.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 13: Xylitol (87-99-0) (1978). Available from, as of July 8, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/LABORATORY ANIMALS: Chronic Exposure or Carcinogenicity/ In a long-term toxicity study xylitol has been administered orally 100 mg/xylitol/kg to 20 female and 20 male Wistar rats for 11 months and to 15 female and 15 male rats for 24 months. Body weights were recorded weekly. At termination detailed gross pathology was performed and a microscopic study was carried out on stomach, intestines, liver, spleen, pancreas, kidneys, adrenals, testes/ovaries, uterus/prostate, lungs, heart, brains, salivary glands, lymph nodes, thyroid, thymus, sphenoid, pituitary, ganglions of Glasser and femur. The experiment was carried out with two generations and the fertility was also checked. The results were compared with 450 control animals kept under similar experimental conditions. No malignant tumors were observed in the animals treated with 100 mg xylitol/kg and no stimulation of the growth of tumors was noticed. According to the author there was no harmful influence on reproduction and xylitol did not produce any pathological changes in the animals.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

from HSDB

/LABORATORY ANIMALS: Chronic Exposure or Carcinogenicity/ In a long-term toxicity study eight female and eight male beagle dogs per group, weighing 6-9 kg, were fed 0, 2, 5, 10 and 20% dietary xylitol during two years. The total amounts of carbohydrates were kept constant. In addition animals receiving 20% sorbitol and 20% sucrose were used as sugar controls. Body weight and food consumption was recorded weekly, while water consumption was recorded over five day periods at the weeks one to four, 9-12, 21-24, 35-38 and 46-49. Ophthalmoscopy, dental examination and a full neurological examination of four male and four female dogs were carried out at 13, 26, 39, 50, 64 and 76 weeks. Hematology, biochemistry and urinalyses were carried out at the beginning of the experiment and at 12, 26, 38, 50, 64 and 76 weeks. The hematology included erythrocyte sedimentation rate, packed cell volume, Hb, RBC, reticulocyte count, mean corpuscular hemoglobin concentration, mean corpuscular volume, WBC, differential count, platelet count, prothrombin index, whole blood clotting time. The biochemistry included determination of total protein, aluminum, glutamic/glutamate pyruvic transaminase, glutamic oxalic transaminase, total lactate dehydrogenase, alpha- hemoglobin dehydrogenase, bilirubin, uric acid, cholesterol, lactate and insulin in serum and urea xylitol, glucose concentration and total reducing substances in plasma. Sodium, potassium, chloride and bicarbonate were also detected in the blood. Urinalyses consisted of estimations of Ph, protein, reducing substances, glucose, ketones, bile pigments, urobiligen and Hb. On completion of 52 weeks, two male and two female dogs per group were sacrificed for interim study, while the remaining dogs will be killed after two years' dosing. The weights of brain, liver, kidneys, pituitary, thyroid, spleen, heart, lungs, adrenals, ovaries, testes, uterus, thymus, prostate, and pancreas were recorded. Histopathology of these organs and of aorta, trachea, lymph nodes, gall bladder, urinary bladder, salivary glands, esophagus, duodenum, stomach, jejunum, ileum, skin, skeletal muscle, mammary glands, tongue, eye with optic nerve and sciatic nerve was performed. The food intake and weight gain of the 20% xylitol group were increased. A dose-related increase of serum alkaline phosphatase and serum glutamic pyruvic transaminase and a decreased lactate level was noticed, while in the 20% xylitol dose group also the total serum protein was significantly increased. At 52 weeks a tendency to increased cholesterol content in serum was observed. Except a dose related decrease in the pituitary of the xylitol dose groups, no effects on organ weights in gross necropsy or histopathology were observed. ...

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

from HSDB

/LABORATORY ANIMALS: Chronic Exposure or Carcinogenicity/ Rats fed a cariogenic diet containing 10% xylitol showed no carious lesions in the first and third molars and only minimal involvement of the second molars was observed.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

from HSDB

/LABORATORY ANIMALS: Chronic Exposure or Carcinogenicity/ In a long-term toxicity and carcinogenicity study five groups of 75 female and 75 male Sprague-Dawley rats, weighing 120-150 g, were fed 0, 2, 5, 10 and 20% dietary xylitol. The total amounts of carbohydrates in the diet were kept constant. Animals receiving 20% sorbitol and 20% sucrose were used as sugar control groups. Food intake and body weight were recorded weekly. Water consumption was recorded during weeks 12, 25, 52 and 78. Ophthalmoscopy, urinalysis, hematology and biochemistry were performed at 0, 13, 26, 52, 78 and 104 weeks. Urinalyses included pH, specific gravity, protein, reducing substances, glucose, ketones, bile pigments, urobiligen, hemoglobin, and oxalic acid determinations. Hematology consisted of determination of packed cell volume, Hb, RBC, WBC, mean corpuscular hemoglobin concentration, mean corpuscular volume, differential count, prothrombin index, and whole blood clotting time. The biochemical estimations were urea and glucose in plasma, total proteins, protein electrophoresis, alkaline phosphatase, glutamic/glutamate pyruvic transaminase, glutamic oxalic transaminase, bilirubin, uric acid, cholesterol, lactate, lactate dehydrogenase, alpha-hemoglobin dehydrogenase in serum and total reducing substances, insulin, xylitol, Na+, K+, Cl- and bicarbonate in blood. After 26 and 52 weeks, five males and five females per group were killed. In the gross pathology the animals were examined visually and by palpation. Brain, heart, liver, kidneys, adrenals, pituitary, thyroid, spleen, testes/ovaries were weighed. These organs and pancreas, thymus, uterus, cervical and mesenteric lymph nodes, stomach, bone marrow, ileum, coecum, duodenum, urinary bladder, eye, lungs and any macroscopically abnormal tissues were histopathologically investigated. Blood smears of these organs were also studied. Significant decreased weight gain in animals receiving 10% or 20% xylitol during the experiment was observed, while there was only tendency to decreased food intake in animals receiving 20% xylitol at 78 weeks. In rats receiving 20% xylitol, higher water intakes were recorded during weeks 26 and 52, and in female rats also during week 78. In male and female rats at 13, 26 and 52 weeks and in female rats at week 78, receiving 20% xylitol greater volumes of

more diluted urine were excreted. After 26 weeks, the urine from animals receiving 5, 10 and 20% showed decreased specific gravity. No increase in mortality was noticed in the treated groups, except a significant decreased prothrombin index in all male rats receiving xylitol and female rats receiving 20% xylitol, no hematological changes were observed at 78 weeks. In biochemistry a tendency to decreased lactate contents in the blood of animals receiving 10 and 20% xylitol and at 78 weeks a tendency to increased uric acid content in male rats of the 20% xylitol group were observed. No morphological abnormalities in the treated groups differing significantly from the control group ware observed. ...

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/LABORATORY ANIMALS: Chronic Exposure or Carcinogenicity/ Chronic consumption by rats of diets rich in sugars or sugar alcohols leads to an increased incidence of pheochromocytomas. This relationship is hypothesized to be based on altered Ca2+ homeostasis due to increased intestinal Ca2+ absorption. Other agents associated with pheochromocytomas in rats in long-term toxicity studies have been shown to increase chromaffin cell proliferation, leading to the suggestion that the tumors occur secondarily to increased chromaffin cell turnover. We have demonstrated marked stimulation of chromaffin cell proliferation by vitamin D3, a potent stimulus to Ca2+ absorption not previously associated with adrenal medullary toxicity. This effect is detectable during the first week of dietary supplementation and persists throughout a 4-week time course. Lactose and xylitol, representative of sugars and sugar alcohols associated with pheochromocytomas, are also mitogenic but to a lesser extent, with their effects first detectable during Week 4 of dietary supplementation. Vitamin D3, its active metabolite calcitriol, lactose, and xylitol all fail to stimulate proliferation of rat chromaffin cells in vitro. The mitogenic effects of these agents may be mediated presynaptically in vivo. The data suggest that altered Ca2+ homeostasis may increase chromaffin cell proliferation and support the hypothesis that diets containing high concentrations of sugars and sugar alcohols cause pheochromocytomas in rats secondarily by this mechanism. Abstract: PubMed

Tischler AS et al; Toxicol Appl Pharmacol 140 (1): 115-23 (1996)

▸ *from HSDB*

/LABORATORY ANIMALS: Developmental or Reproductive Toxicity/ A three-generation reproduction study was conducted in NMRI mice. Initially groups of 12 females and three males were allocated to a control group and a group adapted to 20% xylitol. Diets and water were constantly available. Body weights were recorded weekly. No abnormalities of condition or behavior were observed in any of the successive generations of the control or xylitol treated groups. However, the body weights of xylitol treated animals at birth were decreased as compared to controls and significantly lower weight gains were observed in the F0 litters of xylitol fed animals. Even though the growth rates were lower in xylitol dosed litters, no significant differences were noted in food consumption after weaning. There were no significant differences in mean numbers of pups per litter. The mean birth weights were similar in both groups and minor variations were observed only in relation to litter size. No treatment related differences in mortality figures were observed during lactation periods. Gross examination revealed no abnormalities or differences attributable to xylitol treatment. Only slight increases in the cecum size were observed in xylitol treated mice.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 18: Xylitol (87-99-0) (1983). Available from, as of July 11, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/LABORATORY ANIMALS: Developmental or Reproductive Toxicity/ Groups of 20 yellow-silver does of a closed, randomized outbred rabbit strain, aged three to four months and weighing 2.7-3.0 kg received test diets ad libitum containing 2, 5, 10 and 20% xylitol or 20% sucrose, for comparison, baked into the food pellets. Males were untreated. The test diets were administered from days 7-19 of gestation. Parameters investigated were: /maternal/ body weights, litter data including implantations and pre-implantation loss, litter size and post-implantation loss, litter and mean fetal weights, major and minor anomalies and skeletal variants. Young were incubated for 24 hours to determine neonatal viability. Gross and X-ray examinations were conducted to discover external and skeletal malformations. Alizarin red technique was utilized where necessary. The Wilson technique was used to examine for malformations of brain and skull. No compound-related effects were noted. The incidence of skeletal malformations was similar in offspring of the treatment and sucrose control groups. No major visceral abnormalities were noted.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 13: Xylitol (87-99-0) (1978). Available from, as of July 8, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/LABORATORY ANIMALS: Developmental or Reproductive Toxicity/ Groups of 31-33 CFT strain-specific pathogen-free female weanling rats received test diets containing either 20% rice starch, 2, 5, 10, 20% xylitol, or 20% sucrose. The test diets were administered for five weeks before mating. Groups receiving dietary concentrations greater than 5% were acclimated to the final dosage by beginning at the 5% level and weekly increments to the final concentration. Males received laboratory diets only. After females were acclimated to the final dietary concentrations they ware mated. Parameters investigated included food consumption, body weight change, mating performance, litter data including implantations, pre-implantation loss, litter and mean fetal weights, major malformations and minor abnormalities, and skeletal variants. There was a low overall pregnancy rate of approximately 50% for all groups. No clinical signs of toxicity were noted. Five major malformations were noted in test groups receiving xylitol, and two in the group receiving sucrose. These had been previously seen among control pups and not attributed to treatment since there was no relationship to dose level. No skeletal variations were attributed to treatments. Other parameters were within normal limits.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 13: Xylitol (87-99-0) (1978). Available from, as of July 8, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/LABORATORY ANIMALS: Developmental or Reproductive Toxicity/ A three-generation study was conducted in Sprague-Dawley (CD) specific-pathogen-free rats with 20 males and 20 females respectively per group. Each group received the test material by dietary administration. A control group received 20% rice starch, and a comparison group 20% sucrose ad libitum. Graded diets of xylitol, 2, 5, 10 or 20% were fed to treatment groups with rice starch quantum satis to bring the level to 20%. For xylitol groups receiving diets greater than 5%, administration began at the 5% level with 5% weekly increments until final doses of 5, 10, 15 or 20% were reached. During parturition, the diets were reduced to 5% xylitol. As soon as possible after parturition of second litters of each generation, dams receiving diets greater than 5% were returned to this level. Males and non- pregnant females continued to receive the full dose. After weaning, the dose administered to F1b and F2b generations were progressively increased until full treatment level was attained. The gradual increase to concentrations greater than 5% and the reduction of dietary doses during lactation was considered necessary to avoid diarrhea and stress associated with exposure to high concentrations of xylitol. The pups of the F1a, F2a, and F3a generations were weighed and killed at four days and examined for sex determination or abnormalities. The F1b, F2b, and F3b pups were weighed, sexed, and litters culled to eight per dam. Pups were weighed at 8, 12, and 21 days post partum. Observations were made to determine litter size, litter and pup weights, pup mortality, and gross abnormalities. Gross and histopathological observations were conducted on rats of the F3b generation, which were killed at three weeks of age. Mortalities of parents were randomly distributed. No obvious clinical signs were noted. At low diet levels (2 and 5%) food intake was comparable with controls in all generations. At 10% xylitol food intake was slightly but consistently lower in the second generation. At the 20% level slight depression of food intake of the F0, and marked suppresion in F1 and F2 generations was seen, even when xylitol concentration was reduced to 5%. With this group consistent but generally marked suppression of food intake was noted in all generations even at the lowest level fed. With the sucrose group food intake was comparable with or slightly greater than that of the control group in all generations. At the lower levels, weight gain of males of the first generation (2, 5, or 10%) was comparable to the control group, slight suppression occurred at the 10% level during the second generation, and at all concentrations in the final generation (F3b). There was no treatment effect on mating performance and pregnancy rate. At the highest level (20%) a larger proportion of litters was born on day 23 or 24 compared to controls, or sucrose. Cecal enlargement was noted at terminal necropsy of F2b parents of both sexes for levels greater than 2%. At the 20% dietary level of xylitol there were lower values for total and viable litter size at birth and at day four post partum. This became more accentuated with successive generations. Litter and mean pup weights were greater than concurrent control values except for 20% xylitol at which level lower litter weights reflected smaller litter size. Initially among the F1a pups, when litter size was not culled, a large mortality was noted between seven to 10 days post partum. The litter size was culled to eight with the succeeding generations, at which time no total litter loss was observed. There was no indication of a treatment effect on occurrence of terata. Statistical assessment of organ weight data showed in most instances changes related to differences in body weight. Statistically significant lower absolute thyroid weights were noted with 20% xylitol. Other organ weight changes appeared sporadically, unrelated to treatment. Histopathologically tissues from 10 and 20% xylitol groups did not show microscopic changes that could be attributed to xylitol.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 13: Xylitol (87-99-0) (1978). Available from, as of July 8, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/LABORATORY ANIMALS: Developmental or Reproductive Toxicity/ Rats fed /concentrations/ of xylitol up to 20% of the diet to up to four months had no significant adverse effects. Growth rate was normal. ... Xylitol had no effect on the number or size of pups or dams fed xylitol, and the pups adapted readily to the supplement at 5 and 10% in diet, after weaning...

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/LABORATORY ANIMALS: Developmental or Reproductive Toxicity/ In a reproduction study five groups of 30 male and 60 female rats of the P-generation were fed 0, 2, 5, 10 and 20% dietary xylitol. The total amount of carbohydrates was kept constant. In addition 20% sorbitol and a 20% sucrose group were used as sugar controls. ... In the animals receiving 5, 10 and 20% xylitol decreased food-intake and weight gain was recorded. At the first mating, a slightly higher initial litter size among test groups was counterbalanced by a slightly higher cumulative pup loss during lactation. No other effects on the reproduction were noticed in the F1a-animals receiving xylitol. ...

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/GENOTOXICITY/ Xylitol caused no observable mutagenic effects in an Ames test with Salmonella typhimurium TA1535, TA1537, and TA1538 with and without S-9 metabolic activation; host-mediated assay in the mouse with Salmonella typhimurium TA1530, TA1532, and TA1964; micronucleus test with Fullinsdorf albino mice; chromosome analysis of cultured, PHA-stimulated human lymphocytes.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 13: Xylitol (87-99-0) (1978). Available from, as of July 8, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/ALTERNATIVE and IN VITRO TESTS/. ...The aim was to evaluate the effects of xylitol on Leishmania amazonensis-infected J774A.1 macrophages. Macrophages were infected with L. amazonensis for 3 hr, washed and incubated with 2.5 or 5.0% xylitol for 24, 48, and 72 hr at 37 deg C. Infection indexes for macrophages incubated only in medium were compared to those treated with xylitol. Cell viability and nitric oxide production were determined each time. Xylitol did not affect L. amazonensis or J774A.1 cell viabilities. Xylitol at 5.0% stimulated nitric oxide production by macrophages at 72 hr ($p < 0.01$). At 2.5 and 5.0%, xylitol inhibited nitric oxide production by L. amazonensis at 48 hr ($p < 0.05$) when compared to control. Infection indexes were significantly lower at 72 hr ($p < 0.05$), (16.9% and 9.6%) in cells cultivated with 2.5 and 5.0% xylitol, respectively, compared to control (38.4%). Results suggest a potential leishmanicidal action of the xylitol on infected macrophages.

Ferreira AS et al; Exp Parasitol 119 (1): 74-79(2008)

▸ *from HSDB*

/VETERINARY CASE REPORTS/ ...Xylitol consumption is considered harmless to people but is known to cause life-threatening toxicosis in dogs. Dogs that ingest doses of >0.1 g/kg of xylitol are at risk for developing hypoglycemia, while dogs that ingest >0.5 g/kg may develop acute liver failure. Treatment includes dextrose supplementation for hypoglycemia and aggressive monitoring, treatment, and supportive care for dogs experiencing hepatotoxicosis. The prognosis for dogs with uncomplicated hypoglycemia is good, whereas the prognosis for dogs that develop severe hepatotoxicosis is guarded to poor.
Abstract: PubMed

Piscitelli C et al; Compend Contin Educ Vet 32 (2): E1-4 (2010)

▸ *from HSDB*

/VETERINARY CASE REPORTS/ A 2.5-year-old castrated male English Springer Spaniel weighing 26 kg, was presented after ingestion of half of a loaf of bread containing the sweetener xylitol. Toxic effects of the xylitol in this dog included vomiting, mild hypoglycemia and fulminant hepatic failure. Clinical management of acute hepatic failure and subsequent coagulopathy .../included/ supportive care and fresh frozen plasma .... The dog was discharged 3 days after admission after a full clinical recovery.

Todd JM, Powell LL; J Vet Emerg Crit Care 17 (3): 286-289 (2007)

▸ *from HSDB*

/OTHER TOXICITY INFORMATION/ The effect of xylitol in Long-Evans rats was compared to fructose, glucose or no carbohydrate supplement on levels of ascorbic acid, ketonic metabolites and certain serum hormones in the normal and streptozotocin-diabetic state. Liver glycogen levels were determined. The livers of rats not treated with streptozotocin, glucose or xylitol (4% dietary level) contained significantly smaller glycogen levels than fructose or control groups. In xylitol treated rats, serum insulin levels were slightly decreased as compared to controls and serum glucagon was markedly depressed as compared to the other groups. The liver total ascorbic acid concentrations in untreated rats were significantly smaller than in fructose or xylitol fed rats. Rendering the rats diabetic with streptozotocin produced similar metabolic consequences regardless of dietary carbohydrates tested. The above result is in contrast with previously observed antiketogenic effects of fructose and xylitol.

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 18: Xylitol (87-99-0) (1983). Available from, as of July 11, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

/OTHER TOXICITY INFORMATION/ The Food and Drug Administration is cautioning consumers about the risks associated with the accidental consumption of xylitol by dogs and ferrets. Xylitol is a sugar alcohol approved for use in many common products, including sugar-free baked goods, candy, oral hygiene products, and chewing gum. Xylitol can be found in many over-the-counter drugs such as chewable vitamins and throat lozenges and sprays. It can also be purchased in bulk bags for use in home baking. These products are intended only for human use. FDA is aware of complaints involving dogs that experienced illness associated with the accidental consumption of xylitol. Xylitol is safe for humans but it can be harmful to dogs and ferrets. FDA is advising consumers to always read the label on products and to not presume that a product that is safe for humans is safe for your pet. The FDA reports included clinical signs such as a sudden drop in blood sugar (hypoglycemia), seizures and liver failure. If you suspect your pet has ingested xylitol, some signs to look for are depression, loss of coordination and vomiting. The signs of illness may occur within minutes to days of ingesting xylitol. Owners should consult their veterinarian or pet poison control center immediately for advice if they know or suspect that their pet has ingested a human product containing xylitol.

FDA; Center for Veterinary Medicine, CVM Updates: FDA is Warning Pet Owners on the Dangers of Xylitol Ingestion in Dogs and Ferrets (February 18, 2011). Available from, as of July 21, 2011: http://www.fda.gov/AnimalVeterinary/NewsEvents/CVMUpdates/ucm244076.htm

▸ *from HSDB*

/OTHER TOXICITY INFORMATION/ d-Arabitol was observed to be toxic to many laboratory strains of Escherichia coli K-12, and xylitol was found to be toxic to an existing E. coli C mutant strain. Fructose-specific components of the phosphoenolpyruvate:sugar phosphotransferase system are required for xylitol toxicity. Selection for xylitol resistance results in Fru(-) strains blocked in fructose phosphotransferase. Introduction of the ptsF or ptsI mutation into a xylitol-sensitive strain eliminates sensitivity. (14)C /labeled/ fructose uptake experiments imply that the mutation to xylitol sensitivity, which is co-transducible with ara and leu, results in derepression of normally inducible fructose phosphotransferase. Wild-type strains also become xylitol sensitive if induced by (and then removed from) fructose. Xylitol toxicity is prevented by fructose in both wild-type and mutant strains. Circumstances causing xylitol, a new food additive, to become toxic to an otherwise insensitive wild-type organism have not been reported previously. The d-arabitol-sensitive laboratory strains are galactitol (dulcitol) utilizers, although most other strains are not. Selection for d-arabitol resistance results in Gat(-) strains blocked in a constitutive galactitol-specific component of the phosphotransferase system. A mutation causing d-arabitol sensitivity occurred many years ago in AB284, the parent of AB311, AB312, AB313, and many other strains. d-Arabitol sensitivity also occurs in sorbitol-constitutive strains and is shown, like the previous two instances of pentitol toxicities, to result from a constitutive phosphotransferase, which is blocked in mutants selected for resistance.[Reiner AM; J Bacteriol 132 (1): 166-73 (1977)] Full text: PMC221841 Abstract: PubMed

▸ *from HSDB*

13.1.5 Non-Human Toxicity Values

LD50 Rabbit iv 4 g/kg

Rowe, R.C., Sheskey, P.J., Quinn, M.E.; (Eds.), Handbook of Pharmaceutical Excipients 6th edition Pharmaceutical Press, London, England 2009, p. 788

▸ *from HSDB*

LD50 Rabbit oral 16.5 g/kg

Rowe, R.C., Sheskey, P.J., Quinn, M.E.; (Eds.), Handbook of Pharmaceutical Excipients 6th edition Pharmaceutical Press, London, England 2009, p. 788

▸ *from HSDB*

LD50 Rat iv 10.8 g/kg

Rowe, R.C., Sheskey, P.J., Quinn, M.E.; (Eds.), Handbook of Pharmaceutical Excipients 6th edition Pharmaceutical Press, London, England 2009, p. 788

▸ *from HSDB*

LD50 Rat oral 17.3 g/kg

Rowe, R.C., Sheskey, P.J., Quinn, M.E.; (Eds.), Handbook of Pharmaceutical Excipients 6th edition Pharmaceutical Press, London, England 2009, p. 788

▸ *from HSDB*

LD50 Mouse oral 12.5 g/kg

Rowe, R.C., Sheskey, P.J., Quinn, M.E.; (Eds.), Handbook of Pharmaceutical Excipients 6th edition Pharmaceutical Press, London, England 2009, p. 788

▸ *from HSDB*

LD50 Mouse iv 12 g/kg

Rowe, R.C., Sheskey, P.J., Quinn, M.E.; (Eds.), Handbook of Pharmaceutical Excipients 6th edition Pharmaceutical Press, London, England 2009, p. 788

▸ *from HSDB*

LD50 Mouse ip 22.1 g/kg

Rowe, R.C., Sheskey, P.J., Quinn, M.E.; (Eds.), Handbook of Pharmaceutical Excipients 6th edition Pharmaceutical Press, London, England 2009, p. 788

▸ *from HSDB*

LD50 Mouse oral 20.96 - 23.62 g/kg

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 18: Xylitol (87-99-0) (1983). Available from, as of July 11, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

LD50 Rabbit iv 4,000-6,000 mg/kg bw

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

LD50 Rabbit oral >2,000 mg/kg bw

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

LD50 Rat oral 14,100 mg/kg bw

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

LD50 Rat oral >4,000 mg/kg bw

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

LD50 Mouse iv 3,770-9,450 mg/kg bw

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

LD50 Mouse iv 22,200 mg/kg bw

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

LD50 Mouse iv >4,000 mg/kg bw

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

LD50 Mouse sc >2,000 mg/kg bw

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

LD50 Mouse ip >2,000 mg/kg bw

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

LD50 Mouse oral 25,700 mg/kg bw

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

LD50 Mouse oral >4,000 mg/kg bw

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

LD50 Mouse oral 22,000 mg/kg bw

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

▸ *from HSDB*

LD50 Mouse oral 14,100mg/kg bw

WHO/FAO: Expert Committee on Food Additives. Summary of Toxicological Data of Certain Food Additives Series 12: Xylitol (87-99-0) (1977). Available from, as of July 6, 2011: http://www.inchem.org/pages/jecfa.html

from HSDB

13.2 Ecological Information

13.2.1 Environmental Fate/Exposure Summary

Xylitol's production and use as a bulk sweetener, in dietetic foods, pharmaceutical preparations, and solutions for parenteral nutrition may result in its release to the environment through various waste streams. If released to air, an estimated vapor pressure of 2.5X10-3 mm Hg at 25 deg C indicates xylitol will exist solely as a vapor in the atmosphere. Vapor-phase xylitol will be degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 3 hours. Xylitol does not contain chromophores that absorb at wavelengths >290 nm, and therefore is not expected to be susceptible to direct photolysis by sunlight. If released to soil, xylitol is expected to have very high mobility based upon an estimated Koc of 10. Volatilization from moist soil surfaces is not expected to be an important fate process based upon an estimated Henry's Law constant of 1.5X10-11 atm-cu m/mole. Xylitol may not volatilize from dry soil surfaces based upon its vapor pressure. An 82% of theoretical BOD in 2 weeks using an activated sludge inoculum in the Japanese MITI test suggests that biodegradation may be an important environmental fate process in soil and water. If released into water, xylitol is not expected to adsorb to suspended solids and sediment based upon the estimated Koc. Volatilization from water surfaces is not expected to be an important fate process based upon this compound's estimated Henry's Law constant. An estimated BCF of 3 suggests the potential for bioconcentration in aquatic organisms is low. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyze under environmental conditions. Occupational exposure to xylitol may occur through inhalation and dermal contact with this compound at workplaces where xylitol is produced or used. Use data indicate that the general population may be exposed to xylitol via ingestion of and dermal contact with consumer products containing xylitol. (SRC)

from HSDB

13.2.2 Artificial Sources

Xylitol's production and use as a bulk sweetener, in dietetic foods, pharmaceutical preparations, and solutions for parenteral nutrition(1) may result in its release to the environment through various waste streams(SRC).

(1) Schiweck H et al; Ullmann's Encyclopedia of Industrial Chemistry 7th ed. (2010). New York, NY: John Wiley & Sons; Sugar Alcohols. Online Posting Date: May 30, 2011

from HSDB

13.2.3 Environmental Fate

TERRESTRIAL FATE: Based on a classification scheme(1), an estimated Koc value of 10(SRC), determined from a structure estimation method(2), indicates that xylitol is expected to have very high mobility in soil(SRC). Volatilization of xylitol from moist soil surfaces is not expected to be an important fate process(SRC) given an estimated Henry's Law constant of 1.5X10-11 atm-cu m/mole(SRC), using a fragment constant estimation method(3). Xylitol is not expected to volatilize from dry soil surfaces(SRC) based upon an estimated vapor pressure of 2.5X10-3 mm Hg at 25 deg C(SRC), determined from a fragment constant method(4). An 82% of theoretical BOD in 2 weeks using an activated sludge inoculum in the Japanese MITI test(5) suggests that biodegradation may be an important environmental fate process in soil(SRC).

(1) Swann RL et al; Res Rev 85: 17-28 (1983) (2) Meylan WM et al; Environ Sci Technol 26: 1560-67 (1992) (3) Meylan WM, Howard PH; Environ Toxicol Chem 10: 1283-93 (1991) (4) Lyman WJ; p. 31 in Environmental Exposure From Chemicals Vol I, Neely WB, Blau GE, eds, Boca Raton, FL: CRC Press (1985) (5) NITE; Chemical Risk Information Platform (CHRIP). Biodegradation and Bioconcentration. Tokyo, Japan: Natl Inst Tech Eval. Available from, as of Jul 12, 2011: http://www.safe.nite.go.jp/english/db.html

from HSDB

AQUATIC FATE: Based on a classification scheme(1), an estimated Koc value of 10(SRC), determined from a structure estimation method(2), indicates that xylitol is not expected to adsorb to suspended solids and sediment(SRC). Volatilization from water surfaces is not expected(3) based upon an estimated Henry's Law constant of 1.5X10-11 atm-cu m/mole(SRC), developed using a fragment constant estimation method(4). According to a classification scheme(5), an estimated BCF of 3(SRC), from an estimated log Kow of -2.56(6) and a regression-derived equation(7), suggests the potential for bioconcentration in aquatic organisms is low(SRC). An 82% of theoretical BOD in 2 weeks using an activated sludge inoculum in the Japanese MITI test(8) suggests that biodegradation may be an important environmental fate process in water(SRC).

(1) Swann RL et al; Res Rev 85: 17-28 (1983) (2) Meylan WM et al; Environ Sci Technol 26: 1560-67 (1992) (3) Lyman WJ et al; Handbook of Chemical Property Estimation Methods. Washington, DC: Amer Chem Soc pp. 15-1 to 15-29 (1990) (4) Meylan WM, Howard PH; Environ Toxicol Chem 10: 1283-93 (1991) (5) Franke C et al; Chemosphere 29: 1501-14 (1994) (6) Meylan WM, Howard PH; J Pharm Sci 84: 83-92 (1995) (7) Meylan WM et al; Environ Toxicol Chem 18: 664-72 (1999) (8) NITE; Chemical Risk Information Platform (CHRIP). Biodegradation and Bioconcentration. Tokyo, Japan: Natl Inst Tech Eval. Available from, as of Jul 12, 2011: http://www.safe.nite.go.jp/english/db.html

▸ *from HSDB*

ATMOSPHERIC FATE: According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere(1), xylitol, which has an estimated vapor pressure of 2.5X10-3 mm Hg at 25 deg C(SRC), determined from a fragment constant method(2), is expected to exist solely as a vapor in the ambient atmosphere. Vapor-phase xylitol is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals(SRC); the half-life for this reaction in air is estimated to be 3 hours(SRC), calculated from its rate constant of 4.0X10-11 cu cm/molecule-sec at 25 deg C(SRC) that was derived using a structure estimation method(3). Xylitol does not contain chromophores that absorb at wavelengths >290 nm(4), and therefore is not expected to be susceptible to direct photolysis by sunlight(SRC).

(1) Bidleman TF; Environ Sci Technol 22: 361-367 (1988) (2) Lyman WJ; p. 31 in Environmental Exposure From Chemicals Vol I, Neely WB, Blau GE, eds, Boca Raton, FL: CRC Press (1985) (3) Meylan WM, Howard PH; Chemosphere 26: 2293-99 (1993) (4) Lyman WJ et al; Handbook of Chemical Property Estimation Methods. Washington, DC: Amer Chem Soc pp. 8-12 (1990)

▸ *from HSDB*

13.2.4 Biodegredation

AEROBIC: Xylitol, present at 100 mg/L, reached 82% of its theoretical BOD in 2 weeks using an activated sludge inoculum at 30 mg/L in the Japanese MITI test(1).

(1) NITE; Chemical Risk Information Platform (CHRIP). Biodegradation and Bioconcentration. Tokyo, Japan: Natl Inst Tech Eval. Available from, as of Jul 12, 2011: http://www.safe.nite.go.jp/english/db.html

▸ *from HSDB*

13.2.5 Abiotic Degredation

The rate constant for the vapor-phase reaction of xylitol with photochemically-produced hydroxyl radicals has been estimated as 4.0X10-11 cu cm/molecule-sec at 25 deg C(SRC) using a structure estimation method(1). This corresponds to an atmospheric half-life of about 3 hours at an atmospheric concentration of 5X10+5 hydroxyl radicals per cu cm(1). Xylitol is not expected to undergo hydrolysis in the environment due to the lack of functional groups that hydrolyze under environmental conditions(2). Xylitol does not contain chromophores that absorb at wavelengths >290 nm(2), and therefore is not expected to be susceptible to direct photolysis by sunlight(SRC).

(1) Meylan WM, Howard PH; Chemosphere 26: 2293-99 (1993) (2) Lyman WJ et al; Handbook of Chemical Property Estimation Methods. Washington, DC: Amer Chem Soc pp. 7-4, 7-5, 8-12 (1990)

▸ *from HSDB*

13.2.6 Bioconcentration

An estimated BCF of 3 was calculated in fish for xylitol(SRC), using an estimated log Kow of -2.56(1) and a regression-derived equation(2). According to a classification scheme(3), this BCF suggests the potential for bioconcentration in aquatic organisms is low(SRC).

(1) Meylan WM, Howard PH; J Pharm Sci 84: 83-92 (1995) (2) US EPA; Estimation Program Interface (EPI) Suite. Ver. 4.1. Jan, 2010. Available from, as of Jul 12, 2011: http://www.epa.gov/oppt/exposure/pubs/episuitedl.htm (3) Franke C et al; Chemosphere 29: 1501-14 (1994)

▸ *from HSDB*

13.2.7 Soil Adsorption/Mobility

Using a structure estimation method based on molecular connectivity indices(1), the Koc of xylitol can be estimated to be 10(SRC). According to a classification scheme(2), this estimated Koc value suggests that xylitol is expected to have very high mobility in soil.

(1) Meylan WM et al; Environ Sci Technol 26: 1560-67 (1992) (2) Swann RL et al; Res Rev 85: 17-28 (1983)

▸ *from HSDB*

13.2.8 Volatilization from Water/Soil

The Henry's Law constant for xylitol is estimated as 1.5X10-11 atm-cu m/mole(SRC) using a fragment constant estimation method(1). This Henry's Law constant indicates that xylitol is expected to be essentially nonvolatile from water and moist soil surfaces(2). Xylitol is not expected to volatilize from dry soil surfaces(SRC) based upon an estimated vapor pressure of 2.5X10-3 mm Hg(SRC), determined from a fragment constant method(3).

(1) Meylan WM, Howard PH; Environ Toxicol Chem 10: 1283-93 (1991) (2) Lyman WJ et al; Handbook of Chemical Property Estimation Methods. Washington, DC: Amer Chem Soc pp. 15-1 to 15-29 (1990) (3) Lyman WJ; p. 31 in Environmental Exposure From Chemicals Vol I, Neely WB, Blau GE, eds, Boca Raton, FL: CRC Press (1985)

▸ *from HSDB*

13.2.9 Probable Routes of Human Exposure

NIOSH (NOES Survey 1981-1983) has statistically estimated that 984 workers (796 of these were female) were potentially exposed to xylitol in the US(1). Occupational exposure to xylitol may occur through inhalation and dermal contact with this compound at workplaces where xylitol is produced or used. Use data indicate that the general population may be exposed to xylitol via ingestion of and dermal contact with consumer products containing xylitol(SRC).

(1) NIOSH; NOES. National Occupational Exposure Survey conducted from 1981-1983. Estimated numbers of employees potentially exposed to specific agents by 2-digit standard industrial classification (SIC). Available from, as of Jul 12, 2011: http://www.cdc.gov/noes/

▸ *from HSDB*

14 Literature

14.1 Depositor Provided PubMed Citations

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14.2 NLM Curated PubMed Citations

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from PubChem

14.3 Synthesis References

Hasumi, Fumihiko; Teshima, Chitoku; Okura, Ichiro. Synthesis of xylitol by reduction of xylulose with the combination of hydrogenase and xylulose reductase. Chemistry Letters (1996), (8), 597-598.

from Human Metabolome Database (HMDB)

14.4 General References

Nayak PA, Nayak UA, Khandelwal V: The effect of xylitol on dental caries and oral flora. Clin Cosmet Investig Dent. 2014 Nov 10;6:89-94. doi: 10.2147/CCIDE.S55761. eCollection 2014.
Abstract: PubMed

from DrugBank

14.5 Metabolite References

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1 to 5 of 20 View More

PMID	Reference
11283793	Verhoeven NM, Huck JH, Roos B, Struys EA, Salomons GS, Douwes AC, van der Knaap MS, Jakobs C: Transaldolase deficiency: liver cirrhosis associated with a new inborn error in the pentose phosphate pathway. Am J Hum Genet. 2001 May;68(5):1086-92. Epub 2001 Mar 27.
19212411	Sreekumar A, Poisson LM, Rajendiran TM, Khan AP, Cao Q, Yu J, Laxman B, Mehra R, Lonigro RJ, Li Y, Nyati MK, Ahsan A, Kalyana-Sundaram S, Han B, Cao X, Byun J, Omenn GS, Ghosh D, Pennathur S, Alexander DC, Berger A, Shuster JR, Wei JT, Varambally S, Beecher C, Chinnaiyan AM: Metabolomic profiles delineate potential role for sarcosine in prostate cancer progression. Nature. 2009 Feb 12;457(7231):910-4. doi: 10.1038/nature07762.
7710082	Shetty HU, Holloway HW, Rapoport SI: Capillary gas chromatography combined with ion trap detection for quantitative profiling of polyols in cerebrospinal fluid and plasma. Anal Biochem. 1995 Jan 1;224(1):279-85.
6592775	Roe FJ: Perspectives in carbohydrate toxicology with special reference to carcinogenicity. Swed Dent J. 1984;8(3):99-111.
12359133	Onkenhout W, Groener JE, Verhoeven NM, Yin C, Laan LA: L-Arabinosuria: a new defect in human pentose metabolism. Mol Genet Metab. 2002 Sep-Oct;77(1-2):80-5.

from Human Metabolome Database (HMDB)

14.6 Springer Nature References

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from Springer Nature

15 Patents

15.1 Depositor-Supplied Patent Identifiers

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from PubChem

16 Biomolecular Interactions and Pathways

16.1 Protein Bound 3-D Structures

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16.2 Biosystems and Pathways

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16.3 DrugBank Interactions

Target	Xylose isomerase
General Function	Xylose isomerase activity
Specific Function	Involved in D-xylose catabolism.
Reference	Overington JP, Al-Lazikani B, Hopkins AL: How many drug targets are there? Nat Rev Drug Discov. 2006 Dec;5(12):993-6. Abstract: PubMed
Reference	Imming P, Sinning C, Meyer A: Drugs, their targets and the nature and number of drug targets. Nat Rev Drug Discov. 2006 Oct;5(10):821-34. Abstract: PubMed

from DrugBank

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from DrugBank

View all (5) DrugBank Interactions entries

17 Biological Test Results

17.1 BioAssay Results

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18 Classification

18.1 Ontologies

18.1.1 MeSH Tree

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from MeSH

18.1.2 ChEBI Ontology

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18.1.3 KEGG: Metabolite

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18.1.4 KEGG: Animal Drugs

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18.1.5 WIPO IPC

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from WIPO

18.1.6 ChemIDplus

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19 Information Sources

1. ## ChemIDplus /source/ChemIDplus

   Adonitol
   https://chem.nlm.nih.gov/chemidplus/sid/0000488813 https://chem.nlm.nih.gov/chemidplus/sid/0000488813
   Xylitol [INN:BAN:JAN:NF]
   https://chem.nlm.nih.gov/chemidplus/sid/0000087990 https://chem.nlm.nih.gov/chemidplus/sid/0000087990
   ChemIDplus Chemical Information Classification
   https://chem.sis.nlm.nih.gov/chemidplus/chemidheavy.jsp https://chem.sis.nlm.nih.gov/chemidplus/chemidheavy.jsp

2. ## DrugBank /source/DrugBank

   D-Xylitol
   http://www.drugbank.ca/drugs/DB01904 http://www.drugbank.ca/drugs/DB01904
   Xylitol
   http://www.drugbank.ca/drugs/DB11195 http://www.drugbank.ca/drugs/DB11195
   http://www.drugbank.ca/drugs/DB01904#targets http://www.drugbank.ca/drugs/DB01904#targets

3. ## EPA DSStox /source/EPA DSStox

   Xylitol
   https://comptox.epa.gov/dashboard/dsstoxdb/results?search=DTXSID7042514 https://comptox.epa.gov/dashboard/dsstoxdb/results?search=DTXSID7042514

4. ## European Chemicals Agency - ECHA /source/European Chemicals Agency - ECHA

   Xylitol
   https://echa.europa.eu/ https://echa.europa.eu/

5. ## Human Metabolome Database (HMDB) /source/Human Metabolome Database (HMDB)

   D-Xylitol
   http://www.hmdb.ca/metabolites/HMDB0002917 http://www.hmdb.ca/metabolites/HMDB0002917

6. ## ClinicalTrials.gov /source/ClinicalTrials.gov

   xylitol
   https://clinicaltrials.gov/ https://clinicaltrials.gov/

7. ## FDA/SPL Indexing Data /source/FDA/SPL Indexing Data

   VCQ006KQ1E
   https://www.fda.gov/ForIndustry/DataStandards/SubstanceRegistrationSystem-UniqueIngredientIdentifierUNII/ https://www.fda.gov/ForIndustry/DataStandards/SubstanceRegistrationSystem-UniqueIngredientIdentifierUNII/

8. ## EU Food Improvement Agents /source/EU Food Improvement Agents

   XYLITOL
   http://eur-lex.europa.eu/legal-content/EN/ALL/?uri=CELEX%3A32012R0231 http://eur-lex.europa.eu/legal-content/EN/ALL/?uri=CELEX%3A32012R0231

9. ## HSDB /source/HSDB

   Xylitol
   https://toxnet.nlm.nih.gov/cgi-bin/sis/search/r?dbs+hsdb:@term+@rn+@rel+87-99-0 https://toxnet.nlm.nih.gov/cgi-bin/sis/search/r?dbs+hsdb:@term+@rn+@rel+87-99-0

10. ## FAO/WHO Food Additive Evaluations - JECFA /source/FAO/WHO Food Additive Evaluations - JECFA

    XYLITOL
    http://apps.who.int/food-additives-contaminants-jecfa-database/chemical.aspx?chemID=2620 http://apps.who.int/food-additives-contaminants-jecfa-database/chemical.aspx?chemID=2620

11. SpectraBase /source/SpectraBase

https://spectrabase.com/compound/IGCKWb2OgFh#F8ys0C1WUvk
https://spectrabase.com/compound/IGCKWb2OgFh#F8ys0C1WUvk
https://spectrabase.com/compound/IGCKWb2OgFh#FFRYGRUHGNT
https://spectrabase.com/compound/IGCKWb2OgFh#FFRYGRUHGNT
https://spectrabase.com/compound/IGCKWb2OgFh#FCTSj1bbrNM https://spectrabase.com/compound/IGCKWb2OgFh#FCTSj1bbrNM
https://spectrabase.com/compound/IGCKWb2OgFh#LSKUEzYdtOO
https://spectrabase.com/compound/IGCKWb2OgFh#LSKUEzYdtOO

12. NIST /source/NIST

Adonitol
http://www.nist.gov/srd/nist1a.cfm http://www.nist.gov/srd/nist1a.cfm

13. Springer Nature /source/Springer Nature

Xylit
https://pubchem.ncbi.nlm.nih.gov/substance/341140229 https://pubchem.ncbi.nlm.nih.gov/substance/341140229
Adonit
https://pubchem.ncbi.nlm.nih.gov/substance/341139821 https://pubchem.ncbi.nlm.nih.gov/substance/341139821
xylitol
https://pubchem.ncbi.nlm.nih.gov/substance/341139771 https://pubchem.ncbi.nlm.nih.gov/substance/341139771

14. Wikipedia /source/Wikipedia

ribitol
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xylitol
https://en.wikipedia.org/wiki/Xylitol https://en.wikipedia.org/wiki/Xylitol

15. PubChem

Data deposited in or computed by PubChem
https://pubchem.ncbi.nlm.nih.gov https://pubchem.ncbi.nlm.nih.gov

16. MeSH /source/MeSH

Xylitol
https://www.ncbi.nlm.nih.gov/mesh/68014993 https://www.ncbi.nlm.nih.gov/mesh/68014993
MeSH Tree
http://www.nlm.nih.gov/mesh/meshhome.html http://www.nlm.nih.gov/mesh/meshhome.html
Sweetening Agents
https://www.ncbi.nlm.nih.gov/mesh/68013549 https://www.ncbi.nlm.nih.gov/mesh/68013549

17. ChEBI /source/ChEBI

ChEBI Ontology
http://www.ebi.ac.uk/chebi/userManualForward.do#ChEBI%20Ontology
http://www.ebi.ac.uk/chebi/userManualForward.do#ChEBI%20Ontology

18. KEGG /source/KEGG

Compounds with biological roles
http://www.genome.jp/kegg-bin/get_htext?br08001.keg http://www.genome.jp/kegg-bin/get_htext?br08001.keg
Animal drugs in Japan
http://www.genome.jp/kegg-bin/get_htext?br08331.keg http://www.genome.jp/kegg-bin/get_htext?br08331.keg

19. WIPO /source/WIPO

International Patent Classification
http://www.wipo.int/classifications/ipc/ http://www.wipo.int/classifications/ipc/

20. NCBI

LinkOut is a service that allows one to link directly from NCBI databases to a wide range of information and services beyond NCBI systems.
https://www.ncbi.nlm.nih.gov/projects/linkout https://www.ncbi.nlm.nih.gov/projects/linkout